ST PETER'S
Barton-upon-Humber, Lincolnshire

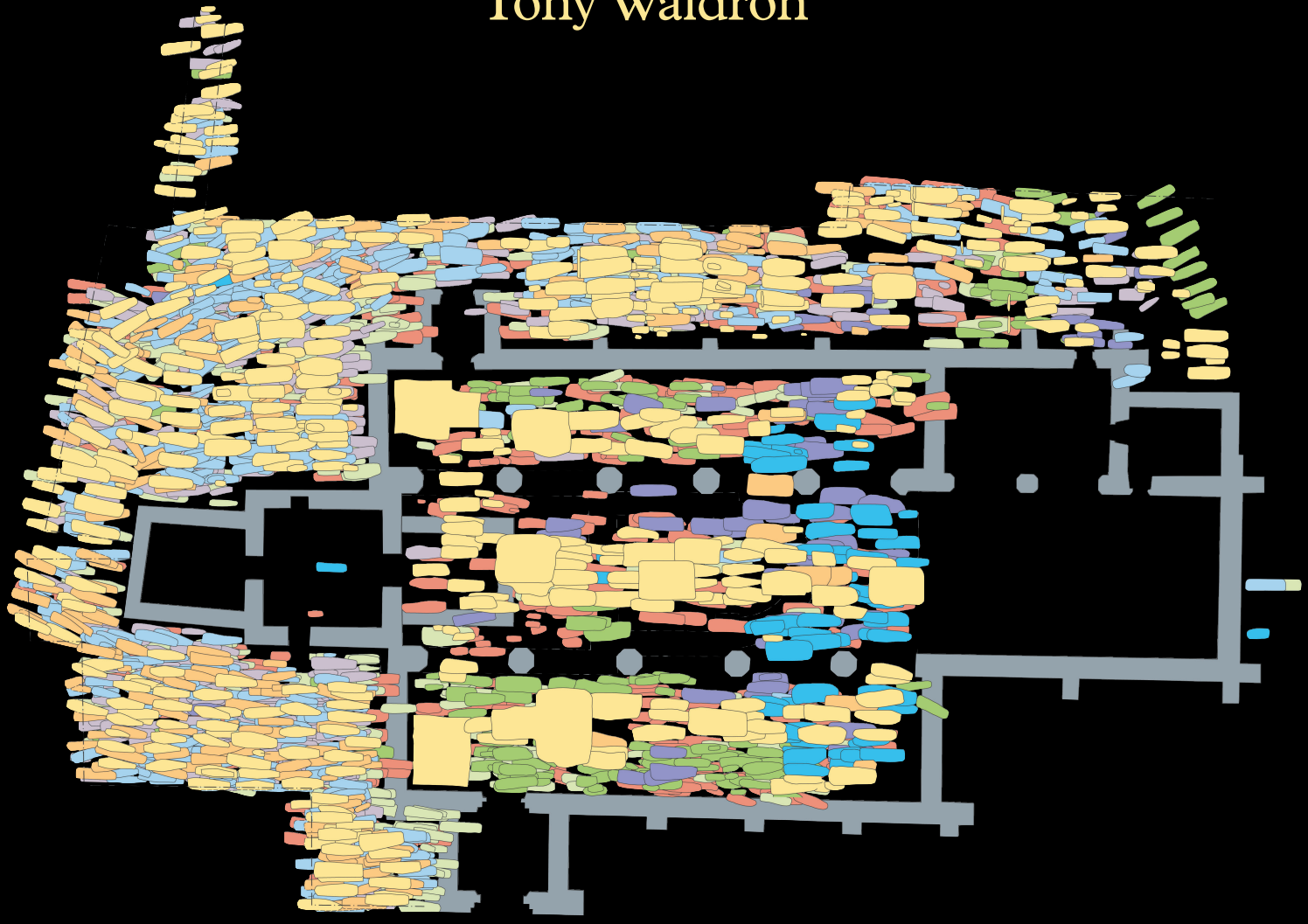
A Parish Church and its Community

VOLUME 2

THE HUMAN REMAINS

by

Tony Waldron



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Barton-Upon-Humber, Lincolnshire

A Parish Church and its Community

Juliet Margaret Rogers, 1940–2001

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with contributions by

Warwick Rodwell

Hic locus est ubi mors gaudet succurrere vitae

Oxbow Books

Published by
Oxbow Books, Oxford, UK

ISBN 978-1-84217-283-4

A CIP record for this book is available from the British Library

This book is published with the aid of a grant from English Heritage.

English Heritage is now Historic England

Text edited by Val Kinsler, 100% Proof
Page layout by Val Kinsler, 100% Proof
Summary translated into French by Charlette Sheil-Small and into German by Heike Neumann
Indexed by Susan Vaughan

Front cover:
Composite plan of excavated graves of all periods
Back cover:
Post-medieval vaults in the south aisle and nave seen during excavation of the church; skeleton of William Goy (sk. 1702); two preserved early Norman coffins

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Preface

It is seldom possible to carry out a major archaeological investigation on an intact parish church, and when an opportunity does arise it is usually restricted to a particular component of the building, or area of the site where repair or new construction is imminent. Moreover, the time available for investigation is generally limited. Very occasionally, exceptional circumstances arise, when large-scale, relatively unhurried investigations can be put in hand, and such was the situation at St Peter's church, Barton-upon-Humber in 1978.

By the early 1970s this sizeable medieval church had become superfluous to parochial requirements, there being another equally large church (St Mary's) only 100 m away, and the maintenance of two buildings was a heavy burden upon the parishioners. St Peter's was therefore declared redundant under *The Pastoral Measure, 1968*, and was placed in the care of the Department of the Environment (now English Heritage) in 1978. The intention was to repair the building and open it to the public as an historic monument.

St Peter's was already well known to architectural historians for its remarkable late Saxon tower, but the history and archaeology of the remainder of this complex, multi-period building were ill-known. The then Directorate of Ancient Monuments of the Department of the Environment therefore determined to carry out a major archaeological research programme in conjunction with the necessary repair and conservation work. The initial phase of that programme, which ran from 1978 to 1984, was directed by the present writer and Mrs Kirsty Rodwell. During this time the upstanding fabric was extensively studied, the greater part of the interior of the church was meticulously excavated, and a large swathe of churchyard around the east, north and west sides of the building was also excavated.

In addition to elucidating the structural and liturgical development of the building, over a period spanning approximately one thousand years, a large sample of the burial archaeology of the site was also investigated. In all, just over 2,800 interments were excavated, recorded, and removed for detailed study, and much disarticulated bone was also recovered. The human remains were all skeletal, there being no soft tissue preserved. However, a great deal of evidence was recovered relating to grave types, coffins, burial posture, and other aspects of funerary practice, from the late Saxon period to the mid-Victorian. Only a handful of interments took place in brick-built vaults, the remainder being earth-cut burials, either within the church or outside it.

The excavated graves span almost a millennium, down to *c.* 1855, when burial within and around the church ceased. The skeletal remains from Barton constitute by far the largest assemblage excavated from an English church and churchyard, and there is every reason to believe that they represent a good cross-section of the community of this small market town and port in north Lincolnshire. The community of Barton was relatively stable throughout the period under consideration: it was neither subjected to dramatic change at the industrial revolution, nor significantly influenced by immigration. It is the stability, continuity, and even the 'ordinariness', of the population that gives the skeletal assemblage its especial interest: in short, we believe that Barton provides a bench-mark for the study of a typical small-town population.

The potential importance of the human remains for detailed study was recognized from the outset, and provision for their careful excavation and recording was therefore included in the initial research design. Arrangements were also made to have a palaeopathologist, the late Dr Juliet Rogers, on site during the main excavation seasons. The skeletal material was subsequently transferred to the School of Medicine at the University of Bristol. There, in the Rheumatology Unit, an eight-year programme of recording and analysis was carried out under the direction of Dr Rogers. Numerous scholars and students worked on the material, and the planned programme was completed in 1999. However, her illness and untimely death, in December 2001, prevented Dr Rogers from completing the preparation of the final report for publication. In the event, Professor Tony Waldron, who had already been closely associated with the Barton project, nobly stepped into the breach and brought publication of the study to fruition.

The results of the investigations of 1978–84 at Barton, and the many years of subsequent research, are presented in two volumes. The first contains a detailed account of the history, architecture and archaeology of St Peter's church, as well as considering its local setting and wider significance. This second volume is devoted entirely to the study and analysis of the human skeletal remains. The size and importance of the collection is such that it merits publication as a separate entity. To try to incorporate the results of intensive study of such a large assemblage in any report which encompassed the entire range of evidence investigated at Barton, would have introduced a serious imbalance.

Since the two volumes are likely to be consulted, for the most part, by scholars working in substantially different fields, it was considered pragmatic to provide sufficient complementary information in each so that it is capable of standing alone. Hence, two introductory chapters are included in this volume. The first summarizes the history and topography of Barton, setting St Peter's church in its local and regional context. In the second chapter, the archaeology and chronology of

burial is discussed, together with an outline of the grave-types, and evidence of rituals, encountered. In a reciprocal vein, a summary of the evidence derived from the study of the skeletal material has been provided in a single chapter in Volume 1.

The site archive is held by English Heritage, and the human remains have been returned to St Peter's church. Disarticulated bones were reinterred on site at the end of the excavation, while the articulated burials are to be housed in a purpose-built ossuary within the former organ chamber, thus enabling the material to remain accessible for re-examination in the future.

Full acknowledgements relating to the excavation and subsequent study are contained in Volume 1, but special mention must be made here of Caroline Atkins, the project manager for the Barton study, whose tireless efforts over many years have been instrumental in bringing this work to completion. Finally, I would like to take this opportunity of paying a personal tribute to Juliet Rogers, who, for almost twenty-five years, was a loyal friend as well as a professional colleague: she was always generous with her knowledge and time, and unfailing in her interest and support. Gratefully, we offer this volume *in memoriam*.

Warwick Rodwell
Downside, Somerset
November 2005

Juliet Margaret Rogers, 1940–2001
An Appreciation

Juliet Rogers came from a medical family. Her father was a general practitioner in Somerset, while her grandfather, a physician at Bristol General Hospital, is remembered for his description of the heart murmur found in rheumatic fever, still known as the Carey Coombs murmur (Coombs 1924). Juliet studied medicine at Bristol and although she qualified in 1965, she chose not to practice: instead, after marrying and having her family, she began to take an active interest in anthropology and archaeology. Then, as has happened to many doctors who dig, she was invited to look at the bones recovered from one of the sites that she was working on. It was only a matter of time before she encountered some joint disease and this took her in search of help from the rheumatologists at the Bristol Royal Infirmary (BRI). She was lucky to encounter two clinicians who were to become life-long collaborators, colleagues and friends, Paul Dieppe and Iain Watt, the first a rheumatologist and the second a skeletal radiologist. The response to her approach was so enthusiastic that in 1977 she was invited to join the rheumatology department at the BRI where she worked until her death. With Dieppe's encouragement she was able to attract research money from the Arthritis Research Campaign and she became the only medically qualified palaeopathologist to work full-time within a clinical unit, something that enabled her to apply modern thinking on the rheumatic diseases to early populations as no-one else could.

In 1978 Juliet was invited to examine the human remains that were being excavated at Wells Cathedral, and in 1980 she began to study the remains from the excavations at St Peter's church, Barton-upon-Humber, Lincolnshire. This was without doubt the most important event in her professional life. From then on, until her death, she and Barton were inseparable. She spent several summers in Barton, first of all going through the approximately three tons of disarticulated material, which was subsequently reburied, and then through some of the most interesting pathological specimens which formed the subject of her early papers. Finally, some 3,000 skeleton boxes were delivered to her house in Nempnett Thrubwell, Somerset, where they were initially stored in her loft.

Barton became probably the best known site in the UK as Juliet described the pathology in papers and at meetings. When she was able to obtain funding for the project, the skeletons were transferred to the basement of the Dental School in Bristol, to be taken from there, across the zebra crossing, to the Rheumatology Unit opposite, and to the small basement room that served as her laboratory. She worked on Barton for the next twenty years, with assistance from Geraldine Barber, and she was beginning to prepare the final report on the site at the time of her death.

Of course, Juliet worked on other projects, one of her favourites being the bishops of Wells; and Giso, the last Anglo-Saxon bishop, became a familiar figure at meetings, special to her because he had DISH (diffuse idiopathic skeletal hyperostosis). She was invited to Lichfield Cathedral, Staffordshire, in 1984 to look at bones discovered there, and from this work she became very interested in the study of relics. Days were stolen from meetings on the Continent to go to the churches and cathedrals to look at the relics they contained and she had the plan to make a grand relic tour after her retirement, a plan in which I was to be a co-conspirator, now – sadly – not to be.

I first met Juliet at the European Paleopathology Association meeting in Middleburg, in 1982, where she was presenting a paper on DISH and where she first introduced her audience to Mary Thorley, a former resident of Barton who died in 1833 at the age of 79, and who was one of the few individuals to be identified by name among the assemblage (Rogers 1982). We very quickly developed a rapport, and subsequently we frequently collaborated and published several papers and one book together (Rogers and Waldron 1995).

Juliet will best be remembered for her work on joint disease in human remains. She was not only an authority on osteoarthritis in the skeleton, but also did important work on the erosive arthropathies, describing cases of rheumatoid arthritis, erosive osteoarthritis and psoriatic arthropathy. She was rigorous in her approach and made significant contributions towards standardizing the diagnosis of joint disease in the skeleton, publishing a number of recommendations for this purpose. She was also an authority on DISH, this interest having originally been stimulated by her study of Giso and the other Saxon bishops of Wells.

She attracted many students to work with her and she gave generously of her time and expertise, particularly to those who wished to enter the field. She supervised undergraduate and postgraduate theses with care and consideration and there can be few who do not look back on their time with her with affection and gratitude. She was much in demand as a lecturer at both clinical and non-clinical meetings and she published and travelled very widely. She had a rather diffident style of presentation which sometimes belied the intellectual force of her arguments; she did not suffer fools gladly – or at all – and could be strenuous in her rebuttal of points which she felt were not supported by sound clinical evidence or judgement, as indeed many are not in this field.

Juliet was a woman of many parts; she loved walking, cooking and gardening; she read widely and liked music and wine; she was interested in history, especially medical history, having come from a medical

family and having herself qualified in medicine. She was funny, and meetings were always good fun when she was there. Her husband had a dense stroke at the Paleopathology Association meeting in Caen in 1980 and she cared for him lovingly for many years. When he died, he was buried in the churchyard opposite her house, which had formerly been the rectory of the parish, and she is buried there with him, across from the view that she loved from the patio of her house.

A few years before her death Juliet noted that she was becoming short of breath, and when this started to limit her activity, particularly her ability to go on the walking holidays she so enjoyed, she sought medical advice and was found to have cryptogenic fibrosing alveolitis. The clinical course thereafter was rapid: she needed hospital treatment on a number of occasions and was forced finally to contemplate retirement because of the severity of her symptoms. On 17 July 2001, the Rheumatology Unit planned a *Festschrift* for her, but on the day it took place she was in hospital and had to be brought down from the ward in her bed to take part. Various colleagues presented work that they had carried out in collaboration with her, and Juliet seemed to enjoy the occasion greatly, surrounded by her family, friends and colleagues who loved her. It was the last time that I saw her.

In the months before her death, Juliet had organised in her mind the structure of her final Barton report and had made a few notes on chapter headings, and written brief introductions to some of the chapters. She had also prepared a lot of tables using the phasing details that were then available to her. Following her death, however, the site was extensively re-phased, after the results of a major programme of radiocarbon dating were obtained which invalidated her earlier analyses and conclusions, especially those which related to changes that had taken place over time, all of which had now to be revised. With the final phasing agreed, I re-analysed all the data so far as I was able, having also corrected some errors in the databases. I had access to an electronic version of the original bone recording sheets which I needed to consult frequently to obtain information which was either not sufficiently clear or incomplete in the databases from which I worked.

This report presents my analysis of Juliet's work. It is necessarily not as complete as it would have been had she written it, because she died taking an enormous amount of knowledge about the assemblage with her, as it was nowhere else but in her head. I have managed to incorporate some of her notes, however, especially those that described particularly interesting skeletons in detail; I present this volume as a tribute to her memory, and in the hope that she would not have been dissatisfied with it.

Tony Waldron

Acknowledgements

I know that Juliet would have wished to extend her thanks to all those who helped her during the many years that the Barton project was underway. In particular I have no doubt that she would have wanted to thank Warwick Rodwell for inviting her to examine the bones in the first place and so change her professional life entirely. Her thanks would also have gone to her many collaborators, and those who undertook special studies. Many – particularly the students whom Juliet taught and who did projects on the Barton material as part of their undergraduate or postgraduate studies – I am unable to thank personally. Others I know had a special place, most particularly Geraldine Barber and Rebecca Wiggins; colleagues who collaborated with her, and whom I know she would wish to thank included Richard Evershed, Friedy Luther, Anita Sengupta and Lee Shepstone. Her best thanks, however, would be reserved for Paul Dieppe and Iain Watt, and latterly John Kirwan, the first who 'spotted' Juliet as a potential member of his department and on whose support she could always count; the second who provided his invaluable radiological expertise which was so essential to her work; and the third who continued supporting her when Paul relinquished his headship of the department.

English Heritage was generous with financial support to Juliet during the early stages of the project and has continued to provide support in other ways since her death. Finally, the Arthritis Research Campaign is to be thanked for ensuring that Juliet was able to continue with her work, and for their good sense in seeing how much her work would be able to contribute to a clinical department.

My own thanks are due – of course – to Juliet for letting me be a part of this interesting and important work; to English Heritage for its support during the writing up; to colleagues who have been understanding and helpful when I needed them to be so; and, as always, to my wife Gill who has yet again put up with a house in total chaos while I complained when I had lost valuable notes, when another hard drive crashed, and when it sometimes seemed too much.

Tony Waldron

List of Publications

The following is an attempt to list, in chronological order, as many of Juliet Rogers' publications as possible: full details will be found in the bibliography at the end of this volume. Some items have doubtless been overlooked, but hopefully this does not include anything of major consequence.

Books, Papers and Articles in Learned Journals

Rogers 1980
Rogers 1981
Rogers, Watt and Dieppe 1981
Rogers 1982
Rogers 1984a
Rogers and Dieppe 1984
Rogers, Watt and Dieppe 1985a
Rogers, Watt and Dieppe 1985b
Rogers and Waldron 1986
Rogers, Waldron, Dieppe and Watt 1987
Rogers 1988
Rogers and Waldron 1988a
Rogers and Waldron 1988b
Dieppe and Rogers 1989
Rogers and Waldron 1989
Rogers and Dieppe 1990
Rogers, Watt and Dieppe 1990
Waldron and Rogers 1990
Rogers, Waldron and Watt 1991
Aaron, Rogers and Kanis 1992
Dieppe and Rogers 1992
Rogers and Dieppe 1992
Goode, Waldron and Rogers 1993
Rogers, Barber, Dieppe and Kirwan 1993
Rogers and Dieppe 1993a
Rogers and Dieppe 1993b
Rogers, Young and Dieppe 1993
Rogers and Dieppe 1994
Rogers and Young 1994
Hacking, Allen and Rogers 1994
Norman, Rogers and Dieppe 1994
Waldron, Rogers and Watt 1994
Rogers, Lim and Shepstone 1995
Rogers, Watt and Dieppe 1995
Rogers and Waldron 1995
Barber, Shepstone and Rogers 1995
Lim, Rogers, Shepstone and Dieppe 1995
Wiggins and Rogers 1995
Rogers 1996a
Crossley, Levy and Rogers 1996
Parish, McNally and Rogers 1996
Rogers, Lim, Shepstone and Turnquist 1996
Sharma, Rogers and Buckland-Wright 1996
Rogers 1997
Barber, Shepstone and Rogers 1997
Rogers, Shepstone and Dieppe 1997
Shepstone, Rogers, Kirwan and Silverman 1997a
Shepstone, Rogers, Kirwan and Silverman 1997b
Rogers 1998
Sengupta, Whittaker, Barber, Rogers and Musgrave 1999
Shepstone, Rogers, Kirwan and Silverman 1999
Rogers 2000
Shepstone, Rogers, Kirwan and Silverman 2000
Shepstone, Rogers, Kirwan and Silverman 2001
Gunnell, Rogers and Dieppe 2001
Rogers, Jeffrey and Watt 2002
Rogers, Shepstone and Dieppe, 2004

Contributions to Archaeological Reports

Everton and Rogers 1982
Rogers 1984b
Rogers 1990
Rogers 1991
Rogers 1994
Rogers 1996b
Rogers 1999
Rogers 2001

Summary

Barton-upon-Humber is a small market town in North Lincolnshire, and its origins lie in the early Middle Ages. The town was served by two large churches, one of which – St Peter's – is famous for its Anglo-Saxon tower. In 1972 that church was made redundant, and was subsequently transferred to English Heritage for preservation and display as an historic site. Between 1978 and 1984, a major programme of archaeological excavation took place both within the church and around three sides of the exterior. The aim was to elucidate the architectural history and setting of this complex, multi-period building, and to recover a substantial sample of the population for palaeopathological study.

The project was accompanied, both during and since the excavation, by an extensive programme of historical and topographical research, in order to set the archaeological evidence firmly in context. The parish registers, which extend back as far as the mid-sixteenth century, were also transcribed, and these provide an important demographic overview of the population. They also record the effects of a plague which hit Barton in 1593. The archaeological and historical evidence is presented in Volume 1, while this volume (2) is devoted entirely to a discussion of the human remains. These were studied on site by the late Dr Juliet Rogers, who subsequently carried out a programme of analysis and research at Bristol Royal Infirmary in preparation for publication; her untimely death in 2001 prevented her from seeing the fruits of many years' labours.

The excavations at St Peter's have yielded what is, to date, the largest collection of human remains in the UK: they span the period from the late tenth century to the mid-nineteenth. Preservation of the bone was, for the most part, good. The population is entirely secular, representing a cross-section of all levels of society living in the town and its hinterland. The total number of inhumations examined was 2,750, but in addition there were thousands of disarticulated bones – a quantity that was estimated to weigh approximately three tons – which were only cursorily examined.

In over half the inhumations, more than 40% of the skeleton was considered to be present, and in almost 20% the skeleton was virtually complete. In about one-third of the adult skeletons an age could not be determined and, of these, over a third could not be sexed either; this is somewhat surprising considering that the condition of the skeletons was generally good. Children under the age of 15 comprised about one-third of the total.

There were 372 adult skeletons for which an estimate of height could be made: 216 male and 156 female. There was a small (<20 mm), but not significant, increase in the mean height of the males from the earliest (pre-1500) to the latest (post-1500) period. For the females, mean height was slightly lower in the later period, but the difference was not significant.

The pathological changes in the assemblage in general conformed to expectation, with osteoarthritis and dental disease accounting for the majority of the lesions. In total, 390 skeletons were found with osteoarthritis, and of these 205 were male, 143 female, and 42 of unknown sex. The substantial excess of male cases is unusual and is particularly marked in the early period when there were 121 males and 70 females with osteoarthritis; in the later period, the corresponding numbers were 61 and 53, respectively. In addition, there were three skeletons with rheumatoid arthritis, three with reactive arthritis, two with what was almost certainly psoriatic arthropathy, and a single case of ankylosing spondylitis. Ten cases of gout were present, and three of erosive osteoarthritis. Sixteen cases of osteomyelitis were diagnosed, two of them children, but there was only a single case with the spinal lesions typical of tuberculosis. By contrast, four cases of poliomyelitis were diagnosed. Evidence of fractures was present in 152 individuals, men outnumbering women by almost three-to-one, and there were two children with skull fractures, neither of which was fatal. Among the metabolic diseases, there were 15 cases of Paget's disease and 41 cases of DISH (diffuse idiopathic skeletal hyperostosis); there were only ten cases of rickets, three in children, and no cases of scurvy. A small number of skeletons had evidence of primary or secondary bone tumours, the latter presumably being the cause of death.

Dental disease was common. Almost one-third of all adults had evidence of dental caries and about a quarter had lost teeth during life, most likely as the result of gum disease.

The strongest impression gained from the study of the human remains from St Peter's church was one of stability. Whether considering the physical characteristics, or the pattern of disease, there seems to have been almost no change worthy of note over the 900-year timespan, and it seems evident that for a long period Barton provided a stable environment in which to live: the population was seemingly well – or at least adequately – nourished, and the toll of disease was unremarkable.

The bones have been returned to St Peter's church, where they will be housed in a specially constructed ossuary, as a reference collection for future study.

Résumé

Barton-upon-Humber est un petit bourg du Nord du Lincolnshire, et sa fondation remonte au début du moyen-âge. La ville était desservie par deux grandes églises dont l'une – St Peter's – est renommée pour sa tour anglo-saxonne. En 1972, cette église est devenue superflue et a été par la suite transférée à English Heritage, dans le but de la sauvegarder et de l'ouvrir aux visiteurs en tant que monument historique. Entre 1978 et 1984 a eu lieu un important programme de fouilles archéologiques à l'intérieur de l'église ainsi que sur trois côtés à l'extérieur. Le but était de tirer au clair le contexte et l'histoire architecturale de ce bâtiment complexe remontant à plusieurs périodes, et aussi de récupérer un important échantillon de la population dans le but de faire une étude paléopathologique.

Ce projet avait été accompagné, à la fois pendant les fouilles et par la suite, d'un programme poussé de recherches historiques et topographiques, dans le but de placer les indices archéologiques fermement dans leur contexte. Les registres paroissiaux, qui remontent au milieu du seizième siècle, avaient également été transcrits, et ils ont fourni une importante vue d'ensemble démographique de la population. Ils décrivent également les conséquences d'une épidémie de peste qui frappa Barton en 1593. Les indices archéologiques et historiques sont présentés dans le Volume 1, alors que le volume actuel (2) est entièrement consacré à une discussion des restes humains. Ces derniers ont été étudiés sur place par feu Dr Juliet Rogers, laquelle effectua par la suite un programme d'analyse et de recherche à Bristol Royal Infirmary pour préparer la publication des résultats; sa mort prématurée en 2001 a fait qu'elle n'a jamais pu voir le fruit de nombreuses années de travail.

Les fouilles de St Peter ont produit ce qui est, à l'heure actuelle, le plus grand ensemble de restes humains au Royaume-Uni: ils couvrent la période allant de la fin du dixième siècle au milieu du dix-neuvième siècle. La préservation des os était, pour la plupart, bonne. La population est entièrement séculaire, représentant un échantillon de tous les niveaux sociaux vivant dans la ville et son arrière-pays. 2750 sépultures en tout ont été examinées mais, en outre, des milliers d'os désarticulés – une quantité dont le poids fut estimé à environ trois tonnes – n'ont été examinés que brièvement.

On a estimé que plus de 40% du squelette était présent dans plus de la moitié des sépultures, et que le squelette restait pratiquement dans sa totalité dans près de 20% des sépultures. On ne pouvait pas déterminer l'âge pour environ un tiers des squelettes adultes et on n'a pas pu déterminer le sexe de plus d'un tiers de ceux-ci; c'est quelque peu surprenant quand on considère que les squelettes étaient généralement en bon état. Les enfants de moins de 15 ans représentaient environ un tiers du total.

On a pu estimer la hauteur de 372 squelettes d'adultes: 216 hommes et 156 femmes. Il y avait une petite (<20 mm), mais non significative, augmentation de la taille moyenne des hommes, de la plus ancienne période (avant 1500) à la plus récente (après 1500). En ce qui concerne les femmes, la taille moyenne était un peu plus basse dans cette dernière période, mais la différence n'était pas significative.

Les changements pathologiques dans l'ensemble étaient, en général, conformes à ce qu'on prévoyait, l'ostéoarthrite et les maladies dentaires représentant la majorité des lésions. On a trouvé 390 squelettes atteints d'ostéoarthrite, dont 205 étaient des hommes, 143 des femmes et 42 de sexe indéterminé. La prépondérance considérable de cas chez les hommes est inhabituelle et est tout particulièrement marquée pendant la période la plus ancienne, où il y avait 121 hommes et 70 femmes atteints d'ostéoarthrite ; pendant la période plus récente, les nombres correspondants étaient 61 hommes et 53 femmes. En outre, il y avait trois squelettes témoignant de polyarthrite rhumatoïde, trois atteints d'arthrite réactionnelle, deux atteints de ce qui était presque certainement une arthropathie psoriasique, et un seul cas de spondylarthrite ankylosante. Dix cas de goutte étaient présents et trois cas d'ostéoarthrite érosive. Seize cas d'ostéomyélite furent diagnostiqués, dont deux étaient des enfants, mais il n'y avait qu'un seul cas témoignant des lésions de la colonne vertébrale typiques de la tuberculose. Par contraste, quatre cas de poliomyélite ont été diagnostiqués. Des indices de fractures étaient présents sur 152 squelettes, les hommes étant presque trois fois plus nombreux que les femmes, et il y avait deux enfants avec des fractures du crâne, ni l'une ni l'autre n'étant fatale. Au nombre des maladies métaboliques, il y avait 15 cas de maladie de Paget et 41 cas d'hyperostose vertébrale ankylosante; il y avait seulement dix cas de rachitisme, dont trois étaient des enfants, et il n'y avait aucun cas de scorbut. Un petit nombre de squelettes avaient des indices de tumeurs osseuses primaires ou secondaires, ces dernières étant sans doute la cause du décès.

Les maladies des dents étaient fréquentes. On a relevé des indices de caries dentaires sur près d'un tiers des adultes, et environ un quart des adultes avaient perdu des dents au cours de leur vie, probablement à la suite d'une infection des gencives.

L'étude des restes humains de l'église de St Peter a donné une forte impression de stabilité. Que l'on prenne en considération les caractéristiques physiques ou le modèle des maladies, il semble qu'il n'y ait eu pratiquement pas de changement notable au cours de cette période de 900 ans et il paraît évident que, pendant une longue période de temps, Barton avait procuré à ses habitants un environnement stable de vie: la population semblait bien – ou tout du moins suffisamment – nourrie, et les décès dus aux maladies n'étaient pas remarquables.

Les os ont été renvoyés à l'église de St Peter, où ils seront conservés dans un ossuaire construit à cet effet, et serviront de collection de référence pour une étude ultérieure.

Zusammenfassung

Barton-upon-Humber ist eine kleine Marktstadt im Norden von Lincolnshire, deren Ursprung im frühen Mittelalter lag. Die Stadt wurde zwei großen Kirchengemeinden betreut, eine der Kirchen – St. Peter – ist für ihren Angelsächsischen Turm berühmt. Im Jahr 1972 wurde diese Kirche geschlossen, und an English Heritage (die Englische Denkmalbehörde) als historische Stätte zur Erhaltung und Ausstellung abgegeben. Zwischen 1978 und 1984 wurde ein großangelegtes Programm von archäologischen Ausgrabungen durchgeführt, im Kirchengebäude selbst und an 3 Lokalitäten außerhalb. Das Ziel war die architektonische Geschichte der Gesamtanlage dieses Gebäudes, dessen Geschichte mehrere Zeitalter überspannte, aufzuklären und eine umfangreiche Anzahl von Skelettresten der Bevölkerung zu bergen, um sie palaeopathologischen Untersuchungen zu unterziehen.

Teil dieses Projekts war es, ein Forschungsprogramm anzulegen, um die archäologischen Funde in ihren historischen und topographischen Zusammenhang zu setzen. Die Kirchenbücher, die bis in die Mitte des 16. Jahrhunderts zurück reichen, wurden ebenfalls übertragen und wurden zur wichtigen Quelle, die einen demographischen überblick über die Bevölkerung erlaubte. Dort ist auch die Auswirkung der Plage aufgezeichnet, die in Barton im Jahr 1593 ausbrach. Die archäologischen Funde und geschichtlichen Belege werden im 1. Band präsentiert, dieser (der 2.) Band befasst sich ausschließlich mit den Untersuchungen der menschlichen Überreste. Diese wurden am Ausgrabungsort von der nun verstorbenen Dr. Juliet Rogers untersucht, und von ihr anschließend an der Bristol Royal Infirmary (Krankenhaus) wissenschaftlich untersucht, mit dem Ziel die Ergebnisse zu veröffentlichen. Ihr vorzeitiger Tod im Jahr 2001 verhinderte, daß sie die Erträge ihrer jahrelangen Arbeit veröffentlichen konnte.

Die Ausgrabungen bei St. Peter haben die größte Fundsammlung von menschlichen Knochen im gesamten Vereinigten Königreich hervorgebracht: Sie umfassen den Zeitraum vom Ende des 10. Jahrhunderts bis zur Mitte des 19. Jahrhunderts. Die Knochenfunde waren zum größten Teil gut erhalten. Sie stammten ausschließlich von der nicht-kirchlichen Bevölkerung, und repräsentierten einen Querschnitt der verschiedenen Gesellschaftsschichten, die in der Stadt und im Umland lebten. Insgesamt wurden 2750 Gräber untersucht, es gab aber zusätzlich noch Tausende von Einzelknochen, deren Bestand auf ungefähr drei Tonnen geschätzt wurde und die nur generell aufgezeichnet wurden.

In über der Hälfte der Gräber waren mehr als 40% des Skeletts vorhanden, in fast 20% der Gräber war das Skelett so gut wie komplett. In ungefähr ein Drittel der erwachsenen Skelette konnte das Alter nicht bestimmt werden und von diesen konnten ein Drittel auch keinem Geschlecht zugeordnet werden. Das war ungewöhnlich, da die Skelette im allgemeinen gut erhalten waren. Kinder unter 15 Jahren machten ungefähr ein Drittel des Gesamtbestandes aus. Von 372 Skeletten konnte die Körpergröße geschätzt werden, davon waren 216 Männer und 156 Frauen. Man konnte einen geringen, aber nicht unbedeutenden, Zuwachs der durchschnittlichen Körpergröße von Männern von der frühesten (vor 1500) bis zur spätesten (nach 1500) Untersuchungsperiode feststellen. Bei den Frauen war die durchschnittliche Körpergröße in der späteren Periode etwas geringer, aber der Unterschied wird nicht als bedeutend angesehen.

Die pathologischen Veränderungen in der Fundsammlung entsprachen weitgehend der Erwartung, Osteoarthrose und Zahnkrankheiten beherrschten das Krankheitsbild. Insgesamt waren 390 Skelette an Osteoarthrose erkrankt, davon 205 Männer, 143 Frauen und 42 unbekannten Geschlechts. Der große Anteil von Männern mit Osteoarthrose ist ungewöhnlich, und ist besonders in der frühen Periode ausgeprägt, der Anteil war 61 Männer und 53 Frauen. Zusätzlich wurden 3 Skelette mit chronischen Gelenkrheumatismus diagnostiziert, drei mit Reactiver Arthritis (Reiter-Syndrom), zwei waren mit großer Wahrscheinlichkeit Fälle von Psoriatischer Arthropathie und ein Fall von Ankylosierende Spondylitis. Zehn Fälle von Gicht waren vorhanden, drei von Erosiver Osteoarthritis. Sechzehn Fälle von Osteomyelitis (bakterieller Knochenhautentzündung) wurden diagnostiziert, zwei davon Kinder, aber es gab nur einen einzigen Fall von Wirbelsäulenveränderungen, die typisch für Tuberkulose sind. Es gab vier Fälle von Kinderlähmung. Es gab Hinweise für Knochenbrüche bei 152 Personen, sie traten bei Männern fast dreimal so häufig auf, es gab zwei Kinder mit Schädelbrüchen, davon war aber keiner tödlich. Bei den Stoffwechselkrankheiten gab es 15 Fälle von der Morbus Paget Krankheit und 41 Fälle von DISH (Diffuse Idiopathische Skelettale Hyperostose); es gab nur 10 Fälle von Rachitis, drei davon Kinder, und keine Fälle von Skorbut. Bei einem kleinen Teil der Skelette waren primäre und sekundäre Knochentumore vorhanden, die letzteren waren vermutlich für den Tod verantwortlich.

Zahnkrankheiten waren weit verbreitet. Fast ein drittel aller Erwachsenen hatten Karies und ungefähr ein viertel hatten Zähne verloren, wahrscheinlich durch Parodontose.

Der überwältigende Eindruck dieser Studie der menschlichen Überreste in der St. Peters Kirche ist ein Bild der Stabilität. Wenn man die physischen Eigenschaften oder das Krankheitsbild betrachtet, dann gab es im Zeitraum von 900 Jahren fast keine Veränderungen, alles scheint darauf hinzuweisen, daß Barton eine stabile Umgebung war, in der die Bevölkerung weitgehend gesund war – zumindest gut ernährt und der Krankheitsbild war keineswegs aussergewöhnlich.

Die Knochenüberreste sind an die Kirche von St. Peter wieder übergeben worden, wo sie in einem speziell dafür errichteten Ossarium aufbewahrt werden, um zukünftigen Studien zur Verfügung gestellt werden können.

1. ST PETER'S CHURCH: ITS SETTING AND COMMUNITY

by Warwick Rodwell

Barton-upon-Humber is a small market town situated at the northernmost extremity of Lincolnshire (now North Lincolnshire), on the south bank of the river Humber, 42 km (26 miles) from the mouth of the estuary, over which the fishing port of Grimsby presides. On the north bank of the river, 8 km (5 miles) downstream from Barton, lies the prosperous port and town of Kingston-upon-Hull – known today simply as Hull – which is the nearest substantial urban centre (Fig. 1). With the opening of the Humber bridge in 1981, Barton is now situated alongside a major north–south thoroughfare (A15), whereas previously it lay in an area of sparsely populated countryside that was not well served by roads. Although Barton was but a short distance to the east of a major Roman road (Ermine Street) which ran the 56 km (35 miles) north from Lincoln to a ferry at Winteringham, and thence on to York, it was separated from that road by the marshy valley containing the river Ancholme. The town was, however, linked in more recent times to Brigg, and thence to Lincoln, by a turnpike road (the former A15).

In the Roman period, and again from the early Middle Ages until modern times, connections between the Lincolnshire and Yorkshire banks of the Humber were maintained by several ferries: Winteringham to Brough, South Ferriby to North Ferriby, Barton to Hessle, Barton to Hull and, latterly, New Holland to Hull. The last-mentioned ferry only closed down when the Humber bridge opened to traffic. Access by water and road to Hull, Beverley (19 km, 12 miles), and even to York (64.5 km; 40 miles), has therefore never been more difficult than access to Lincoln, for example, except in inclement weather. However, Barton's closest connections have long been with Hull.

A flavour of what life was like at the beginning of the eighteenth century is given by Daniel Defoe in his *Tour*:

> 'There are an abundance of very good towns too in this part [of Lincolnshire], especially on the sea coast, as Grimsby, in the utmost point of the county north east, facing the Humber and the ocean, and almost opposite to Hull: a little farther within Humber is Barton, a town noted for nothing that I know of, but an ill-favoured dangerous passage, or ferry, over the Humber to Hull; where in an open boat, in which we had about fifteen horses, and ten or twelve cows, mingled with about seventeen or eighteen passengers, call'd Christians; we were about four hours toss'd about on the Humber, before we could get into the harbour at Hull; whether I was sea-sick or not is not worth notice, but that we were all sick of the passage any one may suppose.' (Defoe 1725/1983, 231–2).

Although Barton is an ancient market town, it has never developed into a thriving regional centre owing to its relative isolation. Its significance has remained strictly local, and it was, moreover, eclipsed economically by Hull, a medieval 'new town' founded by King Edward I in 1293. Nor did Barton have the potential to emerge as a significant east-coast port, a function that was better suited to Grimsby, which lies at the mouth of the Humber. Poor communications, and the limited commercial and industrial potential, ensured that Barton did not experience rapid growth or an influx of population until the early nineteenth century: the industrial revolution did not have a major impact on the town.

Nevertheless, Barton was certainly not devoid of commercial enterprise. Brick and tile making on a modest scale began in the late seventeenth century, and by the later nineteenth century some light industry had arrived in the town, including a cycle works, but this has all now disappeared. There was also a ropery and maltings, together with associated Victorian 'red-brick development' in the Waterside area. Small-scale commerce has long been, and still is, a sustaining factor, although since the Second World War Barton has increasingly become a dormitory town. Still functioning is the terminus of a single-track railway line, linking Barton to Barnetby-le-Wold and thence to main-line services.

The very essence of Barton is that it is typical of a small English market town serving local needs and inhabited by a stable and predominantly indigenous population. There is no evidence to suggest that this situation has changed to any significant degree over the past millennium. It is this long-term stability that makes the pre-Victorian population of Barton so attractive for demographic study.

Setting and Topography of Barton-upon-Humber

Barton, like several other Humberside settlements, lies at the foot of the Lincolnshire Wolds on the river terrace, just below the 15 m O.D. line. The geology is very mixed and principally comprises chalk, glacial till (boulder clay) and alluvial silt (brickearth). At frequent intervals shallow side-valleys run down from the chalk Wolds to the floodplain, and some carry streams with them, but others are now dry. These dry valleys contain small-scale deposits of sand and gravel. Associated with the junction between the chalk and the boulder clay are several artesian springs, known locally as 'blow wells'. One of these springs rises in a pond called the Beck, which lies between the two medieval churches in Barton and which clearly provided a focal point for

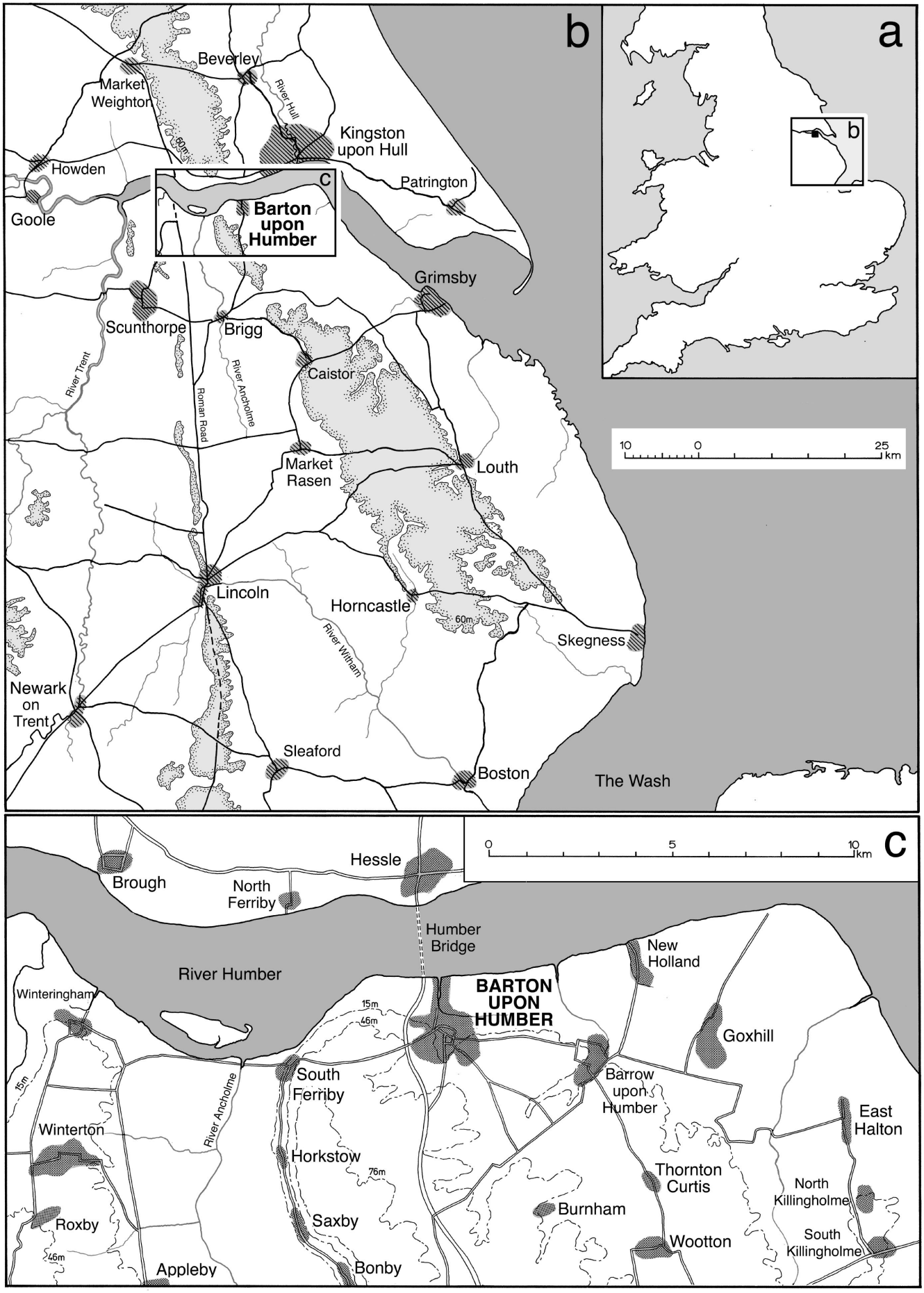


Fig. 1: Location maps. Drawing: Simon Hayfield

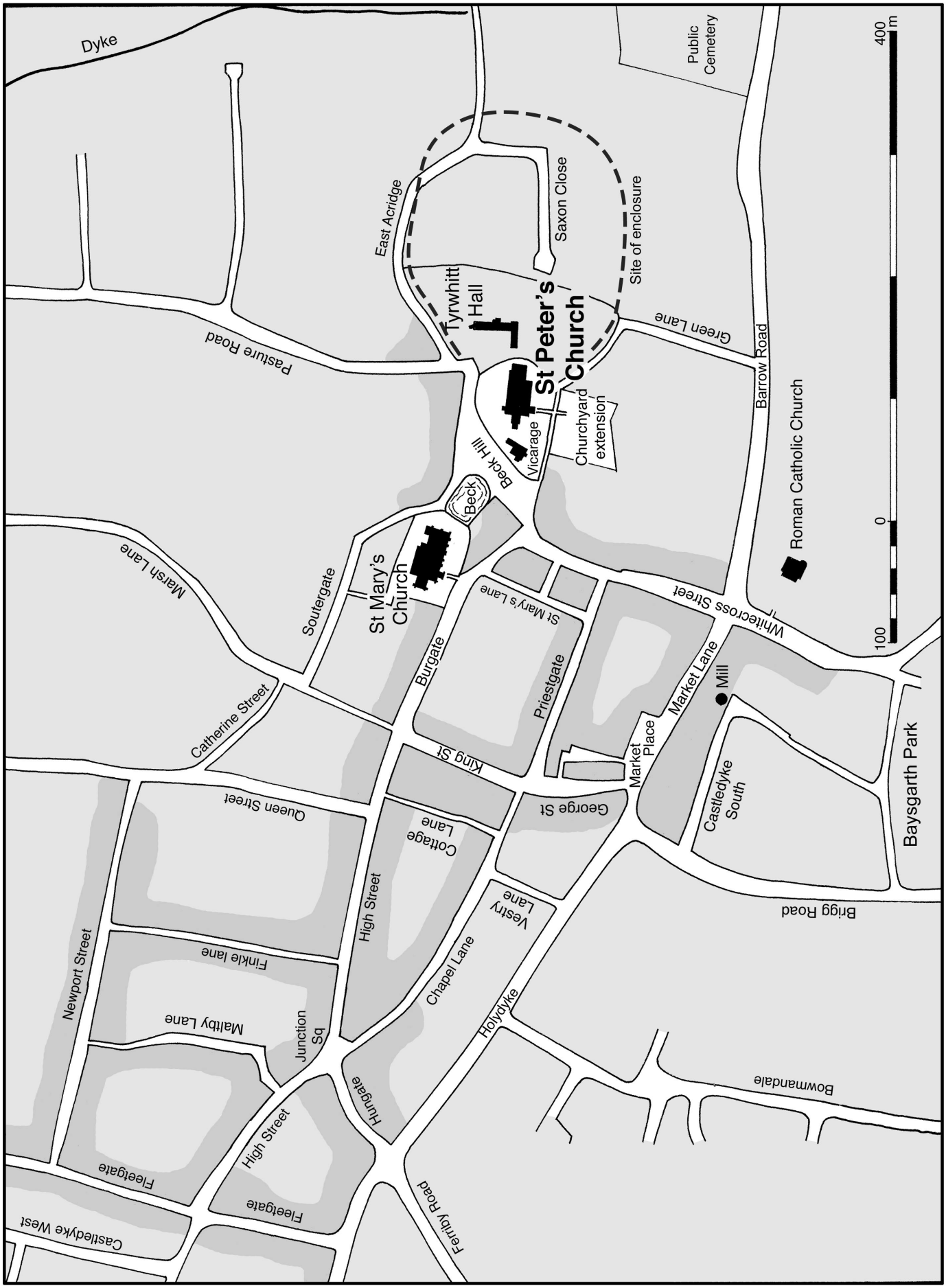


Fig. 2: Plan of the historic core of Barton-upon-Humber, showing the principal streets and topographical features. The darker tone indicates street frontages which were built-up before the middle of the nineteenth century. Drawing: Simon Hayfield

Fig. 3: Aerial photograph of Barton-upon-Humber from the north-east, c. *1960. St Peter's church is on the left and St Mary's on the right. The black tower in the distance, directly above St Peter's, is a former windmill which stands on the site of the Castledyke Anglo-Saxon cemetery. The graveyard of St Peter's was cleared of standing tombstones in 1967. Photo:* Grimsby Evening Telegraph

settlement since the Roman period, if not earlier. Until the early 1980s the Beck was regularly filled with water, and sometimes overflowed, but now it is almost invariably dry as a result of modern drainage.

The parish of Barton occupies a triangular block of land which stretches 6.4 km (4 miles) southwards from the Humber bank, to just beyond the 60 m (200 ft) contour on the Wolds. The parish contains some 2,567 ha (6,343 acres). The northern boundary comprises 5.7 km (3½ miles) of river frontage, and is flanked by a broad belt of marshland. The town lies at the interface between the marsh and the rising ground and, in addition to the Beck, several streams (now culverted) ran through the settlement area. One discharged into the Beck (which may also have been modified to provide the headwater to power a mill in Pasture Road), before finally debouching into the Humber.

The town of Barton is a loosely structured settlement, now centred on the post-medieval market place; the street pattern displays obvious elements of rectilinearity indicative of former planning (Fig. 2). However, a fully, integrated layout is not indicated, and it is clear that planned additions were made piecemeal. At the core of the medieval and later town is the pair of fine churches, separated only by a street and the Beck (Fig. 3). Here, south of the Beck, probably also lay the earliest market place. A formerly distinct focus of settlement was situated on Fleetgate, 500 m to the northwest and at the head of an artificially modified inlet from the river; this is known as the Haven. A second mill and a small, planned block of tenements developed at Fleetgate, and the ferry to Hessle ran from the mouth of the Haven.

History

Barton has been well served by local historians since the middle of the nineteenth century. Early writers include H.W. Ball (1856) and Thomas Tombleson (1905), while the most thorough exploration of the history and topography of the town is contained in two volumes by Robert Brown (1906; 1908), a local attorney. The churches in particular were studied by the Rev'd Canon W.E. Varah (1928), and more recently by Geoffrey Bryant, who has published masterly summaries of the archaeology and early history of Barton, based on extensive research (Bryant 1994; 2003). The archaeological evidence relating to all periods is discussed in greater detail in Volume 1.

Prehistoric and Roman

The river Humber was one of the principal ancient routes into eastern Britain from the North Sea, particularly in prehistoric and early Anglo-Saxon times, and it is not therefore surprising to find traces of early settlement, including the preserved remains of boats, along its banks.[1] Pre-Roman settlement in north Lincolnshire is well attested, and important sites in the locality have been excavated (May 1976). Scatters of prehistoric artefacts have been recorded at several locations in the parish of Barton, but no significant centre of occupation has been located. Early ditches and flint flakes have been found on the site of St Peter's church, and an enclosure with an irregular ditch was discovered during excavations at Castledyke South, a short distance to the south-west. The latter site is ill-explored, but is possibly that of a large enclosure (up to *c.* 175 m across) of early prehistoric origin (Drinkall and Foreman 1998, 23–4).

Likewise, in the Roman period there is no evidence for a major settlement nucleus at Barton, but groups of finds attest several localized centres of occupation; much material has also been recovered from the Humber foreshore, almost certainly indicating that riverside settlements have been inundated. The western boundary of the parish is coincident with the line of a minor Roman road which ran north-westwards from the small town of Horncastle, through Caistor (another small Roman town), to the Humber at Poor Farm, Barton.[2] Here, almost certainly, lay a ferry between the south bank and North Ferriby (the other Roman-period ferry, previously mentioned, was a little further upstream, at Winteringham). Although St Peter's church contains recycled Roman building materials in its fabric, there is nothing to suggest that it is on the site of a Roman-period structure. There was, however, a small settlement just to the north-east, under Tyrwhitt Hall and the modern housing estate in East Acridge (Fig. 2).

Early Anglo-Saxon

In the early Anglo-Saxon period the Humber once again acted as a highway for continental immigrants into eastern Britain; it was also the boundary between two early English kingdoms. The coastal and riverine distribution of both cremation and inhumation cemeteries of the pagan period in Yorkshire and Lincolnshire provides ample testimony to the arrival of Germanic folk in the fifth, sixth and early seventh centuries.[3] Northern and central Lincolnshire emerge in the annals of English history as the small and ill-understood kingdom of Lindsey.

For the early (pagan) Anglo-Saxon period the major site in the Barton locality is the Anglian cemetery at Castledyke South, only 250 m south-west of St Peter's church (Drinkall and Foreman 1998) (Figs. 2 and 3). Excavated sporadically between 1939 and 1990, several sample areas of this cemetery have together yielded in excess of 227 burials, but these represent an unknown fraction of the total number of inhumations. It seems certain that at least 400 interments took place here between the late fifth and the early eighth centuries, and were contained within the eastern part of the prehistoric earthwork mentioned above. A single urned cremation has also been found. Disused earthworks seem often to have been chosen for Anglo-Saxon burial grounds (Williams 1997, fig. 6). One urned cremation was also recovered from the enclosure ditch.

It is impossible to chart the development of the Castledyke cemetery spatially: it seems that there were initially several discrete foci, which developed independently and coalesced. Some graves were plainly laid out in rows, while others were distinguished by tight clustering. In three cases, a large and well-appointed grave seems to have been the focus of a cluster, and it has been argued that these 'special' burials were probably further distinguished by having earthen mounds erected over them (Drinkall and Foreman 1998, 355). Orientation varied considerably, although many of the later graves tended towards a west–east alignment, with the head always to the west. A move towards ordered rows, rather than focal clusters, seems also to be a late feature, and has been interpreted as potentially the result of Christianizing influences. Indeed, it is highly plausible that some of the later burials were those of Christians.[4]

The Castledyke cemetery was associated with a community of moderate affluence, as the quality and diversity of the grave-goods attest: weapons and jewellery, craft implements, vessels of bronze, glass, pottery and wood, and other personal possessions. An exceptionally rare find was a bronze balance and the accompanying set of weights; two bronze hanging-bowls are also noteworthy. From the nature of the cemetery and the origin of some of its grave-goods, there can be little doubt that the people initially buried at Castledyke were not indigenous to Britain.

While various other early Saxon finds have been made in the parish, the location of the principal settlement (or settlements) has yet to be pinpointed, but is unlikely to have been at a great distance from the Castledyke cemetery. The balance of probability favours two settlements, one on the south side of the town, and the other in the area around Tyrwhitt Hall, which was later to become the seat of the medieval manor. A small amount of early Saxon pottery has been found on the Roman site in East Acridge, in the former vicarage garden on Beck Hill, at St Peter's church, and elsewhere. The remains of timber-framed, gravel-floored buildings were excavated at the church site. Their dating is problematical, but they cannot be later than middle Saxon.

Middle and Late Saxon

It is readily apparent from topographical evidence that Tyrwhitt Hall lies within an earthwork enclosure of sub-circular plan, containing *c.* 3 ha (7.5 acres) within

the ditch (Fig. 2). The existing road pattern defines the northern side of the enclosure, and a slightly sunken footpath (running into Green Lane) follows the line of the ditch on the south. The western arc of the enclosure ditch was discovered during the excavations at St Peter's, and was shown to be of middle Saxon date. It can hardly be later than the ninth century, and was demonstrably earlier than both church and cemetery, but later than the gravel-floored buildings previously mentioned.

Although not a massive fortification, it seems likely that the earthwork was constructed to enclose and give limited protection to a middle Saxon settlement nucleus of moderately high status, which was in turn successor to a Roman and early Saxon settlement. The encircling ditch was *c*. 4.8 m (16 ft) wide by 2.4 m (8 ft) deep. By or in the late tenth century, a Christian cemetery was established immediately outside the enclosure to the west, and it was here also that St Peter's church was constructed, but perhaps not until the beginning of the eleventh century. There is no doubt that the cemetery came first, because the late Saxon church was erected over graves, but prior to that happening a 'cleansing' operation was carried out: all the graves which fell within the intended foundation circuit were exhumed.

A fundamental question arises: is the cemetery at St Peter's the direct successor to that at Castledyke South? It is an established fact that cemeteries of the pagan Saxon period did not, as a rule, develop into burial grounds of the late Saxon and medieval periods. Sometime in the middle Saxon centuries there was a conscious shift away from burial grounds of pre-Christian origin to fresh sites: the imperatives and logistics of this process are ill-known, but the general phenomenon is widespread. Pagan cemeteries commonly lay on the edge of a settlement – or at a slight remove from it – while Christian burial grounds, with their associated churches and liturgical foci, are frequently found at the very heart of a settlement. Thus, *prima facie*, a case may be argued for the Castledyke cemetery being associated with, although slightly distanced from, a settlement in the vicinity of Tyrwhitt Hall, and being superseded by St Peter's graveyard when a Christian focus was established closer at hand.[5]

The date at which such a changeover might have occurred will have been dependent upon the progress of Christian conversion in north Lincolnshire. Christianity formally arrived in Lincoln with bishop Paulinus, in *c*. 627, but the political chaos of the ensuing half-century probably militated against the establishment of a solid Christian base throughout the region. Certainly, that is unlikely to have been achieved on the south bank of the Humber until after the mid-century, at the earliest, and this is the point at which Barton enters the annals of English ecclesiastical history. In *c*. 669 King Wulfhere of Mercia appointed a devout cleric called Chad to the bishopric of Mercia and Lindsey, and he established his cathedral at Lichfield, Staffordshire.

Chad was bishop for only three years, dying in 672. However, during his short episcopate 'King Wulfhere gave him fifty hides of land to build a monastery *æt Bearuwe* in the province of Lindsey ...'. The place-name, which is perhaps better known in its Latinized form, *Ad Baruae*, simply means 'At the Wood'.[6] Recent research has established that the bounds of the fifty-hide estate were broadly coincident with those conjointly of the present-day parishes of Barton and its eastern neighbour Barrow-upon-Humber (Fig. 1c) (Everson 1984; Everson and Knowles 1993). Precisely where the focus of the monastery lay is debatable, but current opinion favours one of two locations in Barrow parish: either a long-deserted site in the village, which is today known as St Chad's, or Hann Hill, an 'island' site overlooking the Humber (Stocker 1993). Excavations at St Chad's have revealed a late Saxon cemetery and a church (Boden and Whitwell 1979). Barton must have been a second centre within the *æt Bearuwe* estate: it was probably administrative (as opposed to ecclesiastical) and very likely also a port.[7]

The Castledyke cemetery thus lay firmly within the bounds of the monastic estate and it is almost inconceivable that pagan burials could still have been taking place here in the 670s. Hence, either the cemetery was Christianized, or its use was discontinued in favour of a new burial ground elsewhere. The presence of west–east orientated graves, without sepulchral furnishings, is a strong pointer to the former practice. Although it is tempting to suggest that the shift from Castledyke to St Peter's took place soon after the foundation of Chad's monastery, there is currently insufficient evidence to support such an assertion.

Indeed, there is no denying that, on present evidence, a significant chronological gap exists between the two cemeteries, and that it broadly encompasses the eighth and ninth centuries. But this is not necessarily the obstacle to sepulchral continuity that it might seem, when it is recalled that the physical and chronological limits of neither cemetery have been established. Only four skeletons from Castledyke have been subjected to radiocarbon dating, confirming that burial there continued at least until the early eighth century. A much larger sample has been dated at St Peter's, but has not succeeded in identifying graves earlier than the tenth century. However, it is highly improbable that both the *latest* graves at Castledyke, and the *earliest* at St Peter's, have been found. That being so, an apparent chronological *lacuna* is inevitable.

Dating the fabric of the extant St Peter's church has no relevance to this issue. The architecturally elaborate turriform structure is assignable to the late tenth or early eleventh century, and it is highly unlikely that such an unusual building, which is unparalleled in Lincolnshire, would have been erected on a site where there was previously no church or chapel. Stratigraphy

has also clearly demonstrated that the late Saxon church was sited in a pre-existing cemetery. Any preceding church might have been constructed either in timber or in stone, and the pattern of burials suggests that it could have lain to the west (where the Regency former vicarage now stands). The excavated graves at St Peter's might therefore represent only a developed phase in the history of the cemetery.

Although the Barton area must have been affected by Viking raids in the ninth century, related archaeological evidence is notoriously difficult to find. It is possible that the earthwork which surrounded the town on three sides, generally known as the 'Castledykes', had its origins in a Viking riverside camp. The later years of the tenth century saw the arrival of Danish raiders in the Humber estuary, and the onset of another period of political turmoil. However, out of this somehow came the foundation of Barton as a town and major regional port: to the west of St Peter's church, a simple grid of streets and a market place were laid out before the Norman conquest (*cf*. late Saxon 'urban' development at other Danelaw towns such as Newark, Stamford and Leicester).

An outline picture of Barton in the third quarter of the eleventh century can be reconstructed from the Domesday Survey. There can be no doubting from the two relevant entries that Barton was already a thriving town by 1066, and one of the most important settlements in north Lincolnshire (Bryant 1994, 138–51). The population in 1086 can be calculated at around one thousand persons, which was double or treble that of any of the nearby large villages. The composition of the population suggests that the majority of the inhabitants were engaged in farming, but local trades and occupations related to the sea must have accounted for a significant fraction of the total.

Domesday records that by 1086 Barton had a church and a priest, a ferry, a market and two mills. It is presumed that the church in question was St Peter's, and the fact that it appears under the survey entry for Gilbert de Gant's (or Ghent's) demesne, may point to its proprietary origin (*i.e*. it was his personal property and was consequently listed along with Gilbert's other taxable assets).[8] By extension, this confirms that St Peter's was not a monastic establishment, and thus it may reasonably be assumed that those buried in its graveyard represented a cross-section of the local lay community.

Barton's market was one of only six in the whole of Lincolnshire recorded in the Domesday Survey and was almost certainly located close to St Peter's church. Its most likely site is the markedly rectangular area in the street plan immediately south of the Beck and St Mary's church, which now contains later infill development (Fig. 2: St Mary's Lane). During the medieval period, the market was shifted westwards to a new site (now George Street), and subsequently migrated south-eastwards to the present Market Place.

The ferry was one of only seven mentioned in the Lincolnshire Domesday Survey, and was apparently the most profitable. Of the other four Humber ferries, that at South Ferriby yielded the next highest return.[9]

Medieval

A comprehensive history of medieval and early post-medieval Barton has yet to be written.[10] The town evidently expanded piecemeal during the twelfth and thirteenth centuries, and some street-blocks show clear signs of formal planning: *e.g*. Fleetgate and Newport. The latter was in existence by the 1180s, and on morphological grounds the former was undoubtedly an earlier creation: it was probably early Norman, rather than late Saxon. The prosperity of the market led to the erection of a chapel-of-ease for the use of the traders. A small harbour, called the Haven, was developed at the northern end of Fleetgate, by cutting a channel southward from the river bank, across the marsh. The antiquity of the channel is uncertain: it may be Norman or earlier. There was also a second channel at the east end of the town which probably brought tidal water almost up to the Beck. Clearly, there was substantial investment in urban development at Barton in the eleventh and twelfth centuries. Moreover, there was a short-lived earthwork castle dating from the 1140s, the period of the Anarchy during King Stephen's reign. Its location has not been firmly established, but the leading contender is the slightly elevated ground on the south side of the town, close to where a windmill was later erected. Baysgarth House, the principal mansion of Barton, may be the castle's ultimate successor.

Excavation has demonstrated the developmental stages through which St Peter's church went, to arrive at the large building that has existed here since the fifteenth century (Fig. 6; see below). Similarly, St Mary's church, although technically only a dependant chapel, underwent a complex development and attracted at least two medieval chantry foundations. The opulence of the town is reflected in its church architecture, and it was almost certainly the profits from sheep farming and the wool trade that paid for the late medieval aggrandizement of both buildings.

Adjacent to St Peter's stands Tyrwhitt Hall, the manor house of Barton: this was also a very fine building in the fifteenth century, and parts of that structure survive in the present house. Unfortunately, little can be said of the town's other medieval buildings since they have almost entirely vanished. Among them was a hospital of St Leonard, founded in 1259, but its type, location and date of dissolution are all unknown (Knowles and Hadcock 1971, 313).

Post-Medieval

Towards the close of the Middle Ages, Barton entered a period of economic stagnation, partly consequent upon the rapid rise during the fourteenth century of

Kingston-upon-Hull as Humberside's principal town and port. The population of Barton undoubtedly declined (probably to well below the Domesday figure), trade slumped and the urban fabric fell into disrepair. In effect, Barton became a large, amorphous village and the inhabitants derived their livelihood principally from agriculture. Street frontages were no longer crowded with commercial and residential properties, and derelict plots must have been commonplace. This would account both for Defoe's description of Barton as 'a mean straggling town'[11] and for the fact that it was possible in the late seventeenth and eighteenth centuries for a new entrepreneurial class of owner to buy up and amalgamate large blocks of properties. Unfortunately, the earliest map of the town dates only from 1796, and was produced in association with the Barton Enclosure Act (Russell 2002). The map shows long runs of street frontage with no buildings, and large open spaces in the backlands.

The Town Book of Barton provides a snap-shot of life in the sixteenth and seventeenth centuries. Although the book in its final form dates from 1676, it is based on one of 1600, and also incorporates yet earlier material (WEA 1980). It reveals aspects of daily life, such as: details of the regulation of the sale of coal and other commodities brought in by boats to the Haven; rules for the upkeep of the dam for the water mill; rules to protect property from fire damage; regulations for the repair of streets and sea walls, and for the scouring of the drains; instructions for gathering furze for fuel; and a prohibition on making dung-heaps in the streets. Light is also shed incidentally on some unusual trades: a man was appointed to kill sparrows, so as to protect the corn growing in the common fields; there was a mole catcher for the common pastures and meadows; and the job of the pinder was to round up stray animals. The entire thrust of the Town Book was towards the protection and regulation of a community which depended upon agriculture and livestock. The few trades mentioned, such as brewers and bakers, were all closely related to farming. No hint of industry, or of commerce, is found in the book.

Barton's economy began to enter a slow renaissance around the end of the seventeenth century: importing and exporting, boat-building, fishing and the manufacture of bricks and tiles brought new prosperity. The riverine deposits of brickearth were ideal for making ceramic products: consequently, brickyards and tileries thrived until the middle of the twentieth century (Holm 1976). In the main streets of the town, the innumerable shops, workshops and small houses of the Middle Ages were supplanted by fewer, grander properties which often had large walled gardens. These were the residences of the new commercial proprietors. Timber-framed and thatched buildings disappeared, and street frontages sported little else but brick and tile.

The greatest changes came, however, at the end of the eighteenth century. Between 1793 and 1796 enclosure of the medieval common fields completely transformed the face of the parish: new roads, fields and hedges were established, and agricultural practice changed out of all recognition (Russell 2002). Over the next half-century the livelihood of the inhabitants of Barton was transmuted from being almost wholly dependent on farming, to largely non-farming related trades and professions. The changing face of Barton and the diverse nature of its inhabitants' occupations is revealed in *White's Directory* of 1856:

> 'Barton has now a Railway Station ... many neat modern houses ... A great trade in corn, malt, and flour is carried on here. There are ... several corn mills; malt and lime kilns; brick and tile, and tan and fellmongers' yards; a ship yard; a coarse pottery; and manufactories of whiting, rope, sail-cloth, &c. Gas works were constructed here in 1845.' (cited in WEA 1978, 9–10)

This list of trades is far from exhaustive, and makes no mention of, for example, boot and shoe making, or stone quarrying and the construction industry.

The final decades of the eighteenth century saw the beginnings of a new social fabric, which included the arrival of Nonconformity in the town. The Barton Old Friendly Society (1774) and the Congregational Church (1780s) were established. These were followed, in the first half of the nineteenth century, by the establishment of two Methodist Chapels, the Lodge of Oddfellows, the Temperance Society, the Barton Athenaeum, the Reading and Literary Society, and various charity, day and Sunday schools. The police station was built in 1847, the railway arrived in 1849, the Corn Exchange opened in 1854, banks were constructed, and other institutions appeared for the first time. Also in the 1850s, local newspapers began to circulate, and two were printed in Barton. The social and administrative apparatus of a small Victorian town was all rapidly being put into place, and with it class distinctions were brought sharply into focus (WEA 1977).

Changes in the social fabric of Barton are charted by the architecture of the town, by the press, and by nineteenth-century street directories. These provide instructive lists of the Georgian and Victorian residents of Barton, incidentally chronicling the rise of professions such as ministers, physicians, surgeons, solicitors, accountants and auctioneers. The directories also reveal the growing number of hostelries and places of entertainment: thus, in 1856, there were twelve inns and taverns, and six beerhouses in Barton. The nineteenth-century population censuses reveal many other interesting facts, such as the building boom and its numerous spin-offs. It is instructive to compare the map of 1796 with one of 1855, which shows the dramatic impact of new building on the townscape. The 1855 map is reproduced in WEA 1979, 68.

The population in the seventeenth century was probably little over one thousand, and at the time of the first census in 1801 it was only 1,709 persons.

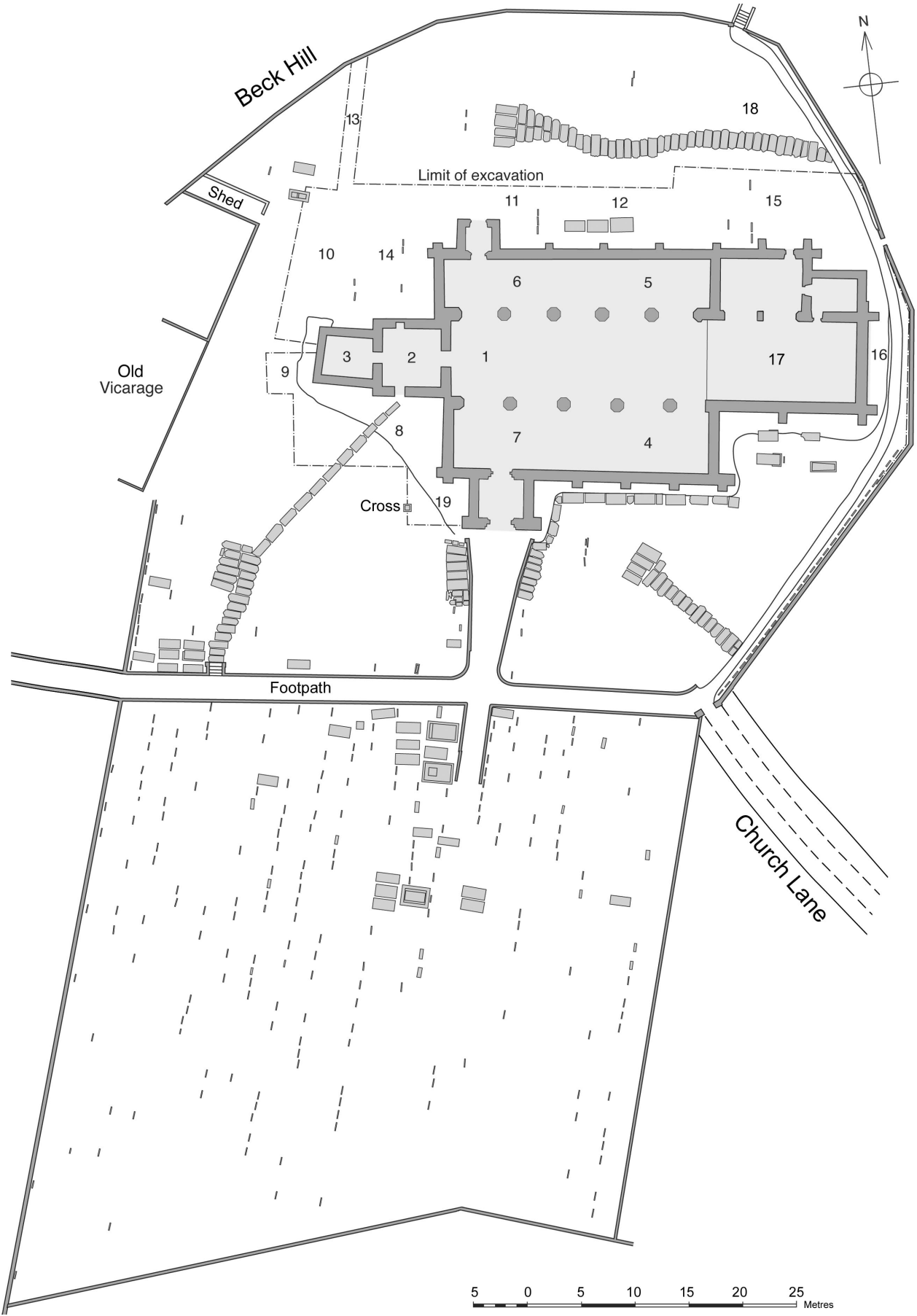


Fig. 4: Plan of St Peter's churchyard and its southern extension, as recorded in 1980–82, showing topographical features after the clearance of 1967. The limit of the excavations of 1978–84 is indicated, and the areas are numbered 1–16 and 19. Drawing: Simon Hayfield

Other, locally recorded, figures are also available for various years, and these give the break-down between the populations served by the two churches.[12] Growth, however, was rapid in the nineteenth century, and in forty years the population had doubled; thereafter the rate slowed and there were even slight reductions.[13] Population growth was accompanied by immigration. In 1851, fewer than half the parishioners were Bartonians by birth. Over 1,100 people had moved into the town from other Lincolnshire parishes, a further 749 had arrived from elsewhere in England, 65 came from Ireland, Scotland and Wales, and three were from outside Great Britain.[14] These developments probably represent the first serious change to the population-base since the Scandinavian incursions of the ninth to eleventh centuries.

The Churches of Barton

Both Barton's churches are, in their present form, moderately large, aisled buildings which reached their zenith in the late Middle Ages (Fig. 3). They are situated only 100 m apart, and both have extremely complex architectural histories, displaying structural elements spanning many centuries.[15] Down to the Reformation, the advowson was held by Bardney Abbey (Lincolnshire), and the abbot was responsible for presenting a suitable candidate for the office of vicar, who was then instituted by the Bishop of Lincoln. At the Dissolution, the patronage of the living was transferred to the Crown.

The thatched vicarage, which originally lay to the north of St Peter's church, was destroyed by fire in 1642. It was replaced by a house of brick and tile which was terraced into the west side of the churchyard, and was described in 1730 as 'a new built Vicaridge House'[16] (Fig. 4). Subsequent alterations and enlargement turned it into a late Regency stuccoed building, and this was sold by the diocese in 1981.

St Peter's church

The more easterly of the churches, dedicated to St Peter, has served as the sole parish church of Barton since the early medieval period (Figs. 5 and 6). For more than two centuries it has attracted antiquarian attention, and has acquired a distinguished rôle in the study of architectural history, largely on account of the survival of its remarkable western tower. The importance of this structure was first appreciated in the early years of the nineteenth century by Thomas Rickman during his quest for authentic examples of Anglo-Saxon architecture, at a time when scholars were divided between those who claimed a pre-Conquest date for almost every building with basic Romanesque features,

Fig. 5: St Peter's church from the south in 1999. Photo: Warwick Rodwell

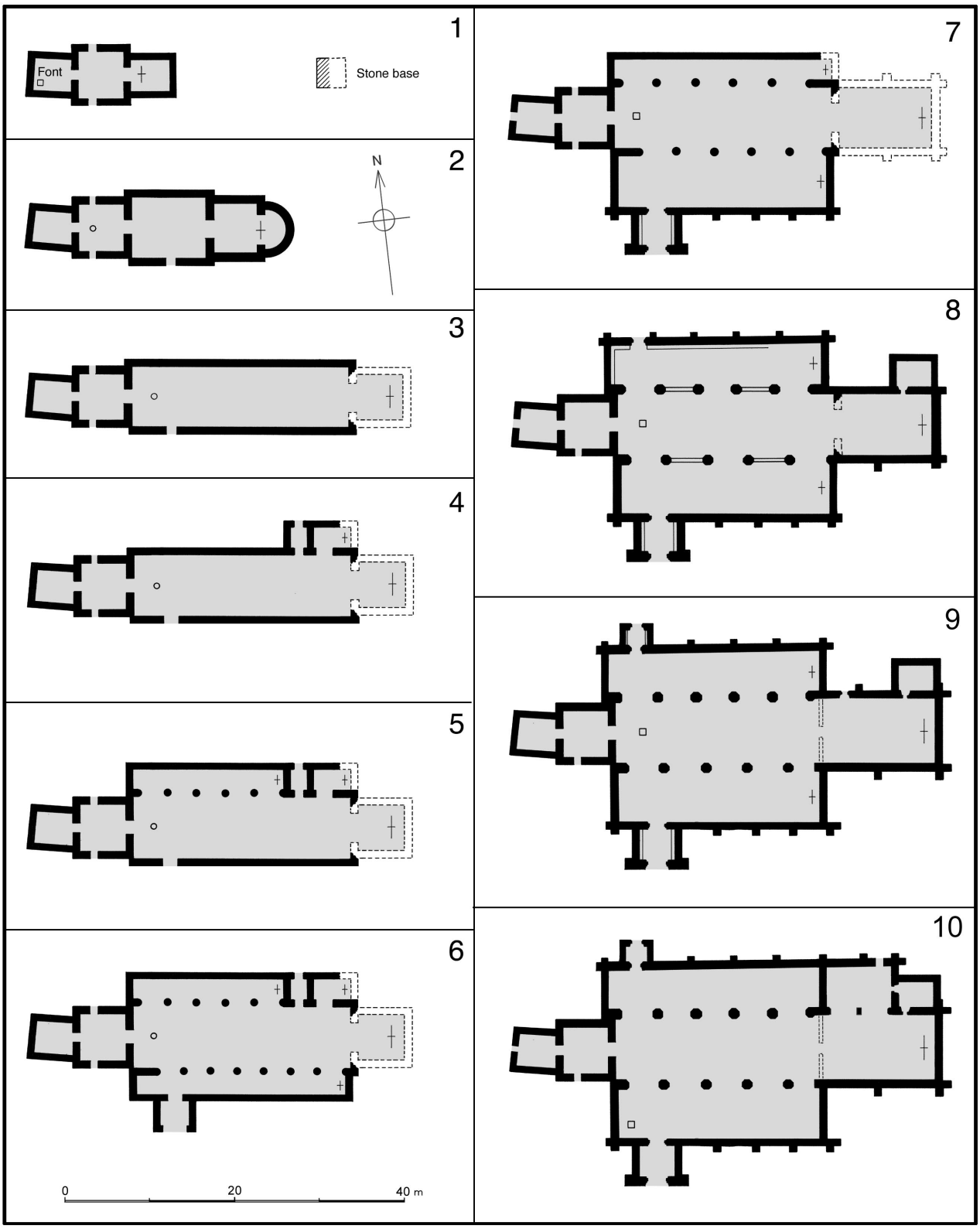


Fig. 6: Phase plans illustrating the development of St Peter's church. Font and probable altar positions are marked. 1 Late tenth or early eleventh century; 2 Mid–late eleventh century; 3 Early–mid twelfth century; 4 Mid-twelfth century; 5 Late twelfth century; 6 Early thirteenth century; 7 Late thirteenth century; 8 Mid-fourteenth century; 9 Mid-fifteenth century; 10 Mid- and late nineteenth century. Drawing: Warwick Rodwell and Simon Hayfield

and those who maintained that little or no pre-Conquest architecture survived at all. Using the principles of archaeological stratification, Rickman deduced that the lower stages of the tower at Barton must be Anglo-Saxon because they are surmounted by a belfry of clearly different style and workmanship, which, by analogy with better datable structures elsewhere, can be assigned with confidence to the Saxo-Norman 'overlap' period (Rickman 1819, 45).

In Rickman's time, nothing was known about the remainder of the church with which the pre-Conquest tower was associated. In particular, the antiquity of the small plain, gabled structure adjoining the tower on the west was unappreciated until the middle of the nineteenth century, and even then its true significance eluded discovery. Generally referred to as the 'western annexe', this feature is now known to be the only extant Anglo-Saxon baptistery.

St Mary's church

St Mary's church has been the subject of a limited amount of architectural study, but no intrusive archaeological investigation. As already noted, its origins lay in a Norman market-place chapel dedicated to All Saints, and when first mentioned in 1115 the chapel was evidently of recent foundation. The change of dedication occurred shortly before 1250. The chapel was a simple rectangular building which was greatly enlarged during the course of the late twelfth and thirteenth centuries: aisles were added, the chancel was reconstructed, and a massive west tower was erected. There were further augmentations during the fourteenth century, and in the fifteenth the impressive clerestory – not dissimilar to St Peter's – was raised above the nave.

As a dependant chapel, St Mary's would not initially have had burial rights, even though it stands in a sizeable churchyard. However, interments were being made within the chapel in the thirteenth century. Although never parochial, St Mary's developed its own identity and attracted a discrete group of parishioners, and tradition asserts that the chapel was built by the merchants of Barton. By degrees, the two churches came to serve the spiritual needs of geographically different sectors of the community, and they began to develop their own administrations. There were separate churchwardens by 1622, but only one vicar;[17] curates were, however, recorded in the parish from time to time. So far as can be ascertained, St Mary's church alone attracted medieval chantry foundations, and a chantry chapel or priest's house was erected in the north-west corner of the churchyard. After the Reformation, it passed into private ownership and later became the parish workhouse, before being demolished in 1938. Two of the recorded chantries were founded by Richard Dinot in 1268, and John de Ouresby in 1397.[18] The absence of chantries in St Peter's might be taken to imply that St Mary's had become the more prestigious of the two churches. Also, from a Bartonian's perspective St Mary's would have been regarded as the grander church in architectural terms.

The date at which St Mary's gained this semi-independence is unrecorded, but the two churches were maintaining separate registers by the mid-sixteenth century. Complete sets of registers recording baptisms, marriages and burials survive for St Peter's from 1566, and for St Mary's from 1570. There are also partial records relating to several earlier years.[19]

The Archaeology of St Peter's Church

St Peter's church underwent major restorations in 1858–59 and 1897–98, and as part of the latter campaign the first trenches were dug for the purposes of archaeological research. They successfully located the foundations of the Anglo-Saxon chancel, beneath the floor of the present nave. It was thereby established that the pre-Conquest church was a three-celled structure, comprising a tower-nave with small squarish adjuncts to the east (the chancel) and west (baptistery). The first reconstruction drawing of the original St Peter's church was published by Baldwin Brown in 1903, and the various theories concerning the history of the building were rehearsed by Robert Brown in 1906.

Further small-scale excavations were carried out in 1912–13, 1945 and 1951–54, but they failed to shed fresh light on the architectural history of the early building. Meanwhile, various scholars published their views on the form and date of the late Saxon turriform church and its possible antecedent, for which it was thought evidence had been found under the floor (*e.g.* Clapham 1946, 179–81; Taylor and Taylor 1965, 52–7). The seminal importance of the church, and in particular the tower, to later Saxon archaeology and architectural history is plainly evidenced by the prodigious number of citations which it has received in academic literature since 1819.

Nevertheless, even in the later 1970s, many fundamental questions remained unanswered, while others still awaited the asking. For example, scarcely any attention had been paid to the history and archaeology of the large medieval church that succeeded the small but elaborate Anglo-Saxon one. How and when was the transition between them effected? Then there was the seminal but unaddressed question of the relationship between St Peter's and the equally large St Mary's. There was, and always had been, only one ecclesiastical parish in Barton, and St Peter's was the parochial church. St Mary's was – remarkably in view of its size, grandeur and close proximity – still only a chapel.

In common with many other small towns in the 1960s, the parishioners of Barton found it impossible to maintain more than one church, and it was therefore decided to close two (a third Anglican church, dedicated to St Chad, had been built in 1902 at Waterside).

Fig. 7: View west along the nave in 1980, showing the excavated Saxo-Norman church of three cells, with apsidal sanctuary. Two early nineteenth-century brick-built burial vaults are also prominently visible in the nave, and others are glimpsed in the aisles. Scales of 2 m. Photo: Warwick Rodwell

Even before the Second World War, services alternated between the two medieval churches, and there never was a simultaneous need for both. St Peter's was duly closed in 1970, and its redundancy was confirmed by Order in Council in 1972: thereafter St Mary's became the parish church of Barton.

In 1974, the late Dr H.M. Taylor published a plea for a full-scale archaeological investigation of St Peter's to be launched. In view of its national importance, the church was taken into public guardianship by the Department of the Environment in 1978, and immediate consideration was given to integrating a programme of archaeological study with the necessary repair works that would be required over the next few years. While Taylor's initial plea was for the elucidation of the architectural history of the Anglo-Saxon church,

it was immediately apparent that this could not be tackled satisfactorily *in vacuo*, and that nothing less than a holistic approach to the study of St Peter's could be satisfactory in academic terms.

Based on the experience gained from other excavations within and around medieval parish churches, it was inevitable that a very large number of burials would be encountered, and it was clearly necessary to formulate a policy for dealing with human remains. The tendency hitherto among archaeologists had been to regard unaccompanied burials (*i.e.* those without grave-goods) as a nuisance and a problem: something to be cleared away as rapidly as possible so that the 'real' archaeology of the site could be investigated. However, by the 1970s the tide had begun to turn, and the importance of according the same attention to the excavation and recording of Christian burials as was given to earlier interments, was beginning to be voiced and acted upon (Rodwell and Rodwell 1976, 49; Rodwell 1981, ch. 9; 1997, 12; 2005, ch. 9).

It was therefore determined at the outset that burial archaeology would be tackled positively at Barton, an approach which was eventually to lead to the investigation of over 2,800 interments. Between 1978 and 1984 seven seasons of excavation were conducted within and immediately around St Peter's church, accompanied by as many campaigns of structural recording and investigation of the above-ground fabric of all parts of the building. An interim report on the first four seasons' work was published while investigations were still in progress (Rodwell and Rodwell, 1982), and provisional guide booklets to the church were issued (Rodwell 1983; Miller 2000). Subsequently, the restoration of the fabric has continued, and in 1985 English Heritage opened the building to the public.

A full account of the architectural development of St Peter's has been presented in Volume 1, and no more than a brief summary is necessary here (Fig. 6). Excavation revealed that the first church (of which we have knowledge) was constructed in a pre-existing cemetery, immediately outside, and to the west of, a large sub-circular earthwork of middle Saxon date. That church, a small turriform structure, was erected in the late tenth or early eleventh century. In or soon after the middle of the eleventh century, the chancel was demolished and replaced by a whole new church to which the existing tower and annexe became western appendages. This Saxo-Norman structure comprised a short rectangular nave, squarish chancel and an apsidal sanctuary (Fig. 7).

That church was in turn replaced, in three stages, by a Norman aisled building which was completed by *c.* 1200. The nave and aisles have been fully excavated, but the eastern arm lies beneath the floor of the present chancel where archaeological investigations have not taken place. In its initial form, the Norman church had a long unaisled nave and short chancel. The first addition comprised a pair of small chambers at the north-east corner of the nave: their purpose remains enigmatic, but they were possibly a chapel and a porch. These features were subsequently incorporated in a new north aisle which extended for the full length of the nave. At about the same time, a south aisle and porch were added.

The aisles were never a matched pair and, typically for the Norman period, were very narrow internally. In the second half of the thirteenth century, the south aisle and its porch were demolished, to be replaced by a new, wider aisle and larger porch. These remain today. The chancel too was probably rebuilt in the thirteenth century, and extended to the churchyard boundary. In the mid-fourteenth century it was the turn of the north aisle to be widened although, again, no attempt was made to impose symmetry on the plan. A north porch was subsequently added. At about the same time, the chancel was reconstructed to include a vestry at the north-east corner. Next, in the fifteenth century, the nave clerestory was added, and the chancel was again remodelled, using the old foundations and maintaining the same plan as its predecessor. The final structural addition to the church was the organ chamber, in 1897–98.

2. BURIAL ARCHAEOLOGY

by Warwick Rodwell

A Brief History of Burial at Barton

Although prehistoric and Romano-British settlements are recorded in the area, virtually nothing is known about their sepulchral arrangements. The history of burial at Barton effectively begins in the late fifth or early sixth century, with the Anglo-Saxon cemetery at Castledyke South (Fig. 2) (Drinkall and Foreman 1998). Adults of both sexes, together with children, were buried here, and there is no reason to doubt that they represent a cross-section of the local community in the latter part of the pagan Saxon era.

The cemetery at Castledyke South

The form taken by the Castledyke inhumations exhibited considerable variety: grave shape and size, orientation, coffin provision, and burial posture. Also, the quantity and nature of the grave-goods included with the burials was equally varied. Graves came in many sizes, the length ranging between *c.* 1.5 m and 2.2 m, and the average depth below subsoil was 20–30 cm. Hence the original depth below the ground surface probably averaged 50–60 cm. While the dimensions of a grave frequently bore a proportional relationship to the size of the corpse, 32 examples of 'outsize' graves were noted. They were among the latest in the cemetery, and probably related to coffin provision. No metal coffin fittings – such as angle-plates – were identified, and nails were very scarce.[1] Clearly, there were no nailed coffins in this cemetery. A maximum of three clenched nails occurred in one grave,[2] and 'miscellaneous' iron objects were noted sporadically, including the presence of a single rove in each of two graves.[3] The occurrence of clenched nails and roves may indicate the inclusion of timbers from clinker-built boats.

Since the site was not waterlogged, timber did not survive, and thus the former presence of coffins, grave linings, covers and other wooden features had to be inferred from circumstantial evidence. It was accordingly suggested that 23 burials were either encoffined, timber-lined, or covered with a board. The principal evidence came from skeletal movement (especially displacement of the skull, or a relaxed lower jaw) which implied that the corpse decayed in a void. It is unfortunate that few firm conclusions can be drawn regarding the use of prefabricated coffins, timber-linings in graves, or simple boards laid over corpses.

The placing of a 'pillow' of crushed or nodular chalk under the skull was noted in six graves, but no stones intended to provide lateral support ('ear-muffs') were recorded. Burial posture varied considerably, there being roughly equal numbers of what might generally be described as extended and flexed burials; within these broad categories there were many variables. Four of the seventh-century burials were 'doubles', that is, two corpses were simultaneously interred in the same grave. In the many examples where graves were closely juxtaposed, or intercutting, it was impossible to infer a familial connection, although that may have obtained in some cases. General clustering may also point crudely to this, and in a few instances it could plausibly be suggested that two graves had been deliberately 'paired'. DNA testing was not undertaken. Evidence for some graves being arranged approximately in rows was much better, but it cannot be claimed that the Castledyke cemetery was well ordered.

In 25 instances, one or more postholes seem to have been deliberately associated with a grave: they occurred singly, paired, and in quartets. Only one certain and two possible examples of the last category were recorded, whereas 19 graves were accompanied by single posts: the loss of the ancient topsoil and subsoil horizons had undoubtedly removed much of the archaeological evidence for upstanding features. It was noted that areas containing potentially coffined burials, and those accompanied by marker-posts (or post-built structures), were mutually exclusive. Finally, orientation varied dramatically: north–south burial was practised throughout the life of the cemetery, but the majority of the graves aligned west–east were of the seventh or early eighth century.

St Peter's churchyard

St Peter's church lies close to the centre of a medium-sized churchyard of irregular plan, with a long, straight boundary on the south (Fig. 4). The area enclosed today is 0.86 acre (0.35 ha), but before the vicarage was built in the late seventeenth century, cutting off the western tip, the churchyard was *c.* 0.96 acre (0.38 ha) in extent. This was a close approximation to the traditional 'God's Acre'. Coincidentally, at 0.84 acre (0.34 ha), St Mary's churchyard is almost identical in size to the reduced St Peter's.

The last burial to take place inside the church seems to have been in 1844 (in the Preston vault in the nave), but regular interment in the surrounding graveyard continued down to the 1840s, and occasionally thereafter.[4] An entirely new graveyard was created in 1850 in an adjoining field to the south of the old churchyard.[5] This extension is of 0.65 acre (0.26 ha), and graves were laid out in neat north–south rows (Fig. 4). Burial in St Mary's churchyard ceased in 1855, and thereafter all Anglican interments were consolidated in St Peter's.[6]

Increased awareness of the risks to public health caused by burial in towns, coupled with the provisions of the *Burial Act, 1857*, led to the widespread estab-

Fig. 8: St Peter's church from the south-west, in the 1890s. This part of the churchyard was popular for burial in the first half of the nineteenth century, as shown by the density of headstones. These were all cleared away in 1967. Photo: Arthur Brummitt, courtesy of John French

lishment of suburban cemeteries in the second half of the nineteenth century, and Barton followed this pattern. In 1867, a new, non-denominational, municipal cemetery was established alongside the road leading eastwards from Barton to Barrow-upon-Humber (Fig. 2). That cemetery continues in use to the present day. The churchyard extension at St Peter's was closed by Order in Council dated 1868, before all the plots had been taken up. Nevertheless, many further interments occurred in existing plots, down to the early years of the twentieth century.[7]

The practice of erecting tombstones in churchyards was rare before the later seventeenth century, and a small number of stones of that date have survived at St Peter's, albeit not *in situ*. They almost all belong to the Gildas family. Memorials of the eighteenth and the first half of the nineteenth century were, however, prolific in the churchyard, as early photographs demonstrate (Fig. 8). Unfortunately, various degradation processes during the twentieth century have reduced the number to a tiny fraction of the former total.

First, in 1907, the external ground level was lowered considerably on the south side of the tower, where a new path was formed. In order to achieve this, a number of nineteenth-century tombstones were removed. Secondly, by the mid-twentieth century, some of the monuments had begun to fragment, and the upkeep of the churchyard was burdensome. Consequently, a Faculty was obtained in 1967 to clear and level the churchyard, removing all but a very few monuments from their original positions. Some headstones were re-erected against the eastern boundary wall, others were laid as paths, and yet more were used to pave 'dry-areas' around the walls of the church. However, some memorials, along with chest panels, kerbs and other components of composite monuments, were simply broken up and carted away. Prior to this clearance, a group of parishioners produced a sketch plan of the churchyard in 1966, on which 244 memorials were marked. An accompanying schedule recorded details with varying degrees of accuracy and completeness.[8]

Thirdly, vandalism in the 1970s took a heavy toll on the remaining memorials, including those in the southern churchyard extension, which had not been affected by the 1967 clearance. Fourthly, inscriptions and decoration were also being lost through weathering. The great majority of the memorials were of Yorkstone, the surface of which tends to delaminate after a century of exposure. Once this process has begun, disintegration can follow very rapidly, and that was happening at Barton.

In an attempt to salvage such historical and genealogical information as remained, a complete survey of the surviving memorials, both *in situ* and *ex situ*, was carried out in 1978–82. The survey recorded 258

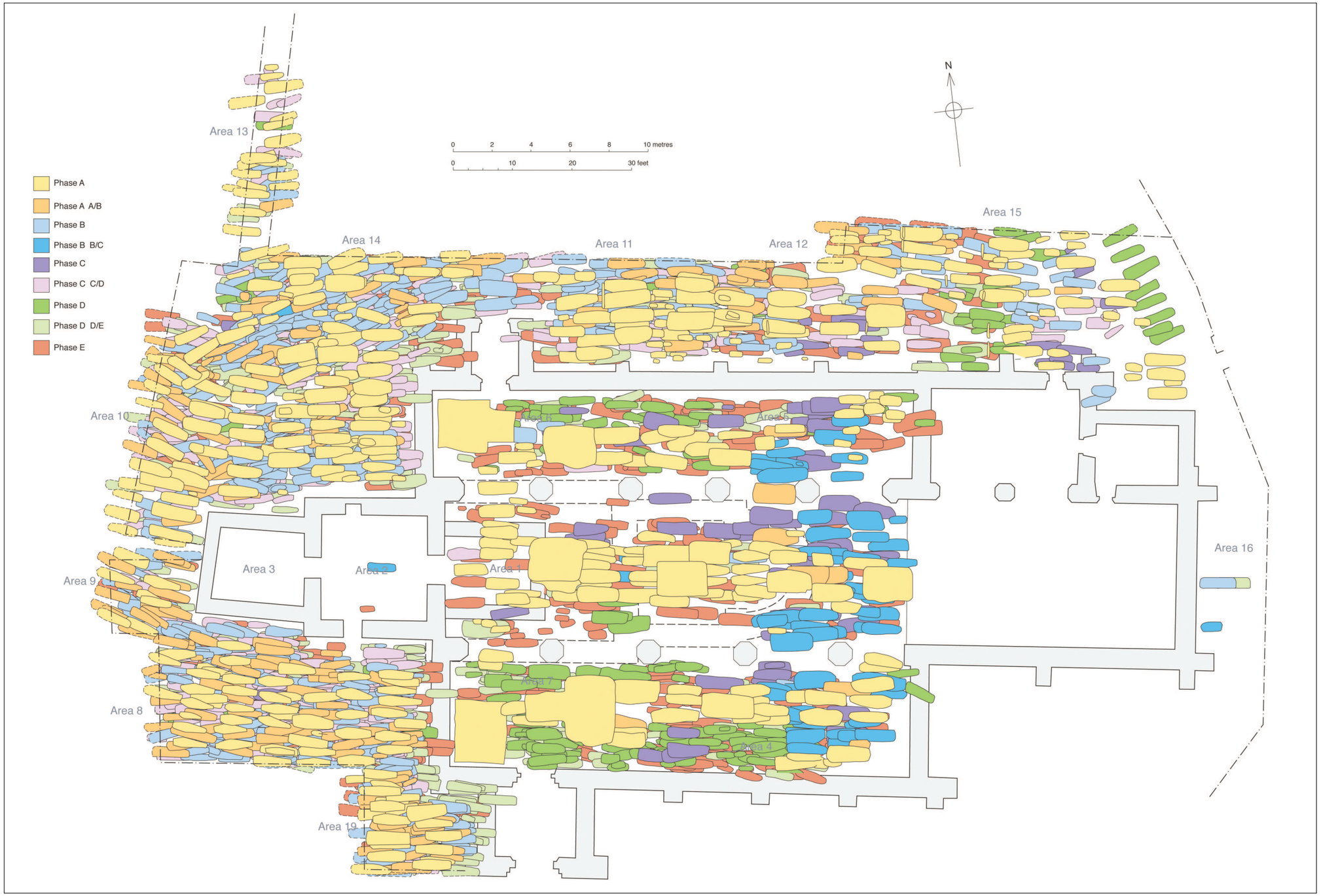


Fig. 9: Composite plan of excavated graves of all periods. Many early graves cannot be shown here because they were overlaid by later burials. Drawing: Simon Hayfield

memorials (complete and fragmentary) in the main churchyard, and 237 in the southern extension. Unfortunately, very few named memorials occurred within the excavated areas, and the imprecise nature of the 1967 survey did not allow confident identification of the exact locations from which named stones had been removed. Consequently, although numerous eighteenth- and nineteenth-century burials that had once been marked with inscriptions were excavated, the identity of the individual skeletons has been lost beyond recall. Had churchyard clearance not taken place, it would have been extremely valuable to be able to compare skeletally derived data with the historical and social record. As it is, only a handful of secure correlations between a named memorial and the underlying skeletal remains were possible.

Excavated Evidence from St Peter's

The 1978–84 excavations at St Peter's church yielded some 2,800 articulated burials, distributed both inside the present building, and outside it to the north, west and south-west (Fig. 9). Many of the 464 burials that were excavated within the walls of the church were not originally indoor graves, but lay in the surrounding cemetery, when the footprint of the church was less extensive. During the Middle Ages the building was expanded both axially (eastwards), and laterally (north and south), progressively engulfing areas of former open cemetery.

The number of excavated skeletons does not equate with the total number of graves within the area investigated: there had been many more interments, the evidence for which came in various forms. First, disarticulated bone occurred prolifically throughout the site, the quantity recovered amounting to more than three tons. Many graves had been totally destroyed by subsequent burials, while others were represented either by a tiny portion of articulated skeleton, or simply by the residual edge of a former grave-cut. There was a significant number of instances where the grave-cut was wholly or partly intact, but lacked skeletal material, either because medieval exhumation had taken place, or because unfavourable soil conditions had caused the bone to dissolve.

Recording methodology

The methodology of excavation is described in Volume 1. For present purposes it is sufficient to record that open-area excavation took place, first within the church (Areas 1–7) and then in the churchyard, as a contiguous series of trenches around three sides of the building (Areas 8–16 and 19: Fig. 4). Once uncovered, each skeleton was planned at a scale of 1:20, along with any associated evidence relating to the burial, such as the outline of the grave-cut, remains of a coffin, pillow-stones, and other features or artefacts. The extent of survival of the skeleton was recorded on a standard *skeleton form*. At the time of excavation, each articulated burial was assigned a *skeleton number* (*e.g.* sk. 1001), and the archaeological context from which it came was given a *context or feature number* (*e.g.* F1234). In the majority of cases that context was the soil filling within an identifiable grave-cut. However, in certain areas, especially around the west end of the church where burial had been very intense, it was impossible to distinguish individual grave-cuts, and all-embracing context numbers had to be assigned to large expanses of grave earth.[9]

Disarticulated remains were collected, and identified by the number of the feature from which they came.

Burial density

The density of burial varied considerably, both within the church and outside it. Internal burial in the nave and aisles was relatively rare until the seventeenth century, but then increased markedly. In the eighteenth century, when the church was packed with box pews, burials were crammed into the central alleys of the nave and aisles, and into the cross-alley between the north and south doors. The chancel is unexcavated, and thus burial disposition and density there are unknown. However, the chancel is likely to have received a greater intensity of sepulchral activity than other parts of the church, both in the Middle Ages and subsequently.

The area available for excavation within the present walls of the church was just under 500 m². If a deduction is made for medieval foundations and later vaults, which destroyed many early graves and inhibited the digging of later ones, the 464 excavated burials[10] came from an area of approximately 400 m². In very rough terms, this means that 1.2 interments per square metre were either deposited within, or sealed under, the late medieval church. The majority fell within the latter category, and the calculated density is probably well below that which actually obtained, many early graves having been destroyed by later activity.

Outdoor burial was almost invariably more dense on the south and east sides of a church than on the north, and Barton was no exception.[11] Excavation along the whole of the north side revealed a moderate burial density, but on the south investigation has only taken place around the south-west corner of the present building (Fig. 10). Hence a direct comparison between sides cannot be made. However, it is abundantly clear that burial to the south-west of the church, at all periods, was intense. Moreover, excavation within the present south aisle demonstrated that early medieval graves were densely packed against the exterior of the previous, unaisled building.

Excluding Area 16 (basically the footpath between the east end of the church and the boundary wall), the total extent of external excavation was *c.* 570 m². This yielded 2,340 burials of all periods, an average of 4.1

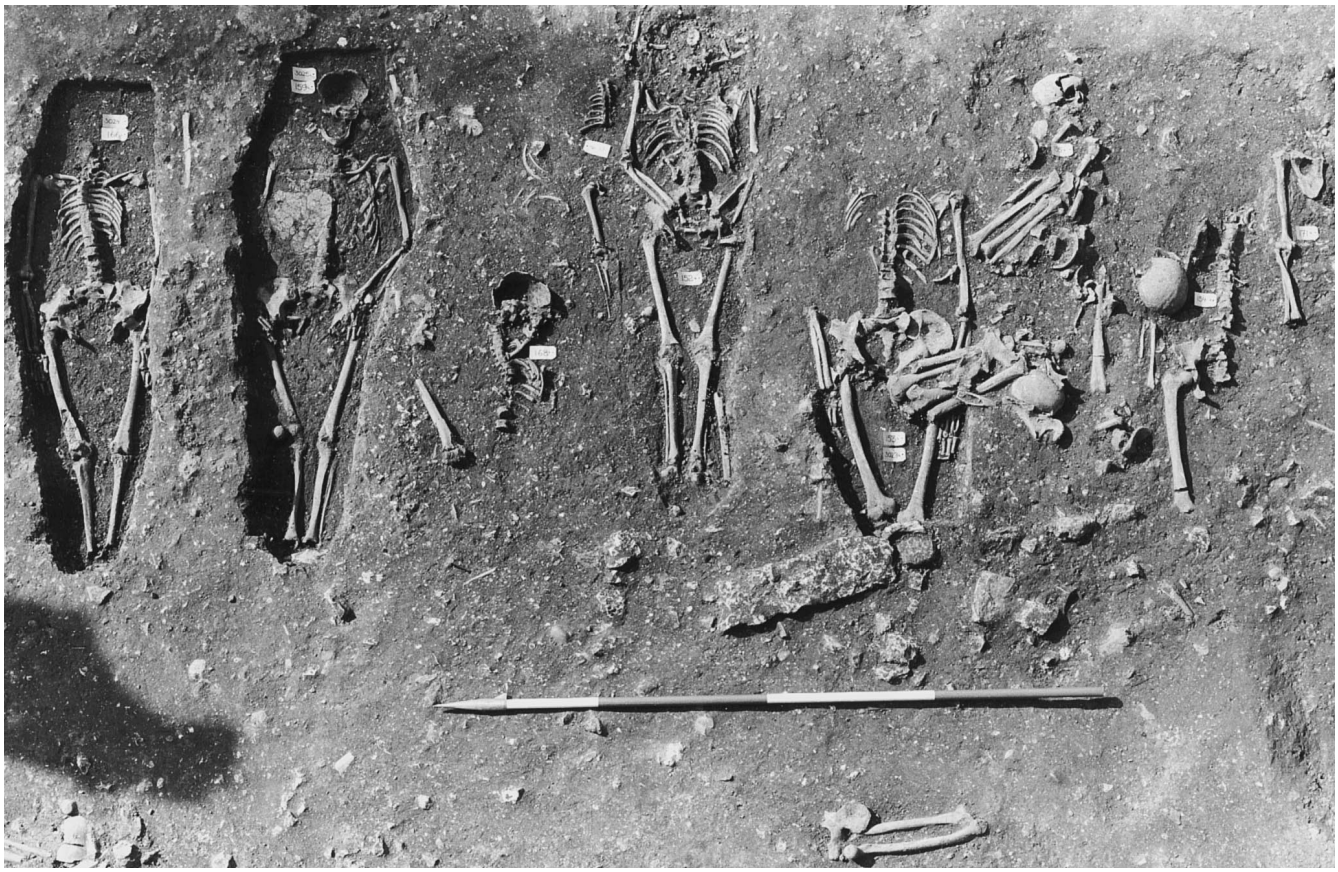


Fig. 10: A typical example of cemetery archaeology encountered adjacent to the west wall of the south aisle (Area 8). On the left are two early nineteenth-century burials in double-tapered coffins (skeletons 166 and 159). Although mostly post-medieval, the other fragmentary burials and small groups of reinterred bones are of various dates. These were all shallow interments. Scale of 2 m. Photo: Warwick Rodwell

per square metre. In the excavation south-west of the tower and annexe (Areas 8, 9 and 19), some 910 burials were recovered from 136 m^2, which gives a density of 6.7 burials per square metre. Even this is likely to be an underestimate of the true number of interments, since babies and young children were poorly represented in the overall skeletal assemblage. In the ground, their remains were relatively fragile, and were easily dispersed through intercutting burial, animal disturbance and bone decay. The true density of burial across the site is likely to have ranged between seven and ten per square metre.

While acknowledging the dangers of extrapolating from these figures to arrive at an overall burial population for St Peter's churchyard, the exercise is worthwhile, if only to provide a very approximate order of magnitude. If we take the average burial density throughout the churchyard to have been four persons per square metre, the number of interments within the present boundaries is likely to have been in the order of 14,000. The full extent of the medieval churchyard (before the vicarage was intruded on the west) potentially contained at least 18,000 burials. Since St Peter's cemetery was in use for almost a millennium, this would imply an average of only eighteen burials per annum, a reasonably modest number for a small town. Allowing for losses, it is possible that St Peter's graveyard received up to 20,000 burials altogether. For a more conservative estimate, based on population statistics, see Chapter 3.

It must, of course, be remembered that for at least half of the period in question interments were also being made in St Mary's church and churchyard.[12] It is instructive to compare these rough calculations with the earliest recorded burial statistics for Barton. In the fifteen-year period 1566–80 the combined burial registers for the two churches record 357 deaths, an average of twenty-four per annum.

Orientation and depth

The principal axis of the Norman and later church lies seven degrees off east–west (relative to the Ordnance Survey National Grid), the deflection being in a north-west to south-east direction. The vast majority of graves, of all phases, were orientated on the church and were thus more-or-less west–east. The head was invariably at the west end.

As the site plans show, there were minor, localized variations, probably caused by factors such as the alignments of paths and boundaries. The presence of trees and clumps of vegetation was doubtless also an

influencing factor. A very few burials were markedly askew from the average (either clockwise or anticlockwise), and these were mostly at the west end of the site. While some were clearly influenced by a path which crossed the churchyard diagonally, in other cases no specific reasons for their aberrant alignments could be found.

The depths at which burials occurred below modern surfaces varied from less than 30 cm to 1.7 m. The great majority of Anglo-Saxon and medieval graves were dug to a depth of *c.* 60–70 cm below the contemporary ground or floor level, but some medieval graves inside the church were up to 1.0 m deep. Post-medieval graves varied considerably: children were seldom buried at a greater depth than *c.* 60 cm, and adult graves inside the church tended to be between *c.* 1.2 m and 1.5 m deep. Outside, many burials in the sixteenth- to eighteenth-century bracket had a depth of

Fig. 11: A late Saxon grave containing two adult males and three children (skeletons 1909–1913). Their limbs were intertwined. Although the skeletons exhibit some interesting pathology, there was nothing to indicate the potential cause of these multiple deaths. Photo: Warwick Rodwell

only *c.* 50–70 cm. Some appeared to be even shallower, but that was probably a consequence of later ground-lowering in the churchyard. In the first half of the nineteenth century, however, deeper burial became common: on the north side of the tower, in particular, many graves were in the order of 1.5 m deep. These had, inevitably, caused massive destruction to earlier burials.

Establishing a close chronology for many of the graves proved difficult, but stratigraphic analysis and a major programme of radiocarbon dating provided a framework. The history of burial has been divided into five broad bands, designated as Phases A to E; for further details, see p. 29.

Grave markers

Unlike the Castledyke cemetery, postholes were very few at St Peter's. The only interesting association of postholes was in relation to a rectangular, coffin-sized pit in Area 5, where there was one hole at each corner (F1680). The feature was almost certainly a grave from which the coffin (perhaps of stone?) had been removed. Two earlier graves more-or-less on the same site were probably unrelated. The four-post structure is interpreted as a potential shrine (Phase E).

One of the exhumed graves (F746) of Phase E, found under the Anglo-Saxon tower, had a voided rectangular socket in the fill at its west end (F748). This had the appearance of being the setting for a timber 'headboard', 58 cm wide and 4 cm thick.

Eighteenth- and nineteenth-century burials inside the church were generally associated with ledger slabs, and many interments in the graveyard had once been provided with headstones. Some of the latter had been further defined by kerbs, and a few had been surmounted by chest-tombs. Virtually all of this outdoor evidence was swept away in or before 1967. Nevertheless, in several instances the sockets for headstones were identified during excavation, and the brick footings for six chests were uncovered.

Multiple burials and posture

The great majority of graves of all periods consisted of a single interment, laid supine, with the legs outstretched, and the hands placed either to each side of the body, or together on the stomach. One multiple grave was exceptional in that two adult males and three children were interred together in a single rectangular pit (sk. 1909–1913, F5032; Fig. 11). This grave dated from Phase E. At the east end of the nave lay a pair of older children (sk. 339 and 340) in a single grave (F1524) of Phase C (Fig. 12).

Other multiple graves generally comprised one adult and a baby, although in the instance of skeleton 584 the adult was accompanied by two infants in the same grave (sk. 560 and 561, F1627). Apart from the two cases where a woman was carrying a fetus (sk. 2145/2146), there were numerous instances of infant bones in adult graves. Often, these were disarticulated and so fragmentary that it was impossible to confirm that an infant had deliberately been placed in the grave with an adult, however likely that may be. In other instances, the articulated skeleton of an infant, or young child, was found to overlie that of an adult. In most cases this was probably not a multiple burial, but a secondary insertion even though a separate grave-cut for the infant could not be detected.

Fig. 12: Two adolescent children (skeletons 339 and 340) interred in the same grave in the nave of the church. Scale of 75 cm. Photo: Warwick Rodwell

Variations in posture, beyond the normal range mentioned, were rare. A few examples of legs flexed at the knees were noted, and there were two cases of the legs being crossed at the ankles. One was a priest's burial (sk. 228, F1186; Fig. 13). A single instance of double-crossed legs also occurred (sk. 1787, F3933): there, both the femora and the tibiae were crossed, a configuration which is of course unnatural, and can only have been achieved during decay. The corpse was presumably buried with the knees together, and raised somewhat towards a vertical position. As decay took place, the left knee fell towards the right, and the right knee towards the left.

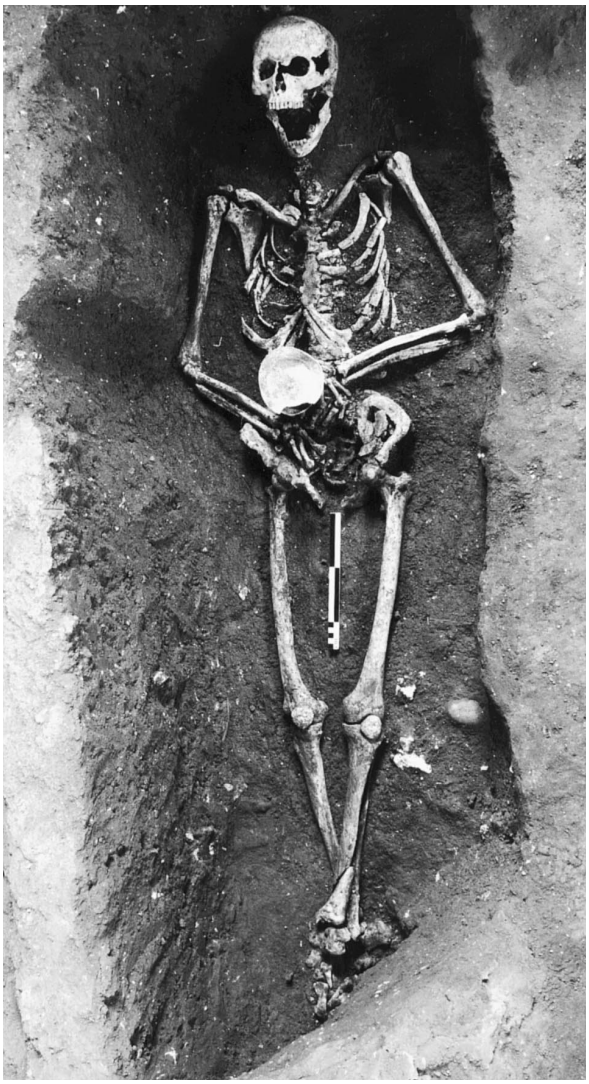


Fig. 13: Uncoffined burial (skeleton 228) of a priest inside the south porch of the early thirteenth-century church. On his stomach he holds a pewter chalice and paten. Note also the crossed ankles. Scale of 25 cm. Photo: Warwick Rodwell

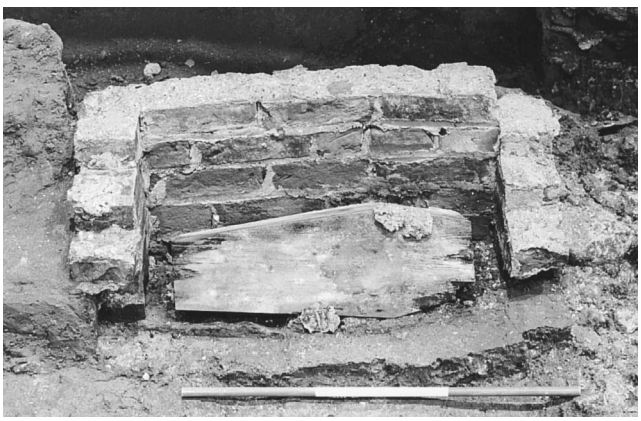


Fig. 14: Early nineteenth-century brick burial shaft (F284) in the nave, partly dismantled to reveal a child's elm coffin. The base had decayed and the coffin had fallen onto its north side. The lid, seen here on edge, is well preserved, although nothing but a few crumbs remained of the skeleton. Scale of 75 cm. Photo: Warwick Rodwell

Bone condition

Bone survival and condition was very variable. On the whole, the soil was favourable to preservation, but extremely localized factors within the cemetery played an important rôle. The best preserved skeletons were among the oldest, and were those contained in wooden coffins, buried below the water-table. There, the bones were usually in perfect condition, and were stained black (Fig. 19). The staining was almost certainly caused by tannic acid leaching out of the oak coffin boards: for a discussion of this phenomenon, see Rodwell 2001, chapter 15.

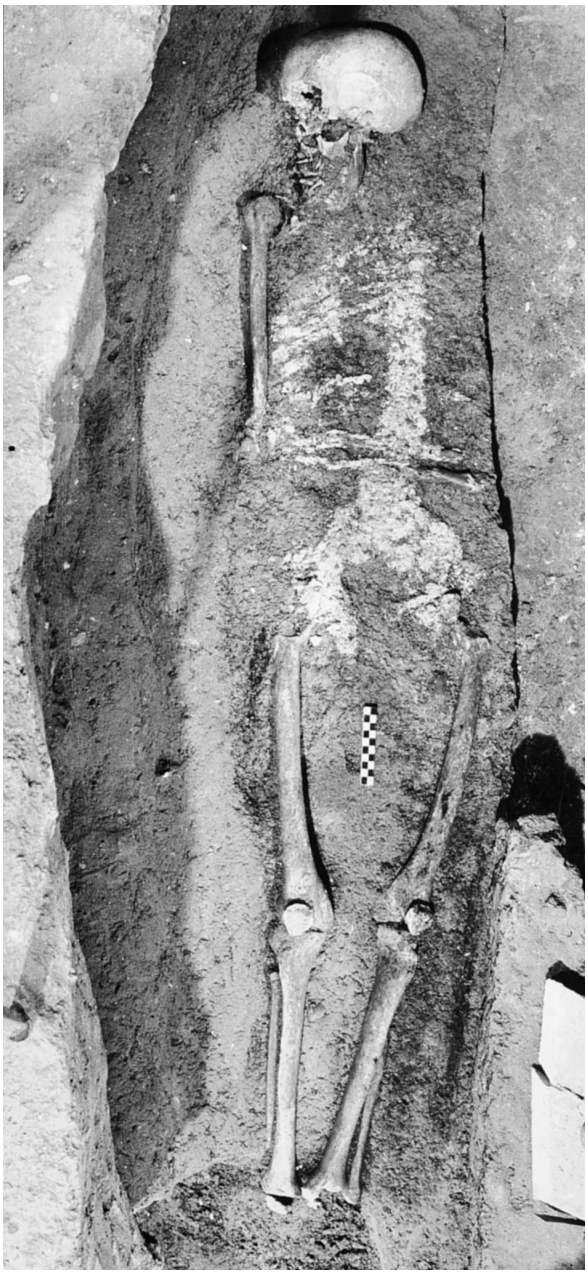


Fig. 15: Late Saxon interment (skeleton 637) in the chancel of the earliest church. The bones of the torso had decayed to a cream pulp, but the limbs and skull remained in relatively good condition. Scale of 10 cm. Photo: Warwick Rodwell

At the opposite end of the scale, the worst decay was encountered in post-medieval graves inside the church, where burial had taken place at a depth of around 60–75 cm below present floor level. In several instances, especially in children's graves, virtually all traces of bone had dissolved, even though remains of the elm coffin itself were sometimes preserved (Fig. 14). The bones of adults buried at the same level were usually soft and friable, sometimes having the consistency and appearance of a soggy biscuit.

The phenomenon of crumbling bone was not, however, wholly restricted either to post-medieval burials, or to those that were internal to the church. The torso was invariably the worst preserved part of the body (Fig. 15). For discussion of this condition, again see Rodwell 2001.

Poor bone preservation was also encountered in some early medieval graves where an unusual micro-environment had deliberately been created at the time of burial, in that the coffin had been filled with alluvial clay (p. 25; Fig. 22). In these examples it was usual to find that smaller bones had dissolved, and the ends of the long-bones were either missing or in a very poor state.

Forms of corporeal containment

In the past, there was a tendency for archaeologists to make a simple equation between the occurrence of numerous nails in a grave, and the former presence of a timber coffin; if only a few nails occurred, they would probably be regarded as 'stray' finds. The absence of nails was generally taken to imply that the burial was uncoffined. The excavations at Barton radically changed this perception, revealing a much more complex situation than was hitherto envisaged.

Waterlogging in the eastern part of the site gave rise to anaerobic conditions favourable to the preservation of wood (Fig. 16). Timber, in varying conditions, was found in more than forty graves, all dating from Phases D and E, but mostly the latter. In the best preserved examples, a complete coffin was found with the lid intact and the skeleton lying in a water-filled void. A small amount of mud may have seeped in. At the other end of the scale, a pulpy sliver from the side or base of a coffin may be all that survived.

Most of the coffins were parallel-sided or very slightly tapered, and made from six pieces of oak board. In one instance, these were held together solely with nails, but in all the others the principal jointing medium was the wooden peg. In a few cases one or two nails were also employed. It is clear, not only from the evidence at Barton but also in early cemeteries elsewhere, that iron was a semi-precious commodity in the late Saxon period and it was not used profligately: wooden pegs and dowels were cheaper than nails.[13] Although poorly preserved, several examples of a hybrid between a coffin and a bed or bier were found: the structure had solid timber sides, while the base was made from woven wattles. One burial took place in a dug-out coffin (sk. 1241, F3564).

Fig. 16: Two early Norman timber coffins preserved in waterlogged conditions to the north of the church. The nearer coffin (F5475) is of oak and contained an adult female (skeleton 2624); the further one (F5474) is of pine and contained a young child (skeleton 2623). Both coffins were constructed without the use of nails or other metalwork. Scale of 75 cm. Photo: Warwick Rodwell

Fig. 17: Child's pine coffin (F5474), containing skeleton 2623. It is held together with wooden dowels and pegs. Photo: English Heritage

In other cases, the grave cut was found to be lined with a miscellany of boards, some of which displayed evidence of previous use in a non-sepulchral context. Especially interesting among these was the occurrence of planks that had been lapped and joined on their long edges, using clenched nails and roves. These are diagnostic of clinker-built construction. Unfortunately, in no case was any significant amount of timber preserved, but lines of roves indicated that the planks ran for the full length of the grave. At first it was thought that coffins may have been constructed from narrow boards, lapped and clench-nailed, but that would have been a cumbersome process, and wasteful of nails and roves.[14] It is now appreciated that the graves which yielded roves were lined with sections of timber taken from clinker-built boats. Several other Anglo-Saxon cemeteries on the eastern seaboard of Britain have yielded roves, but their significance has not been appreciated until recently. The initial recognition that boat parts were used in the Anglo-Saxon period to line graves came at Caister-by-Yarmouth (K.A. Rodwell 1993).

All preserved coffins and other timbers at St Peter's were of oak, with the exception of a child's coffin that was made entirely of pine (F5474; Fig. 17).

The diversity of timber used in the containment and covering of burials is unlikely to have been correctly deduced had not part of the site been waterlogged. Traditionally, most of the pre-eighteenth-century burials at Barton would not have been regarded as coffined. It was instructive to compare the well-preserved evidence from waterlogged burials at the east end of the site with the contemporary 'dry' graves at the west end, where, for the most part, not a hint of former timber structures was detectable. Between these two extremes were the 'damp' graves, where coffin outlines were often preserved in the form of thin grey, clayey streaks in the soil. Sometimes these streaks contained tiny flecks of charcoal, indicating that charred boards had been used in coffin-making. This is a recurrent observation in the excavation of Anglo-Saxon cemeteries: see, for example, Hereford (Shoesmith 1980) and Wells Cathedral (Rodwell 2001).

Where no remains of timber, or silhouettes, survived in the ground other indicators of 'contained' burial were sought. The most obvious were lines of nails indicative of a conventionally constructed coffin. These, however, were scarce before the eighteenth century, when iron grips, breastplates and other accoutrements became fashionable. Numerous examples were found, dating from the later eighteenth century and the first half of the nineteenth (Phase A). Nailed coffins of earlier periods were few and mostly encountered inside the church, where they were of fifteenth-century and later date (Phases B and C).

It has long been recognized that large, squarely cut graves are indicators of coffined burial, and many examples of these were found, particularly in Phases D and E. Conversely, graves which were scarcely larger than the corpse, and had rounded ends or irregular sides, could not have received prefabricated coffins or timber linings. These occurred at all periods, but were least common in Phase A, when coffined burial was clearly the norm. Skeletons exhibiting a 'parallel-sided' effect are usually taken as indicators of the former presence of a tightly fitting coffin (Boddington 1987; Rodwell 2001). There were good examples of this phenomenon at Barton, sometimes accompanied by other indications of encoffinment (Fig. 18).

It was often possible to deduce the former presence of a contained burial as a result of post-depositional movement of bones within the grave, and Barton was one of the first sites where this phenomenon was studied in detail. Skeletal disorder has been noted, and discussed, in relation to several other excavated cemeteries, such as Raunds, Northamptonshire (Boddington 1987), Jewbury, York (Brothwell 1987),

Fig. 18: A late Saxon burial in a spacious grave. The skeleton (554) exhibits the 'parallel-sided' effect, apparently resulting from a snug fit in a coffin, although no trace of timber remained. The parallel lines, to either side of the legs, mark a slight trough in the floor of the grave. Two large pebbles, inside the presumed coffin, supported the skull ('ear-muffs'). Scale of 75 cm. Photo: Warwick Rodwell

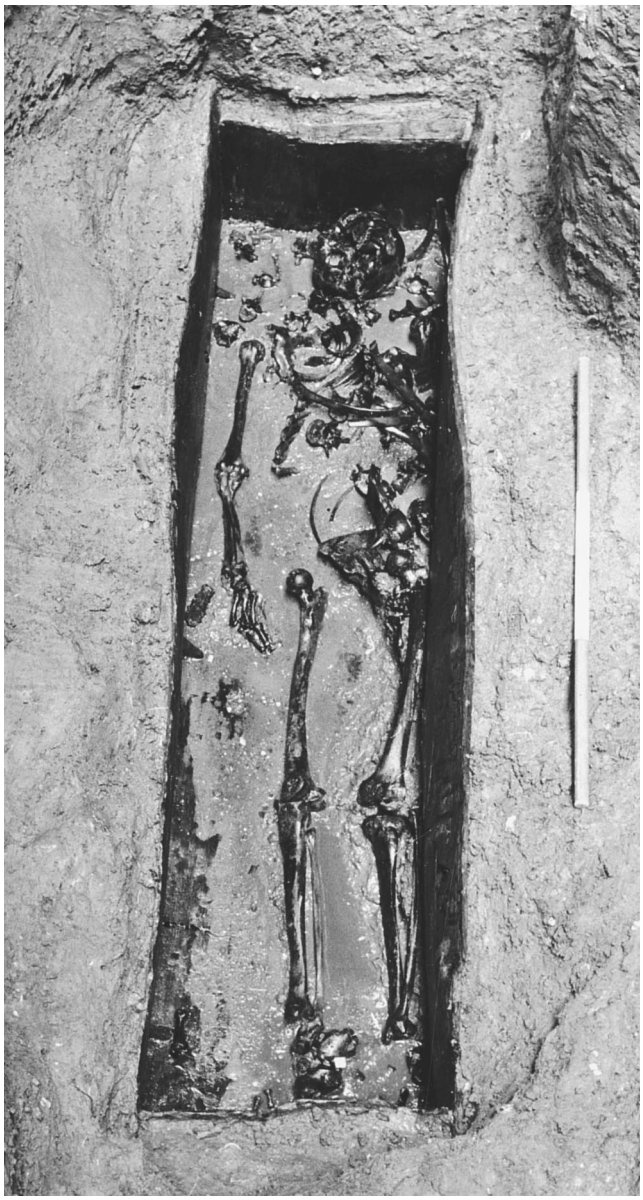


Fig. 19: Skeleton 776, as revealed when the lid was removed from this well-preserved and undisturbed early Norman coffin. The bones, which were in serious disarray, and stained black, were otherwise in perfect condition. Scale of 75 cm. Photo: Warwick Rodwell

and Wells Cathedral (Rodwell 2001, chapter 15). When a corpse is surrounded by loose soil, very little bone movement is possible: as the flesh decays the soil above sinks down and the skeleton is trapped. But when the corpse decays in a void, considerable movement of bones is possible, and commonly occurred. Relaxation of the lower jaw is not, on its own, a reliable indicator of contained burial. However, rolling of the skull, axial rotation of limbs, and the jumbling of ribs and vertebrae, are all definitive indicators of corporeal decomposition within a void. Again, it was fortunate that post-depositional movement could be studied inside fully preserved coffins as well as in burials where the container had decayed and soil had subsequently enveloped the skeleton (Figs. 19, 20 and 21).

For the sake of completeness, it should be mentioned that no medieval stone coffins were uncovered in the excavations, although the emplacement for one was found in the south-west corner of the nave (F80). Undoubtedly, there would have been interments in stone coffins in the thirteenth-century and later chancels (unexcavated). It is relevant to note that the buttresses on the south side of St Peter's church contain several blocks of oolitic limestone which have clearly been produced by cutting up medieval stone coffins. This is not an uncommon phenomenon. Several decorated grave-covers – potentially the lids from these same coffins – were reused in the foundations of the arcade piers in the late thirteenth-century south aisle.

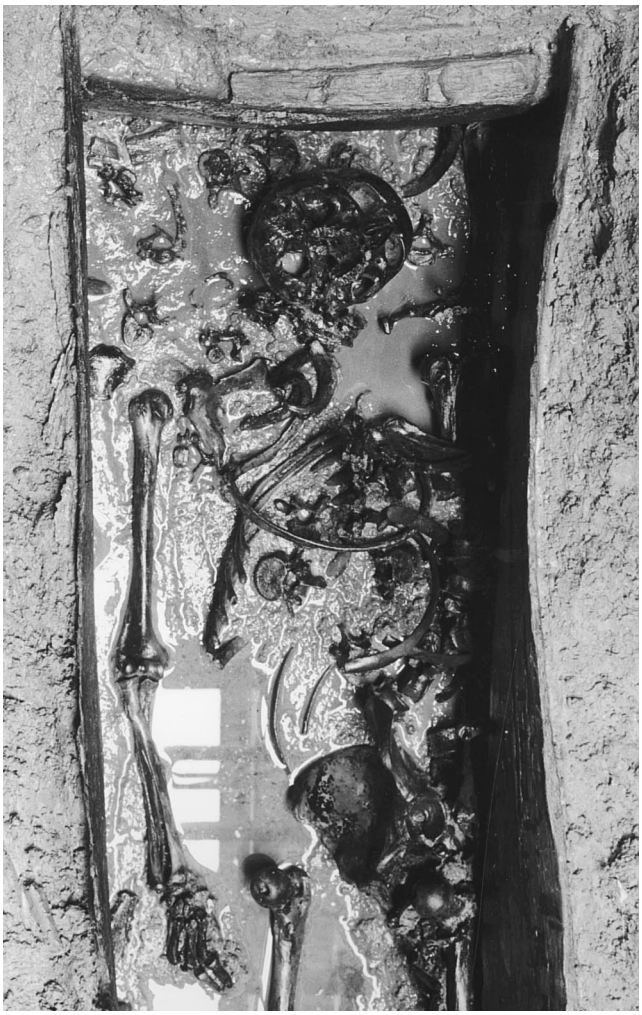


Fig. 20: Detail of the upper part of skeleton 776. Although the right arm and hand remained in articulation, the rib-cage and vertebrae were in complete disarray, many bones having moved considerable distances from their correct positions. Photo: Warwick Rodwell

Fig. 21: Detail of the lower part of skeleton 776. The left leg remained in its correct position, but the entire right leg had rotated axially through 180°. Photo: Warwick Rodwell

Interments of the later eighteenth and nineteenth centuries at Barton were commonly in elm coffins, the boards of which were held together with iron nails. Iron grips were provided and there was often a thin sheet-iron breastplate affixed to the lid and upon which the name and other details of the deceased were recorded in white paint. Decorative 'lace' edgings of pressed tinplate were also provided in some cases. The grips usually survived in the soil, but very rarely did much else. Indoor burials in brick vaults were better furnished, and some had tinplate mounts. However, no copper or brass nameplates and no brass grips were found among the excavated burials. Their omission from coffins of the middle classes in the nave is slightly surprising, but better furnished burials may, of course, occur in the chancel.

Lead shells inside elm coffins were present in several of the vaults and brick-lined shafts, but these were mostly left undisturbed, and therefore their details are not recorded (Fig. 7).

Encapsulation of the corpse

Charcoal-filled graves are a well known phenomenon in the late Saxon period, although the purpose of encapsulating the corpse in this material is disputed. It was almost certainly not related primarily to the containment of disease, but was a symbol of status: charcoal is most commonly found in prestigious burials. A single charcoal burial (sk. 1215, F3234) was found at Barton (Phase E), lying outside the south door of the Anglo-Saxon turriform church. It was unusual in that the torso had been coated with a layer of sticky blue-grey clay, before charcoal was poured over the corpse.

Controlling the spread of disease is the most plausible explanation for the appearance of alluvial clay inside coffins: these were colloquially referred to as 'clay burials'. Although no example was found where the coffin timber was preserved, it is clear that liquid mud was poured into the coffin, completely encapsulating the corpse (Fig. 22). Two groups of burials exhibiting this feature were encountered at St Peter's, both assigned to Phase E.

Fig. 22: Phase E burial in a coffin which was filled with liquid clay, completely encapsulating the corpse (skeleton 957). Nothing remained of the coffin, but its outline was well defined by the clay filling. Photo: Warwick Rodwell

Fig. 23: Skeleton 204, buried inside the church. The head and shoulders were covered with lime. Although no trace of the coffin remained, the outline of its west end was clearly indicated by the crisp edge to the lime filling. Photo: Warwick Rodwell

One group lay to the north of the tower. Here, some thirty burials were found to have bluish-grey clay enveloping the skeleton, and most of the graves yielded firm evidence of a coffin outline. The clay was always contained within that outline. Since some of the graves were cut into one another, the burials were obviously not strictly contemporaneous, and cannot therefore be regarded as linked to a particular epidemic.

The second, close-knit, group of 'clay burials' was found within the south aisle of the present church, but had originally lain outside the Norman nave. There were seven, and all had been coffined.[15] Here, either a grey or a yellowish-brown alluvial clay was used to encapsulate the corpse. The exact sources of the two types of alluvium have not been traced, but they were local: similar clays could be obtained from the marsh to the north of St Peter's, or from the Beck.

An oddity that might be related to containing disease was the use of lime in a Phase D burial (sk. 204, F1164) in the south aisle of the church. Lime, probably in powder form rather than wet, had been poured over the head and shoulders of the corpse, after it had been placed in the coffin (Fig. 23). The latter was not preserved, but the outline of its west end was crisply defined by the lime. In more recent times, quicklime has been used to cover human and animal corpses infected with anthrax.

Furnishings and accoutrements

Being an entirely Christian cemetery, grave-goods in the conventional sense were not present at St Peter's, thus drawing a sharp contrast with the majority of the Castledyke burials. There were, however, various inclusions in graves which are of interest.

Several of the Phase E coffins contained long sticks, or 'wands', made of willow/poplar. No more than two were present: they could be placed on the floor of the coffin beside the corpse, or one might be laid centrally on top of it. It is generally believed that wands had a

Fig. 24: The west end of a well-preserved coffin (F3968), after removal of the lid, showing the ghost of a grass-filled pillow that supported the skull of skeleton 1863. Photo: Warwick Rodwell

Fig. 25: Skeleton 107 of early nineteenth-century date, buried in the north aisle of the church. The feet lay on a white china plate which had been placed inside the coffin. Scale of 25 cm. Photo: Warwick Rodwell

magical significance, and were thus a quasi-pagan inclusion. They have occasionally been recorded in Anglo-Saxon and early graves in Britain, and are well known in waterlogged Scandinavian burials. One of the latest observed occurrences is in an early fourteenth-century priest's burial at Lichfield Cathedral (Rodwell 2004).

The presence in a grave of a pair of stones providing lateral support for the skull is a widespread phenomenon in middle and late Saxon cemeteries; they are usually referred to as 'ear-muffs'. One or more stones placed under the skull ('pillow-stones') are equally common. Both arrangements may be found in graves where there is no evidence for a coffin, and also in those where there probably was one. At Barton, however, ear-muffs were a feature associated mainly with coffined burial of Phase E. One of the intact, waterlogged coffins (F3968) provided evidence for their exact mode of deployment: in this instance the stones were not wedged either side of the skull, but were supports for a pillow made of organic materials. The form of the grass-stuffed pillow was preserved, although none of the covering material remained (Fig. 24). In the case of grave F5402, an object which was almost certainly a rolled-up mattress had been dumped in the grave directly on top of the coffin, presumably because the person had died of a contagious disease.

The status of two medieval burials was proclaimed by the presence of eucharistic vessels (F1186, 4115). This pair of priests had been interred in the primary south porch in Phase D, each accompanied by a mortuary chalice and paten (Fig. 13). The majority of medieval priests' graves will have been in the chancel, but the practice of porch burial is well attested. A burial on the north side of the nave yielded a fourteenth-century bronze crucifix, which was not on the corpse, but seems to have been thrown into the grave (F449) during backfilling. Close by was a grave (sk. 198, F325) containing fragments of silk and gold textile.

Fig. 26: Skeleton 258, an early to mid-nineteenth-century outdoor burial of a male wearing a leather hernia truss. Scale of 25 cm. Photo: Warwick Rodwell

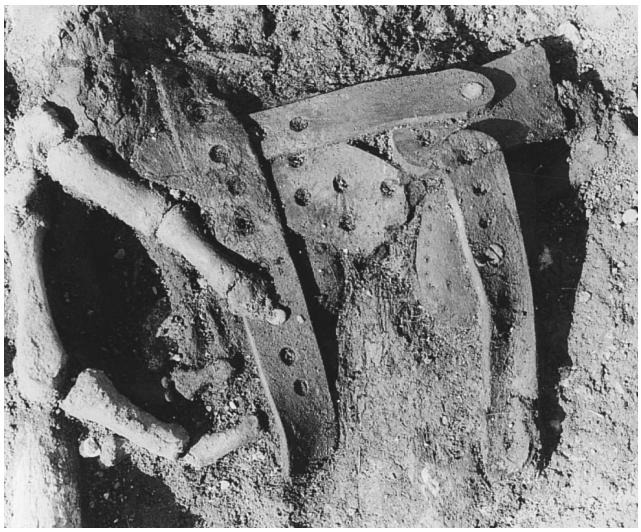


Fig. 27: Detail of the hernia truss on skeleton 258. The various components were adjustable and held together with brass studs. Beneath the leather were a few wisps of pubic hair, the only instance of its survival on the site. Photo: Warwick Rodwell

Fig. 28: Skeleton 219, which had undergone a post mortem *investigation before being buried outside the church to the south-west. Note the charred stake inserted in the torso as a substitute for the vertebral column. Scale of 25 cm. Photo: Warwick Rodwell*

Fig. 29: Detail of the skull of skeleton 219, showing the saw-cut which had removed the vault. The two parts had been reunited, and glued, for burial. Photo: Warwick Rodwell

Deliberate inclusions were recovered from several nineteenth-century graves: thus one in the north aisle had a white china dinner plate under the feet of the corpse (sk. 107, F365; Fig. 25), and one in Area 8 had a blue-and-white transfer-decorated china bowl under the coffin, beside the skull (sk. 174, F3027). At least two nineteenth-century burials seem to have been accompanied by the deliberate inclusion of a coin in the grave, a practice reminiscent of pagan burial in the Roman period. In the late Saxon grave F3548 a third-century Roman coin had been laid on the left shoulder. In grave F3022 (sk. 156) a George III halfpenny lay on the left humerus, and five other burials contained Georgian coinage, either accidentally or deliberately. A bronze finger-ring was found on skeleton 265 (also Phase A). An identifiable female, Margaret Swallow, buried in 1845 (sk. 1450, F3697) was accompanied by a gold wedding-ring.

Medical conditions of a non-skeletal nature were revealed in two nineteenth-century burials in Area 8 (sk. 257, F3337 and sk. 258, F3041), both of which were accompanied by hernia trusses made of leather (Figs. 26 and 27). Another yielded trouser buttons. Nearby lay the only burial on the site to display evidence of having been subjected to a *post mortem* examination, a male aged over 45 years (sk. 219, F3033; Figs. 28 and 29); see further, p. 38. The vault of the cranium had been sawn off, and glued back again for burial. The whole of the rib-cage and vertebral column had been removed, doubtless with all the organs inside, leaving an envelope of skin which was then stuffed with vegetation. A charred timber stake was substituted for the spine, to stiffen the torso for a seemly burial.

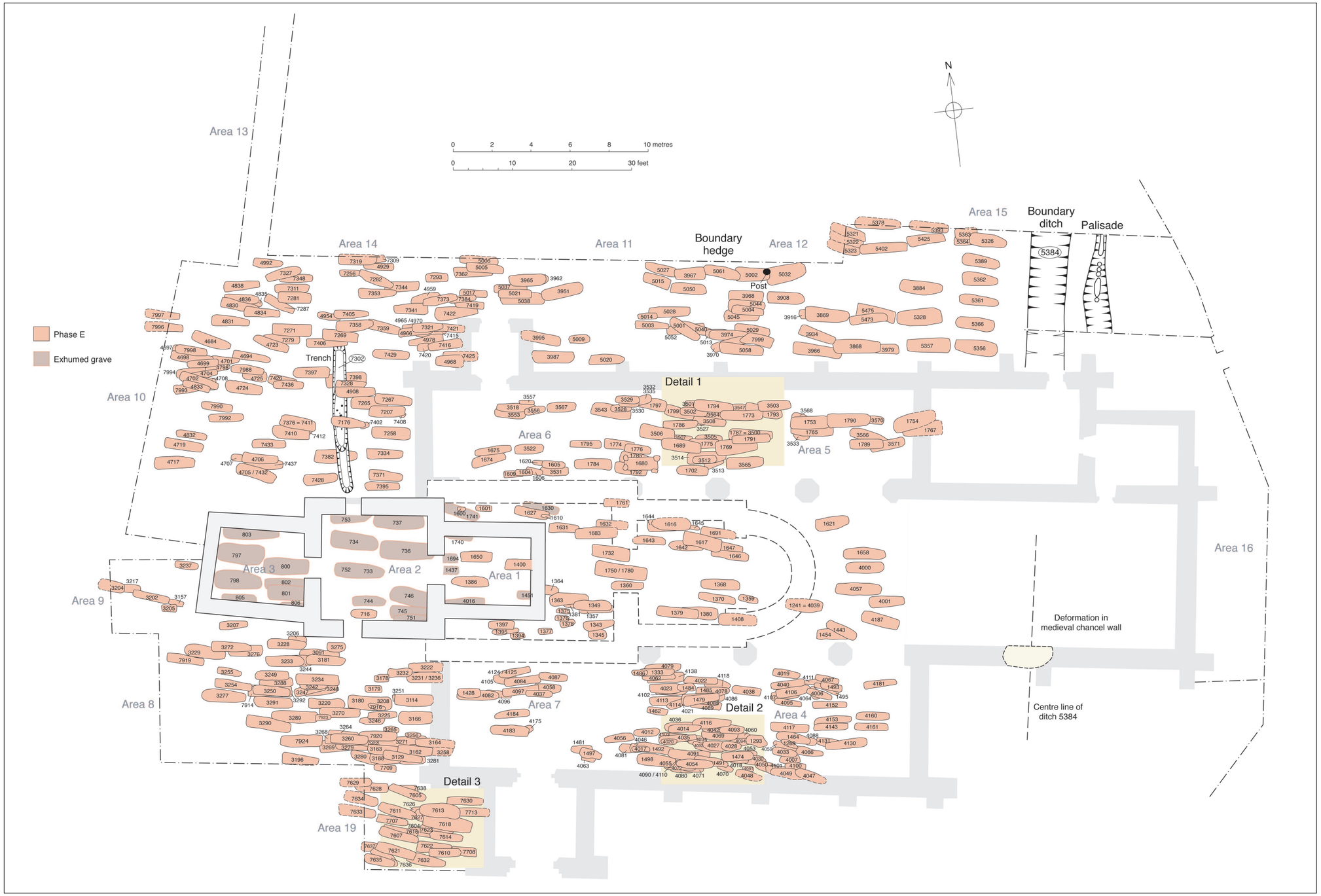


Fig. 30: General plan of the excavated graves attributed to burial Phase E. The notation given refers to the feature numbers of the graves, not the skeleton numbers. Scale 1:200. Drawing: Simon Hayfield

Cemetery Chronology

On the south, the west and, to a lesser extent, on the north, repeated reuse of the same spot led to the intercutting of graves to such an extent that many were extremely fragmentary, and disarticulated bones were abundant in backfills. Nevertheless, collectively, hundreds of stratigraphic relationships could be established within small groups of burials, providing a series of 'cemetery yardsticks'. Indications of the approximate numbers of 'burial generations' in individual localities could thereby be obtained from the matrices that were drawn up to illustrate stratigraphic relationships. Typically, up to a dozen burial generations could be counted in these sequences.

Establishing relative chronologies, through localized grave sequences, is one thing: determining absolute dates for individual burials is another. The occurrence of datable and relevant artefacts in graves – as opposed to residual and irrelevant inclusions – was extremely rare: the eighteenth and nineteenth centuries fared best in this respect. The notable exception was a group of eleventh- and early twelfth-century burials in the north-eastern part of the site, where timber coffins had been preserved because the ground was waterlogged. It was possible to date 31 of these coffins through dendrochronology, in some instances to a precise year: full details are given in Volume 1. Three more were dated, less precisely, by radiocarbon.

This information obtained from the coffins was augmented by a substantial programme of radiocarbon dating on skeletons, which was directed primarily towards the earlier phases of the cemetery. In all, 75 skeletons were dated by this means, and the results collectively analysed (see Volume 1). The conclusions pointed to the origins of both the Anglo-Saxon church and cemetery being later than was previously supposed.

Phasing

It is impossible to create an overall burial matrix for the excavated area which was, in any case, physically compartmented by the deep foundation trenches of both the existing church and its predecessors. There were also *lacunae*, where few or no burials had taken place, and thus stratigraphic connections could not be established. Consequently, for the purposes of overall analysis and discussion, it was decided to subdivide the history of the cemetery into five basic chronological phases, each embracing roughly two centuries.

Phase E (Anglo-Saxon and Norman)	— *c.* 950–1150
Phase D (early medieval)	— *c.* 1150–1300
Phase C (late medieval)	— *c.* 1300–1500
Phase B (early post-medieval)	— *c.* 500–1700
Phase A (Georgian and Victorian)	— *c.* 1700–1855

In a significant number of instances there was insufficient evidence to place a grave firmly within one particular phase, as opposed to the next: in such cases, the burial is designated as Phase A/B, B/C, etc. Within these parameters, it has been possible to assign about 80% of the excavated burials to a single or combined phase (see Table 1, Chapter 3).

Phase E burials (late Anglo-Saxon and Norman) (Figs. 30 and 31)

As noted in the previous chapter, the earliest known church on the site was erected within an existing cemetery, in the late tenth or early eleventh century. That cemetery lay on a small and reasonably well defined spur of land to the west of the large sub-circular enclosure of middle Saxon date, in which the manor house of Barton (Tyrwhitt Hall) was later built.

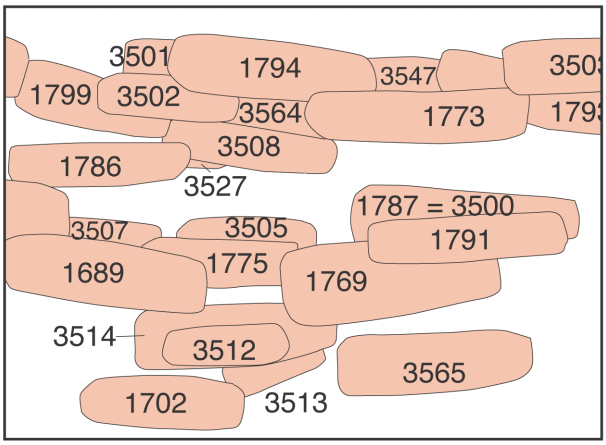
There is reasonable evidence to show that some graves were arranged roughly in rows, while others were more scattered. Towards the eastern part of the site lay a boundary ditch (successor to the middle Saxon enclosure) which defined the limits of burial. The earliest burials were uncoffined and had no distinguishing features. The absence of coffins or other timber constructions in these graves could be demonstrated emphatically on the waterlogged eastern part of the site, where second-generation graves with well-preserved coffins overlay or cut into the primary burials.

Coffins and timber grave-linings appeared early in the history of the cemetery, but did not entirely supersede uncoffined burial: the two types were found stratigraphically interleaved. A potential indication that coffined burial had become the norm by the end of the tenth century is found in a group of graves sealed under the earliest church. Here, at least 16 of the 29 graves were identified as likely to have held coffins, but their contents had been cleanly exhumed immediately prior to the erection of the late Saxon building.[16] These graves were mostly under the tower and western annexe. Other lost graves may be inferred along the lines of the foundations.

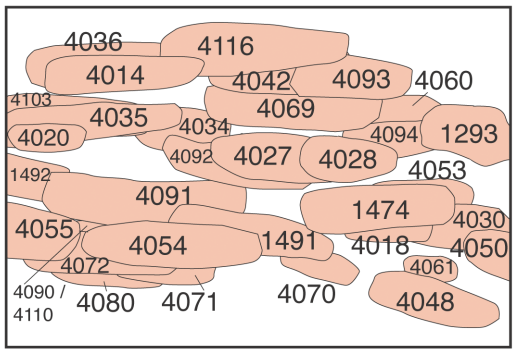
The exhumation was a cleansing operation, the intention of which was to remove all corporeal remains – and thus potential corruption – from the site of the new church. The process of cleansing the ground prior to construction is recorded in other Anglo-Saxon and medieval contexts. The emptied graves contained no human bones, and it is virtually inconceivable that such clean exhumations could have been carried out unless the corpses were encoffined. Some of the small bones of the hands and feet invariably get overlooked when decomposing, uncoffined interments are exhumed. Also, the size of some of the graves was such as to imply the presence of a coffin. Significantly, one of the burials which was overlooked during the cleansing was that of an uncoffined child (F716): it remained undetected beneath the floor of the turriform nave.

A second unexhumed grave seems to have been uncoffined too (F1364), and it was truncated by the east wall of the new chancel.

The overall distribution of graves assigned to Phase E is given in Fig. 30, which also shows the outlines of the Anglo-Saxon turriform church and the later eleventh-century apsidal building that superseded it. The great density of interments to the south of both churches is immediately apparent, but there were also significant concentrations of graves on the north. It must be stressed that most of the areas which appear devoid of burials on this general plan (and on others which follow in Figs. 32–36) were taken up by foundations or vaults. There are, however, a few restricted areas where burials were genuinely sparse (*e.g.* around the western annexe), and some of these may ghost impermanent features such as trees, or even timber structures. Thus, it can be seen that relatively few, but moderately well ordered, graves were dug on the western part of the site prior to the erection of the first church. The area of exhumations provides a convenient bench-mark for this early phase.



Detail 1



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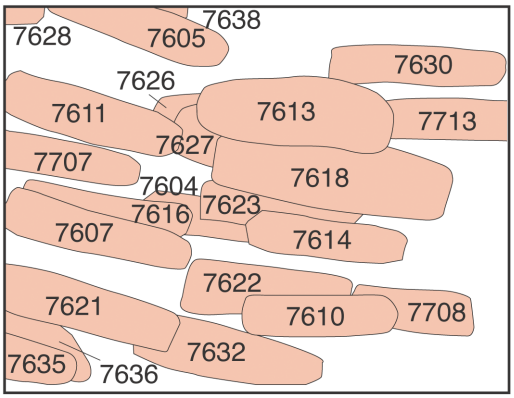


Detail 3

Fig. 31: Enlarged detail plans of three areas of dense burial in Phase E: see Fig. 30. Drawing: Simon Hayfield

The eastern limit of the cemetery is readily apparent, and there is clear evidence of at least four orderly rows of graves, running up to the Saxon boundary ditch. The approximate western limit may be inferred from a diminution of the number of burials at the very edge of the excavation in Areas 8–10. The east–west dimension of the cemetery was thus *c.* 45 m. The northern edge clearly lay beyond the limit of excavation in Areas 11, 12 and 14, but was perhaps not much further removed on the evidence of the lack of burials in Area 13 attributable to Phase E (although a few are possibly D/E). The southern limit is unknown, but did not extend beyond the footpath which forms the historic churchyard boundary, and consequently the north–south dimension was probably *c.* 50–55 m. Thus a maximum area of *c.* 2,475 m² is indicated, or somewhat more than half an acre.

Finally, it may be noted that only three internal burials took place within the life of the turriform church, all in the chancel (F1386, 1400, 1650). This accords with Anglo-Saxon religious practice, which eschewed burial within the body of the church, but allowed it inside appendages.[17] Likewise, very few of the graves recorded within the footprint of the Saxo-Norman church are likely to have been internal, the majority clearly antedating its construction. One grave that certainly was internal was a coffined burial (F1750) which was placed centrally under the chancel arch. This prominent location indicates an important burial, and it was doubtless for that reason that the coffin was exhumed when the church was again extended eastwards. The burial was probably translated to the new chancel.

Phase D and D/E burials (early medieval) (Fig. 32)

Burials of these phases were associated with the long-naved Norman church in both its unaisled and narrow-aisled forms, and also with the later thirteenth-century wide-aisle phase on the south.

Internal burials were present but not numerous. A cluster of four was identified on the south side of the nave (F1184, 1206, 1220, 1332), and a single,

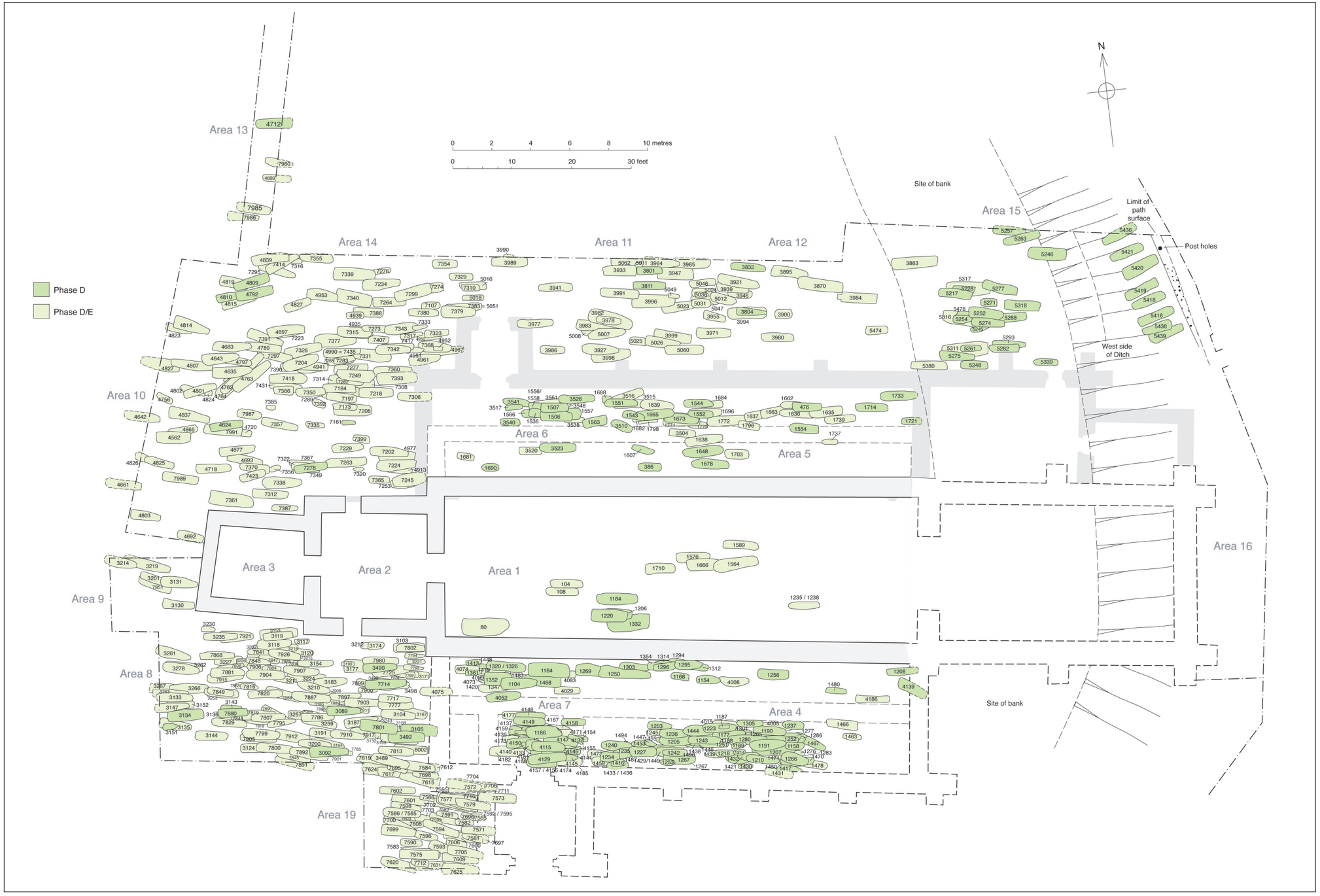


Fig. 32: General plan of the excavated graves attributed to burial Phases D and D/E. The notation given refers to the feature numbers of the graves, not the skeleton numbers. Scale 1:200. Drawing: Simon Hayfield

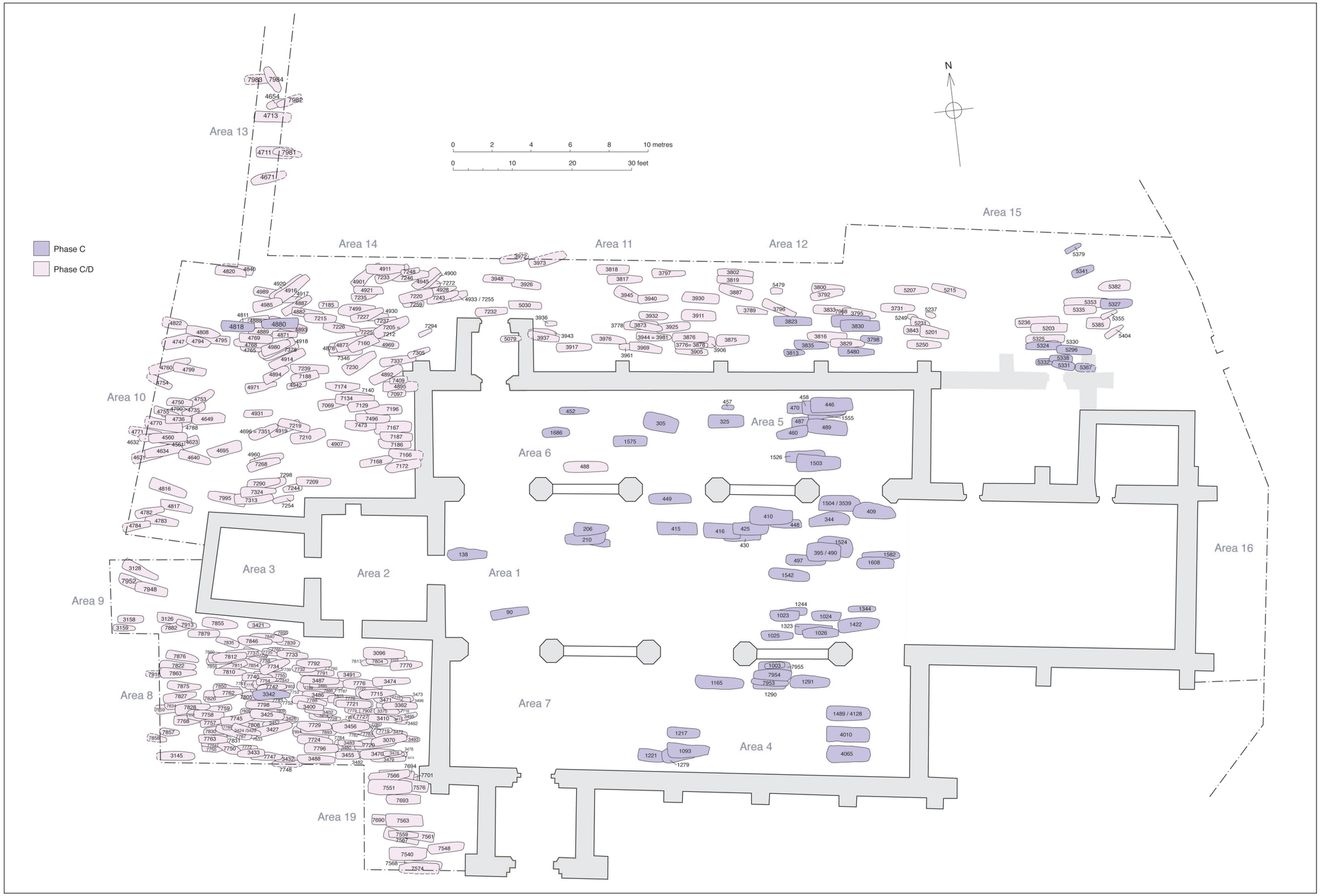


Fig. 33: General plan of the excavated graves attributed to burial Phases C and C/D. The notation given refers to the feature numbers of the graves, not the skeleton numbers. All graves shown within the walls of the church were internal burials. Scale 1:200. Drawing: Simon Hayfield

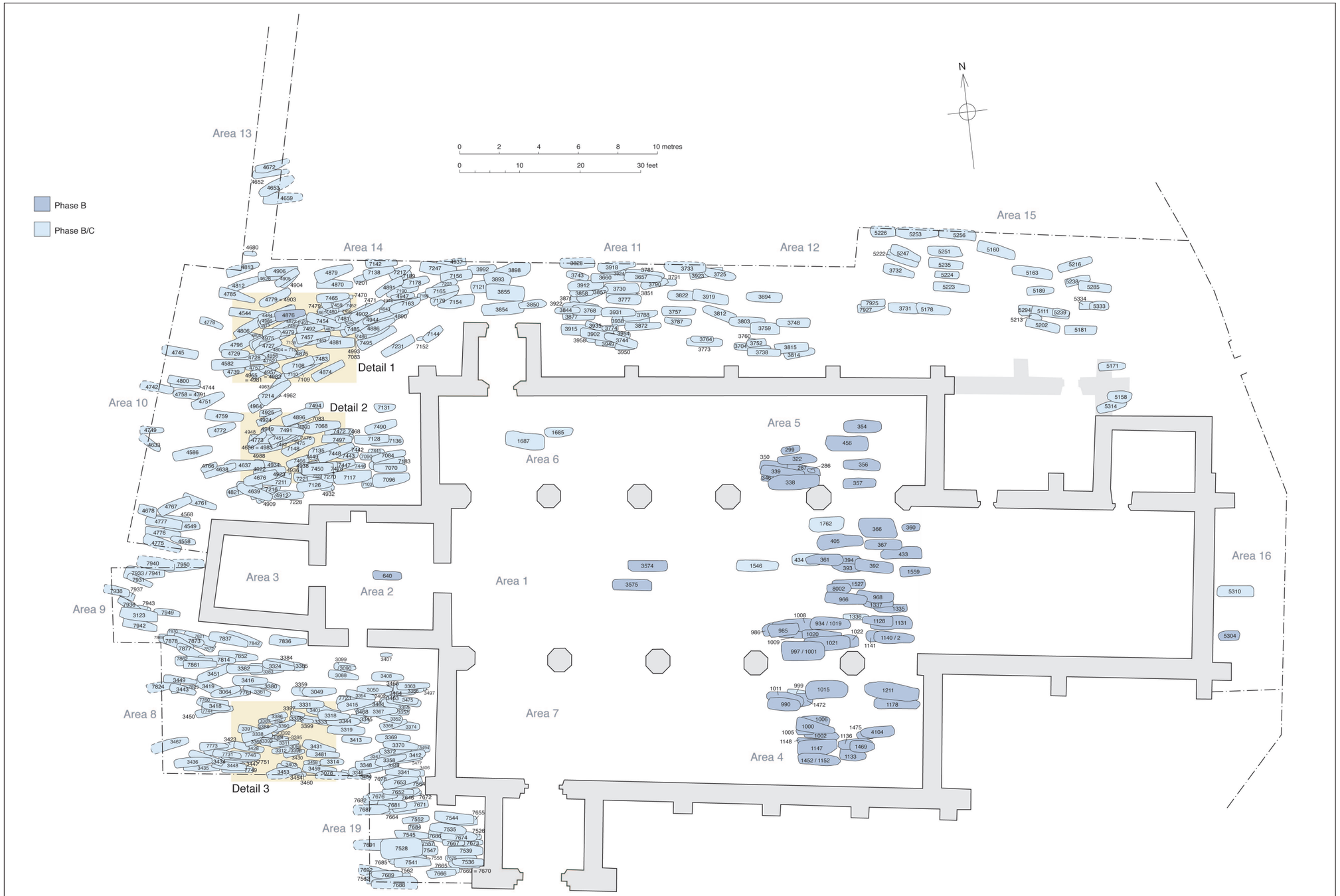


Fig. 34: General plan of the excavated graves attributed to burial Phases B and B/C. The notation given refers to the feature numbers of the graves, not the skeleton numbers. All graves shown within the walls of the church were internal burials. Scale 1:200. Drawing: Simon Hayfield

exhumed grave occurred in the south-west angle (F80). The latter, it seems, had originally contained a stone coffin. A line of graves had been dug along the western half of the narrow south aisle, and a family group may be represented by some of these (F1250, 1269, 1294–96, 1303, 1312). Another group, comprising two adults and two babies, lay just inside the threshold of the south door (F1164, 1468), with two further burials to the west (F1104, 1352).

The south porch attached to the narrow aisle contained three burials side-by-side (F1186, 4115, 4129); in addition, the central grave partially overlay an earlier one on the same site (F4133). The two northernmost (F1186, 4115) were both priests' burials; the former contained skeleton 228 (Fig. 13). Hard against the wall in the north aisle was a single grave (F3523) and, further east, a pair (F1648, 1678).

Outside, to the south and south-west, lay a great concentration of burials, with much intercutting. To the north of the church they were somewhat sparser and in Areas 10/14, in particular, difficulty was experienced in separating out graves of Phases D and E, respectively. A greater diversity of alignment was also evident here, perhaps indicating the influence of paths within the churchyard. To the north-east, however, were two discrete groups of burials, one of which lay in the backfill, and followed the line, of a Norman defensive ditch. For discussion of Barton's defences, see Volume 1.

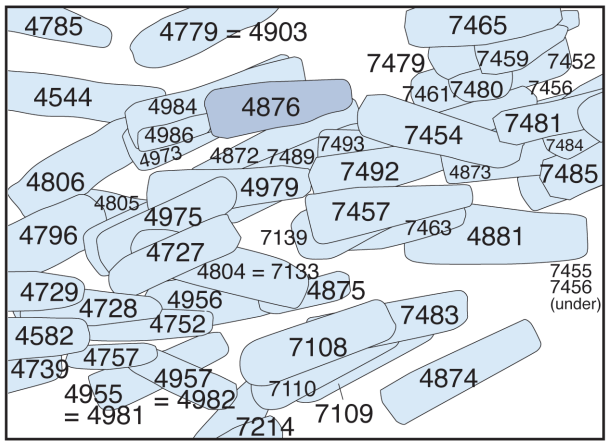
Phase C and C/D burials (late medieval) (Fig. 33)

The disposition of fourteenth- and fifteenth-century graves in the nave and aisles of the present church is well evidenced on the plan, and conforms to expectations. A line of interments running down the central axis of the nave has almost certainly been eradicated by later, deeper graves, but otherwise the picture is probably fairly complete. In the nave, the principal concentration was in the easternmost bays, in front of the rood screen, and at the east end of each aisle a north–south row of graves lay immediately in front of the altar. The arrangement in the south aisle was particularly neat (F1489, 4010, 4065). Graves which were close to and more-or-less centred on an arcade bay may point to the presence of monuments under the arches (particularly F449, 1504/3539). Archaeological evidence was also found for late medieval pew enclosures in the nave, associated with particular burials.

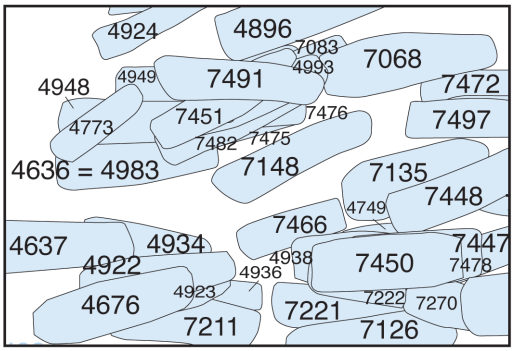
Outside the church, the major concentration of burials was again to the south-west, and doubtless many more continued all along the unexcavated south side too. The sparser use made of the north side better enables small groupings of potentially related graves to be recognized. The skewed and disparate alignments of graves to the north-west of the church, noted in the previous phase, is found also in this one; and the phenomenon continued into Area 13.

Phase B and B/C burials (early post-medieval) (Figs. 34 and 35)

The internal burials of this phase were almost entirely confined to the two easternmost bays of the nave and aisles, although not of course related to the rood



Detail 1



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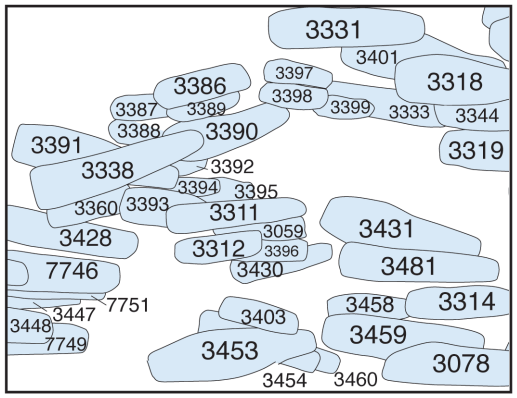

Detail 3

Fig. 35: Enlarged detail plans of three areas of dense burial in Phases B and B/C: see Fig. 34. Drawing: Simon Hayfield

screen or altars, which had all been expunged at the Reformation. More likely, the ends of the aisles were colonized by important Protestant families, who would have erected funerary monuments where the altars once stood. The Nelthorpe family adopted the two eastern bays of the south aisle for their burials. The almost complete lack of graves in the centre and western parts of the church suggests that there was extensive pewing here, although the Gelder family constructed a vault in the south-west corner. A single interment occurred in the base of the tower (F640).

Outside, tightly packed burials filled the south-west angle between the aisle and tower, and another dense block extended northwards from the tower. Once again, the skewed alignment in the latter area is pronounced, and the course of a path around the north-west corner of the church is clearly ghosted. It is interesting to observe that burials on the north side of the church, together with those south of the tower, did not crowd against the walls. Also, graves were sparsely distributed to the north of the chancel.

Two further external burials are of potential significance, one being axially sited just beyond the east wall of the chancel, in an area not otherwise used as cemetery (F5310), while the other lay a little to the south (F5304).

Phase A burials (Georgian and Victorian) (Fig. 36)

By the eighteenth century, the popularity of indoor burial had increased so much that space commanded a significant cash premium. Moreover, the interior of the church was filled with box pews, greatly restricting the available space. This is well demonstrated by the tightly packed graves running down the central alleys of the nave and both aisles. A few interments were contained in brick-built vaults or shafts (Figs. 7 and 14). The font stood axially at the west end of the nave, and straggling lines of graves ran north and south from it, along the cross-alley. In the early nineteenth century the organ stood at the east end of the north aisle, and a group of children were buried behind it, a fact which was retained in local memory and confirmed archaeologically.

Burial in the churchyard was characterized in Phase A by neat rows, particularly to the north and south of the tower. However, the diagonal path across the north-west corner of the churchyard continued to exercise a powerful influence on grave alignment in that area. Another path, which snaked its way eastwards, from the north porch to the gate in the boundary wall, was also clearly reflected in the distribution and, to a slight extent, in the alignment of graves. Burials entirely avoided the footpath running alongside the eastern boundary (Areas 15–16).

A few brick vaults and burial shafts were constructed in the churchyard in the early nineteenth century, but the only one investigated was a shallow shaft just east of the north porch. It belonged to the Goy family.

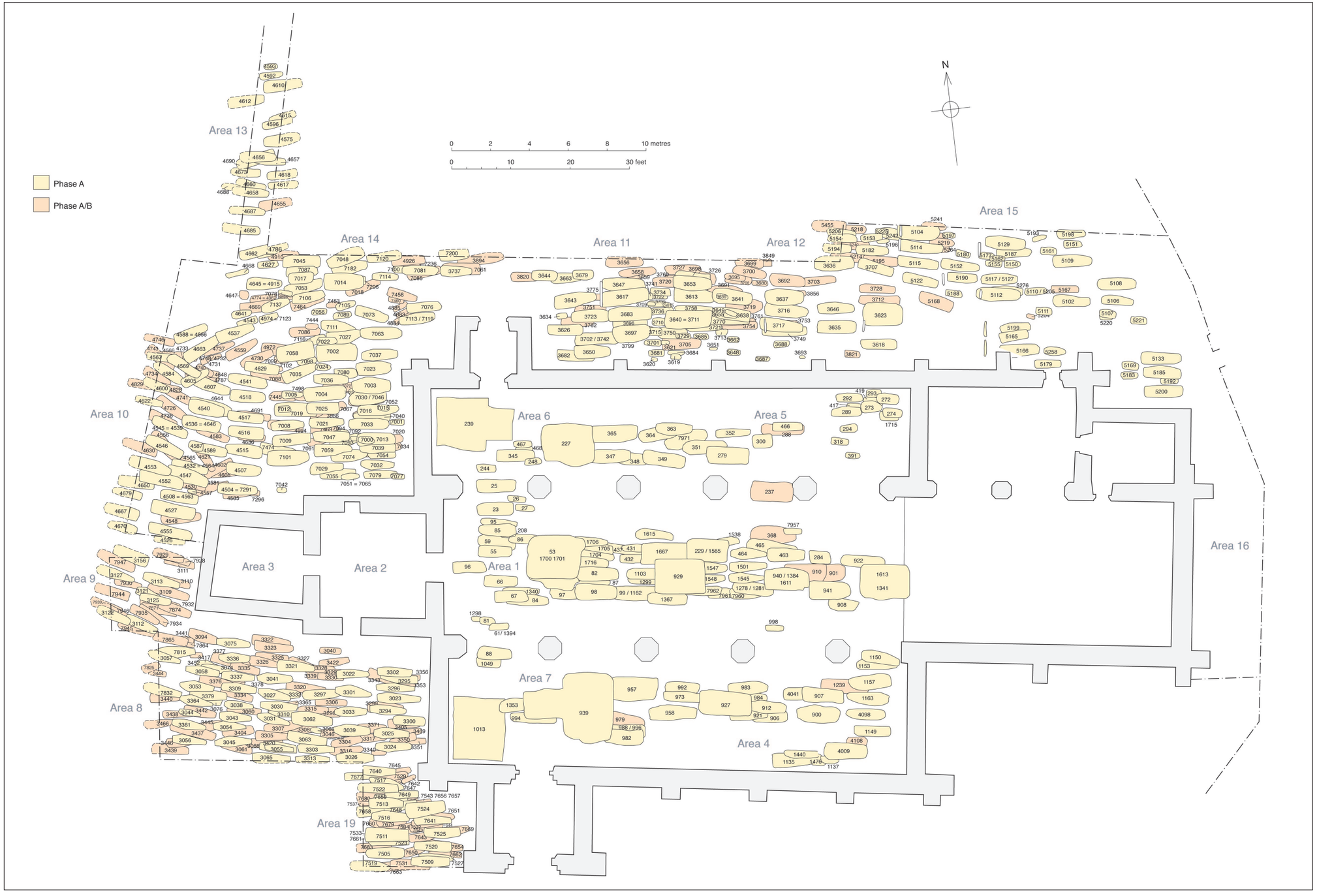

Fig. 36: General plan of the excavated graves attributed to burial Phases A and A/B. The notation given refers to the feature numbers of the graves, not the skeleton numbers. All graves shown within the walls of the church were internal burials. Scale 1:200. Drawing: Simon Hayfield

3. THE ASSEMBLAGE

The study of the human remains from an archaeological site brings us closer than anything else to those who lived in the past. If we are not actually 'face-to-face' with our ancestors, then we are as close as we will ever get in the absence of time travel. A skilled examiner will be able to construct many of the physical characteristics of those who came to be buried at the site – their sex, their height, their approximate age at death, some of the diseases that were present among them – but it is important to remember that this is not a living population but a dead assemblage. As obvious as this seems, it is sometimes overlooked and characteristics of the assemblage are extrapolated to the living population of which it was once a part, frequently with erroneous and misleading consequences.

When an assemblage of skeletons such as this from Barton is examined, the bone specialist is, in effect, carrying out a cross-sectional epidemiological study usually – as here – with a long time base. The similarity to a modern cross-sectional population study is slight, particularly in respect of the nature of the sample. For further details on epidemiology applied to skeletal assemblages see Waldron 1994. The skeletal assemblage is almost completely non-random and the investigator has no control over its composition and it is, therefore, very difficult to know to what extent the information gained can be generalized to the once-living population. The age and sex composition is, of course, completely different, and the structure of the living population cannot be constructed from that of the assemblage unless the age and sex-specific death rates are known, which they will be only when reliable data become available, and these will generally be too recent in origin to be helpful.[1] Nor can other rates that give an index of the health of the population be determined from the assemblage since any such rate – crude death rate, still-birth rate, infant mortality rate, maternal mortality rate, for example – all require for their calculation numbers of living individuals in the denominator, numbers which are both unknown and unknowable. We are also unfortunate in that it is seldom that the cause of death can be ascertained, nor the burden of morbidity within the population, since most human diseases affect the soft tissues rather than the skeleton. On the brighter side, it is probable that the prevalence of diseases that do not lead to death is approximately equivalent in the assemblage and the living population, so that the frequency of several of the diseases that are found in the skeleton is likely to reflect that in the population quite closely (Waldron 1994, 52–5).

The site of St Peter's has yielded what is to date by far the largest collection of human remains to have been studied in the UK. In addition to the great number of discrete inhumations, there were thousands of disarticulated bones, a number that was estimated to weigh approximately three tons. The disarticulated material was examined on site over several seasons by Juliet Rogers (JR) and the inhumations were examined in Bristol by JR and Geraldine Barber, JR's research associate for the project. The disarticulated material was identified as to anatomical element, and any pathology noted and recorded but unfortunately none of the data was in a form that permitted any useful analysis and the original electronic database does not appear to be extant. This means that the disarticulated material cannot be reported on here and it is certain that some interesting information has thereby been lost.

The data from the inhumations were recorded on bone forms and subsequently entered into electronic databases which had been designed by Juliet Rogers and the writer over a period of some years, but always with the Barton material in mind. In general, what might be called the anthropological data – age, sex and measurements – were collected and recorded by Geraldine Barber and the pathological data by Juliet Rogers. After JR's death the archaeological phasing of the site was extensively revised, following a major programme of radiocarbon dating; this rendered her original analyses invalid and all needed to be repeated using the new phasing, which had affected a substantial number of the inhumations. The databases were also checked and any errors found were corrected by reference to the original recording sheets, which had been scanned onto disc.

Number of Inhumations

The number of inhumations on which this report is based is 2,750 and their distribution over the different phases of the site is shown in Table 1, together with the dating for each.[2] This table reveals that over half the skeletons fall into the longer, overlapping phases and the presence of so many in phase B/C is particularly disappointing because we have generally used the date of 1500 as a convenient point to distinguish the 'early' from the 'late' period when there are some interesting changes in the pattern of disease, especially of osteoarthritis (see Chapter 6). It will also be seen from Table 1 that there are approximately twice as many skeletons in the pre-1500 as in the post-1500 period: 1,515 compared with 729, respectively. For convenience, in what follows the pre-1500 phases will be referred to as the 'early' period and the post-1500 phases as the 'late' period.

Although the number of inhumations is large, it averages out to approximately only three per year for the 900-year long use of the cemetery. This is a much

Table 1: Numbers of inhumations at Barton, by phase

Phase	*Date (AD)*	*Number of inhumations*
A	1700–1855	427
A/B	1500–1855	224
B	1500–1700	78
B/C	1300–1700	457
C	1300–1500	85
C/D	1150–1500	368
D	1150–1300	179
D/E	950–1300	437
E	950–1150	446
Others		17
Not phased		32

lower figure than the expected number of deaths during this period. The population in Barton at the time of the Norman Conquest is estimated to have been about 1,000, and in 1563, 1603 and 1676 the estimates are 1,040, 920 and 1,010, respectively, while the 1801 census gives the population as 1,709; in the 1841 census it was 3,475 (Bryant 1994, 145–6; Clark and Hosking 1993). It seems reasonable to assume that over the period during which St Peter's was in use, the population of Barton might have averaged about 1,000 and that the annual number of deaths would have been about twenty-five. If only half had been buried at St Peter's, about 11,000 bodies would have entered the ground there, a quarter of which have been recovered.[3]

Condition of the Material

When the skeletons were examined an estimate was made of the proportion of each skeleton that was present, and a note was also made of the general condition of the bones, which tended to correlate closely with the amount present. Many of the skeletons had *post mortem* breaks but this is nothing out of the ordinary in such an assemblage. About a quarter of the inhumations contained bones which were from one or more additional burials, presumably reflecting the crowded nature of the graveyard and the frequency with which burials would have been cut into by the grave digger. By contrast, only a small proportion of the inhumations (6.2%) contained animal bone, indicating that the site was relatively undisturbed apart from the efforts of the grave diggers.

In over half the inhumations more than 40% of the skeleton was considered to be present, and in almost 20% the skeleton was virtually complete (Table 2). There were some differences in the mean amount of the skeleton present throughout the various phases, but no consistent trend and preservation was not noticeably better in the later than in the earlier phases. The condition of the skeleton had a marked effect on the ability to ascertain sex, as is shown in Table 3. As might be expected, when the skeleton was fragmentary it was much less easy to determine sex than when it was well preserved. Infant and juvenile skeletons were relatively less complete than those of adults, which conforms to the general experience of those who examine human remains.

Ageing and Sexing

Determining the age and sex of individual human remains is the fundamental task of the bone specialist and there is a wide variety of methods that can be used for the purpose.[4] The sexing of adult skeletons depends on the changes that follow puberty, especially those that affect the pelvis which, in the female, becomes adapted for the purposes of child-bearing. The female skeleton tends to be more gracile than the male, the skull lacks the prominent brow ridges and relatively massive mastoid processes of the male, and where both pelvis and skull are well preserved there should be little difficulty in correctly determining the sex of the skeleton. There are several landmarks on the pelvis and the skull which can be used to make the designation and there are several schemes for scoring each of these features to obtain an overall index of 'maleness' or 'femaleness'. If only the pelvis or the skull is present, the determination of sex is more problematic but can usually be achieved with a reasonably high degree of confidence. When neither is present, then recourse can be made to measurements which show a high degree of sexual dimorphism. Those most frequently used are the maximum diameter of the head of the femur and the humerus, the maximum length of the clavicle, and the length of the glenoid. If the skeleton is too poorly preserved to permit these measurements to be taken, then the likelihood of being able to sex the skeleton is so remote that only the reckless or inexperienced will attempt it.

Sexing of pre-pubertal skeletons is virtually impossible except by DNA analysis and none of the methods that have been described is reliable. For details of these see Scheuer and Black 2000. Sexing by DNA involves examining extracted DNA for the amelogenin gene which differs in males and females and can readily be distinguished using the polymerase chain reaction (PCR). The amelogenin gene is carried on both the X and the Y chromosomes but that on the Y chromosome is shorter than on the X. This difference is apparent when the products of the PCR are separated by electrophoresis on a gel, females having a single band and males two bands on the gel (Stone *et al.* 1996). Although this method yields unequivocal results when DNA has been well preserved in the skeleton it is not suitable for routine purposes, mainly on grounds of cost.

As far as ageing the skeleton is concerned, the difficulties are the reverse of those connected with sexing; that is to say, it is relatively easy to age the pre-pubertal skeleton and difficult to age the adult. Children's

Table 2: Barton: amount of the skeleton present

Amount of skeleton present (%)	*0–*	*5–*	*20–*	*40–*	*60–*	*80–*	*90+*
Proportion of total assemblage	0.5	25.4	17.1	9.8	16.7	11.2	19.4
Cumulative proportion	0.5	25.9	43.0	52.8	69.5	80.7	100

Table 3: Barton: amount of the skeleton present (%), by phase and sex

Phase	*Male*	*Probably male*	*Female*	*Probably female*	*Infant*	*Juvenile*	*Unknown adult*	*Mean*
A	72.8	64.2	73.8	64.0	49.8	69.9	24.2	59.8
A/B	50.2	37.9	44.0	21.0	41.5	35.9	16.4	35.3
B	59.4	43.8	60.3	10.0	35.0	39.0	18.0	37.9
B/C	56.9	28.6	52.3	32.5	42.3	41.8	13.4	38.3
C	62.7	75.0	73.8	70.8	52.3	76.3	15.2	60.9
C/D	60.2	32.5	58.6	38.5	43.2	45.7	16.9	42.2
D	69.8	37.5	69.7	42.5	50.5	55.4	20.4	49.4
D/E	70.1	46.4	63.4	40.0	48.4	59.4	21.1	49.8
E	65.6	56.9	70.2	42.9	50.9	62.8	23.5	53.3
Mean	63.1	47.0	62.9	40.2	46.0	54.0	18.8	47.4

skeletons can be aged from the state of formation and eruption of the teeth. The teeth are formed and erupt in a sequence which is well understood and which seems to be very consistent and persistent in the face of internal and external factors. There are several dental charts available to bone specialists and these permit an age to be given to a juvenile skeleton with a high degree of confidence, especially if the jaws are X-rayed so that the complete dentition can be visualized. If the teeth are not present, two other methods are available for ageing the juvenile skeleton. The first is based on the maximum length of the long bones which can then be referred to published modern data, and the second utilizes the pattern of epiphyseal fusion. Using modern data on long bone length tends to under-age the child because contemporary children are taller for their age than those in the past, and it is more reliable to construct tables using measurements obtained from children from the same assemblage who can be aged from dental eruption; this is much to be preferred to using published data.

As the pubertal growth spurt comes to an end the epiphyses of the long bones start to fuse and they do so sequentially and at different ages, all the epiphyses being fused (in modern individuals, at least) by the age of 25. Many of the charts of epiphyseal fusion are based on radiographic data and it is likely that they under-age children in a skeletal assemblage because the epiphyses almost certainly fuse at a younger age nowadays.

Ageing the adult skeleton depends upon either the morphological changes that occur with advancing age in structures such as the pubic symphysis, the auricular surface of the ilium and the rib ends, or on the state of dental wear. None of these methods is very accurate and there is a huge literature attesting to the large margins of error associated with relying on morphological change as an ageing criterion. The most commonly used system of staging dental wear was elaborated by Miles (1962) on Anglo-Saxon skeletons,[5] and this is still probably the most widely used method although it has been modified, and others have been described. It must be remembered that it is only applicable where the diet was abrasive, and certainly by the middle of the eighteenth century the diet had become so soft that the method is no longer reliable.

The estimate of age in the adult skeleton will normally be made using all the criteria available but, however the age is determined, it will not be possible to do more than suggest an age-range within which the age at death is likely to have lain and this range will seldom be less than ten years. Those who claim to age an adult skeleton closely to within a year or two (or even within five years) must have access to methods of ageing that are not widely available.

The Barton assemblage was aged and sexed using standard methods and employing as many criteria as were available and the results are shown in Table 4. There are several features of note in this table, the most obvious being the large number of adult skeletons to which an age could not be assigned – over a third of the total – and of these, over a third could not be sexed either. This is somewhat surprising considering that the condition of the skeletons was generally good and it severely reduces the numbers for subsequent analyses of disease and other frequencies by sex. There is also an apparent dearth of both males and females dying between the ages of 35 and 44 for which there

Table 4: Age and sex of the Barton assemblage

Age	*Male*	*Female*	*Unknown sex*	*Total*
0–			457	457
5–			353	353
15–	76	104	44	224
25–	161	143	14	318
35–	47	6	2	55
45+	209	90	12	311
Adult	254	332	444	1,030
Unknown age			2	2
Total	747	675	1,328	2,750

Table 5: Male-to-female ratio in different phases at Barton

Phase	*Males*	*Females*	*Ratio (M:F)*
A	124	121	1.02
A/B	42	45	0.93
B	24	19	1.26
B/C	96	85	1.13
C	29	28	1.04
C/D	81	81	1.00
D	40	42	0.95
D/E	108	94	1.15
E	156	118	1.32
Total	747	675	1.11

Table 6: Proportion of children under 15 (% of total) in the Barton assemblage, by phase

Phase	*0*	*<1*	*1–*	*5–*
A	11.2	2.3	8.4	7.3
A/B	2.6	0	2.6	2.6
B	4.7	0	5.9	5.9
B/C	8.9	1.7	13.4	11.2
C	1.8	2.0	6.7	6.5
C/D	3.1	0.4	8.5	9.4
D	2.4	0.4	7.7	14.2
D/E	1.6	0	9.0	11.4
E	3.2	0	12.4	11.0
Total	4.2	0.9	8.7	9.6

can be no biological explanation and it seems very likely that there has been some misdiagnosis of age, most probably by putting older individuals into the 25–34 year age-range which seems to be somewhat over-represented. There are remarkably few females in the 45+ year age-range and, again, this does not conform to expectation and must have resulted from misdiagnosis of age. Among the females in particular there is an over-representation in the youngest age groups and very great under-representation in the two older age groups, illustrating how difficult it is in practice to ascertain age in adult skeletons.[6]

The male-to-female ratio is somewhat in excess of unity (1.12:1, in favour of males) and although it has been noted that there is often a bias towards identifying males in a skeletal assemblage, the excess is particularly great in two phases (B and E) and hovers around unity in several others. The ratios show no obvious trend to suggest that the differences noted reflect some genuine biological phenomenon that has changed over time (Table 5).

Almost a third of the assemblage comprised children who died under the age of 15, and this is a common finding at virtually all periods in the past, reflecting poor obstetric and post-natal care and, presumably, a high incidence of infectious disorders, with younger children being especially prone to gastro-intestinal infections. The numbers of children vary considerably by phase as the data in Table 6 show. Those aged 0 in the table would be likely to include both stillbirths and perinatal deaths, which cannot be separated morphologically; those in the <1 year age group reflect what would nowadays be referred to as infant deaths. There is no clear trend in this table, but when the data for the early and late periods are compared there is a marked and significant difference in the proportion of children dying between the ages of 1 and 15 in the later period; the proportions of younger children dying is the same (Table 7). This suggests that there was no great improvement in the care of infants and neonates, nor much change in the quality of obstetric care. These data indicate, however, that the health of older children did improve substantially in the later period.

Stillbirth and Infant Mortality Rates

The data shown in Table 7 are not the rates of stillbirth or infant mortality, as is sometimes supposed; they are merely the prevalence of child burials in the assemblage. It is important to remember that stillbirth and infant mortality rates can only be calculated when the number of contemporaneous live births is also known.[7] At Barton it is possible to hazard a guess at the stillbirth and infant mortality rates, however, since the number of christenings is known from the parish records, and this can be taken as a reasonable proxy for the number of live births. At St Peter's the number of christenings between 1570 and 1850 was 7,359, an average of 26 per year (for further details see Chapter 15). The number of fetal burials which were reliably phased to the same period is 57. Assuming that this represents a quarter of those actually buried, then the stillbirth rate for these years is approximately 31 per thousand, a rate which seems plausible and which compares to the 20 per thousand which is typical of developing countries at present. In England and Wales the SBR currently is 5.3 per thousand (*Childhood and Infant Deaths in 2001*: www.statistics.gov.uk/releases).

There were only twelve deaths in the first year of life in the late period, which gives an infant mortality rate

Table 7: Proportion of children under 15 in the Barton assemblage, by period

		0	*<1*	*1–*	*5–*
Early	% of total	6.8	1.7	20.6	20.3
	95% CI	5.1–8.9	1.0–2.9	17.8–23.7	17.5–23.4
Late	% of total	7.8	1.5	7.8	7.4
	95% CI	6.1–10.0	0.8–2.7	6.1–10.0	5.7–9.5

of 6.5 per thousand, based on the same assumptions used to calculate the stillbirth rate. Given that the IMR is presently 5.6 per thousand in England and Wales, it is obvious that this rate is much too low and reflects either inaccuracies in the age determination or, perhaps, differential preservation of the skeleton.

Age at Death of Infants and Juveniles

For many of the infant and juvenile skeletons a precise age at death was given and the distribution of these is shown in Fig. 37. The largest number of deaths normally occurs at or below the age of one year but, as discussed above, the deficit of deaths in the first year is probably an artefact and some of those placed in the succeeding age group probably belong to this early stage.

From the age of two years onwards the number of deaths is more or less evenly spread up to 15 (the end point in Fig. 37) and the apparent deficits at 13 and 14 are most likely due to mistaken attribution. The large apparent excess of deaths at age 6 probably reflects bias in favour of that number – as do other excesses which occurred at the later ages of 17 and 20 – rather than indicating that more individuals really did die at those ages.

It is difficult to know what may have caused the death of such a large number of young people. From about the age of 15 complications of pregnancy may account for some of the deaths of the females but, as can be seen from Table 4, the number of males and

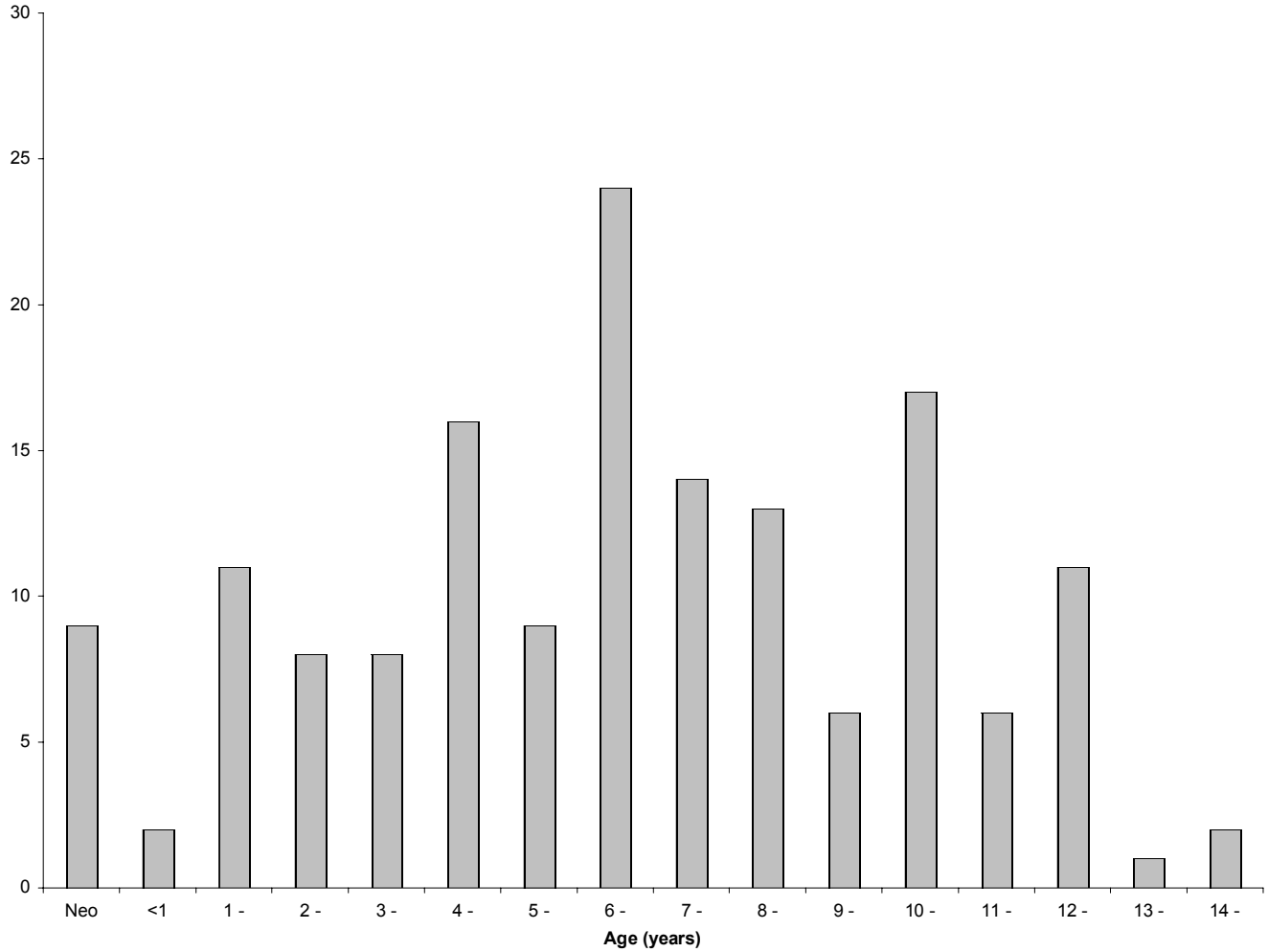

Fig. 37: Age distribution of infant and juvenile skeletons at Barton

Table 8: The named sample at Barton, with associated pathology

Skeleton number and context	*Name*	*Details of death and associated pathology*
47 (F973)	Margaret Roberts	Died June 1822
640 (F1341)	Ann Latham	Died December 1831 (buried 5 January 1832), aged 77. Osteoarthritis of hands, spine and elbow; spinal fusion, osteoporosis with vertebral collapse
740 (F940)	Mary Thorley	Died November 1833, aged 79. DISH; osteoarthritis of the knee; fractured ribs
1356 (F3641)	Mary Goy	Died when pregnant, February 1820, aged 36
1450 (F3697)	Margaret Swallow	Died April 1845, aged 55
1376 (F3638)	Ann Goy	Died January 1825, aged 35
1702 (F3640)	William Goy	Died December 1836, aged 48. Solicitor
1425 (F3683)	Joseph Bromley Swallow	Died November 1842, aged 28. Poliomyelitis

females in the child-bearing age groups (15–34) is approximately equal[8] and while this by no means rules out the likelihood that some of the women died in or around child-birth, it does suggest that this was not a major cause of death in young women.

The large number of juvenile and immature individuals (aged 5–24) is difficult to explain since these are the ages when deaths in most societies are at a minimum. Certainly some will have succumbed to infections, accidents, congenital disorders, and trauma – especially one supposes in the case of young men – while some of the young women may have died in pregnancy or childbirth. None of the skeletons of these young people provided any clue as to the cause of their death, however, and this must remain a conundrum.

The causes of death in very young children are easier to explain and are likely to have been similar to those in modern developing countries (Walsh 1989; Assefa *et al.* 2001). The majority of deaths are due to gastro-intestinal and other infections which are caused by poor nutrition, inadequate sanitation and the absence of a clean water supply (Black *et al.* 2003): all these would almost certainly have been a common feature of everyday life at Barton for much of the period. Breastfeeding protects the infant against diarrhoeal diseases but the child becomes susceptible after it is weaned. Infections *in utero* are relatively uncommon but some infectious agents can cross the placenta, including those that cause syphilis, toxoplasmosis, rubella and cytomegalovirus infections (Finch 2001). There is no reason not to suppose that at least some of the very early deaths at Barton were due to intrauterine infections. Some other early deaths would have resulted from prematurity and very low birth weight, from respiratory distress in the newborn, and from congenital anomalies. Regrettably, none of these causes of death (except, rarely, the last) gives rise to skeletal changes and this is an area where evidence must inevitably give way to speculation.

The Named Sample

The removal of the grave-stones from the churchyard in 1967 was a senseless act of desecration which ensured that only eight of those buried at St Peter's could be identified. Although the date of death was known for these individuals, they died before registration was introduced and therefore their cause of death remains unknown. Two of the individuals had some interesting pathology, however, and one seems to have died during pregnancy, as noted in Table 8.

An Autopsy

One of the eighteenth-century burials, a male of at least 45 years of age at the time of death (sk. 219; F3033), was found with the characteristic signs of an autopsy (Figs. 28 and 29). The calvarium had been removed with a cut that ran just above the squamous part of the temporal bone and the lambda at the back of the skull. In addition, it seemed that the whole of the spinal column had been removed with the exception of the first two cervical vertebrae. The ribs and sternum were also missing, suggesting that these elements too may have been removed at the time of the *post mortem* examination. A charred wooden stake had been put in the place of the spinal column for burial, presumably to give it some semblance of normality for the benefit of the family.

There was no evidence to suggest the reason why the autopsy had been carried out. There were some signs of osteoarthritis affecting the metatarsal head and the odontoid peg, and there was a healed fracture in the right radius. Perhaps the death of this individual had been suspicious and the autopsy was undertaken for forensic purposes, but there would seem no point in removing the spinal column except to examine the spinal cord. The autopsy was probably performed by William Benton who was a surgeon in Barton and lived at Laurel House in Whitecross Street (Rodwell and Rodwell 1981), but it would be remarkable if he had the knowledge, skill or facilities to carry out a neuroanatomical examination.[9]

Expectation of Life at Barton

The expectation of life is estimated by constructing a life table. In the case of the contemporary population

Table 9: Expectation of life at Barton, by phase

Phase	e_0	e_5	e_{20}
A	23.3	29.7	43.9
B	25.1	26.7	37.6
C	28.5	31.1	36.2
D	22.0	26.7	38.4
E	30.2	32.9	38.4

this is done by applying current age- and sex-specific mortality rates to a hypothetical population which is followed up from birth until the age by which all members of the population have died. This technique obviously cannot be used for a skeletal assemblage but methods have been suggested for constructing a form of a life table; the results obtained are by no means accepted by all authorities, principally because of the limitations of ageing adult skeletons. The expectation of life has been calculated for the Barton assemblage but the data must be treated with caution; they will almost certainly *not* accurately reflect the actual expectation of life of the population although the trends may be reliable.[10]

The expectation of life at birth (e_0) during the different phases is shown in Table 9[11] and compares rather poorly with the expectation of life at birth during the eighteenth and early nineteenth centuries, which was between 30 and 40 years (see, for example, Wrigley 1969). The results are rather surprising in that expectation of life appears to *decrease* during the later phases, which is counter-intuitive. The expectation of life at the age of five is also shown (e_5), when the trend towards decreasing life expectancy with time is not as marked, but in no case is the expectation of life at the age of five greater than 33 years, indicating that, on average, these individuals would die at the age of 38. These data, however, merely reflect the high death rate among the children at the site. When the expectation of life at age 20 is calculated, it can be seen that for the latest four phases the expectation is between 36 and 38 years, indicating an age at death of between 56 and 58 on average. There is a considerable increase in e_{20} in Phase A, which suggests that the mean age of death of those who reached age 20 would be almost 64 years, and this is more in line with the expectation that life-span would increase in the later periods. The expectation of life determined from some of the parish records is discussed in Chapter 15, where a comparison with the data obtained from the skeletons will be considered.

4. PHYSICAL CHARACTERISTICS OF THE ASSEMBLAGE

A number of physical characteristics can be determined from the skeleton. The most straightforward of these is the determination of final achieved height, which is estimated from the maximum length of the long bones. The robusticity of the bone will indicate to some extent the overall physical size of the individual, and marked muscle insertions may suggest considerable physical activity during life, although this relationship is by no means as simple and straightforward as is sometimes inferred. A number of metrical indices can be used to describe the shape of bones, most commonly the skull and the shafts of the femur and tibia. It has also been suggested that the weight of an individual can be determined from the measurement of the cortical thickness of the upper part of the femur, the rationale behind this being that bone responds to the stresses put upon it by laying down bone. This may be achieved through the alignment of trabecular bone or by increasing the thickness of cortical bone; this relationship is sometimes referred to as Wolff's law.[1] Finally, the weight of an individual at about the age of 19 can also be determined from the maximum diameter of the femoral head. Final achieved height, cranial, femoral and tibial indices, and weight at the age of 19, were all estimated for the Barton assemblage; weight at the time of death could not be estimated as this can only be undertaken from X-rays of the proximal shaft of the femur (Ruff *et al.* 1991), and these were not available.

Final Achieved Height

The importance of estimating final achieved height in a skeletal assemblage is that it is the best indicator of the state of nutrition at the time of maximum bone growth and, all other things being equal, it is reasonable to assume that where different populations vary in height, this is due to differences in nutritional status at this time (Styne 2003). This rule cannot apply to populations in whom there will be genetic differences (Preece 1996), but at Barton this is not a factor that needs to be taken into account since the genetic determinants of height will not have varied over so short a time as that during which the cemetery was in use.

The height of a skeleton is calculated from the maximum length of the long bones, entering the measurement into an appropriate regression equation. There are equations which utilize measurements derived from fragmented or broken bones, but they are seldom used mainly because of the inconsistency of the landmarks used to take the required measurements on the bones. The equations that are most commonly used were published in the 1950s and 1970s by Mildred Trotter and her colleague Goldine Gleser (Trotter and Gleser 1952; 1958; Trotter 1970). They derived their equations from the study of material in the Terry and Todd collections, and of American servicemen who died during the Korean war. The equations relate to each of the long bones and each equation is associated with a standard error term which defines the range within which the 'true' height is likely to lie. Where there is a choice of which measurement to use, anthropologists will typically select the long bone length that has the lowest standard error term but often they are forced to use whichever long bone happens to have survived intact. It can easily be shown, however, that markedly varying estimates of height are obtained from a single skeleton by using different long bones; this difference may amount to several centimetres. Using any long bone to estimate the mean height of an assemblage will almost certainly introduce an error, particularly in the male skeletons, and it will render comparisons between different assemblages invalid (Waldron 1998). Estimating height based on a single long bone length removes these objections and is a much more sound procedure. Of all the long bones, it is probably best to rely on the maximum length of the femur, if only because this is a robust bone that tends to survive well. It is the procedure that has been used here to estimate the height of the Barton assemblage.

There were 372 adult skeletons with femoral measurements that could be used to estimate height in 216 males and 156 females. The mean height for the males was 1.70 ± 0.6 m, and the range 1.51–1.85 m. For the females the respective values were 1.58 ± 0.5, and the range 1.47–1.69 m.[2] The height distribution for males and females is shown in Fig. 38; there is a considerable

Table 10: Mean height of the Barton assemblage, by sex and phase

	Male			*Female*		
Phase	*Mean (m)*	*SD*	*Range*	*Mean (m)*	*SD*	*Range*
A	1.71	0.07	1.58–1.85	1.59	0.05	1.47–1.70
B	1.72	0.02	1.70–1.74	1.56	0.04	1.52–1.60
C	1.71	0.05	1.64–1.81	1.59	0.05	1.51–1.70
D	1.70	0.05	1.57–1.76	1.58	0.03	1.53–1.62
E	1.69	0.06	1.54–1.83	1.61	0.03	1.54–1.68

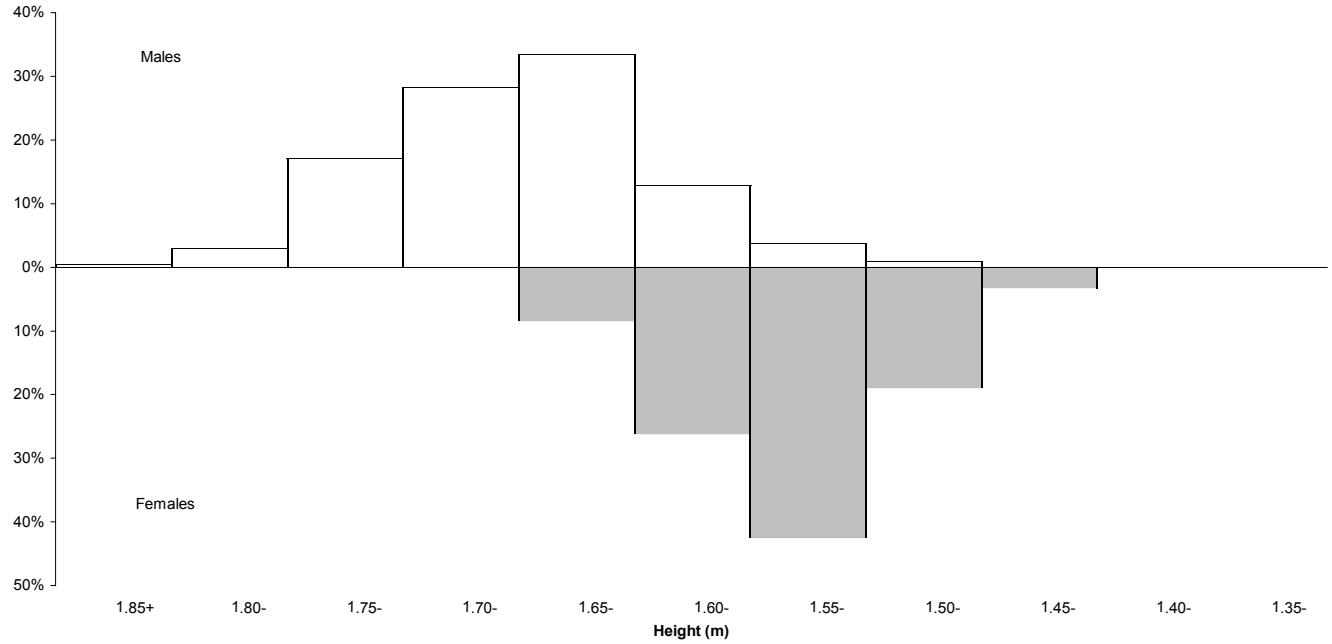

Fig. 38: Distribution of estimated heights of male and female skeletons at Barton

overlap between the two and the distribution in both sexes is approximately normal.

There has been a great deal of discussion among historians about changes of height in the past and it is widely believed that there have been considerable variations.[3] The mean height in the five distinct phases at Barton is shown in Table 10, where it can be seen that there has been a small (<2 cm) increase in the mean height of the males from the earliest to the latest phase, but this is not a statistically significant difference ($F = 0.63$, $p = 0.64$). For the females, if there is a trend at all it is towards a lower mean height in the later phases but, again, the differences seen in Table 10 are not statistically significant ($F = 1.78$, $p = 0.14$). The conclusion to be drawn from these data is that mean height remained remarkably stable at Barton over almost a millennium and this further suggests that the state of general nutrition changed little over the same period.

Naturally, there are some objections that can be made to this conclusion. Firstly, it is possible that Trotter's equations are not strictly applicable to an historic British population. There is no means of countering this argument and the heights that have been estimated are in all probability not an entirely accurate reflection of the height of these individuals during life. The use of Trotter's equations, however, is, in effect, a means of standardisation and, given that the same equation was applied throughout, the results are comparable. The conclusion that the means show no variation, other than that owing to chance, is therefore valid. Another, and more significant, objection is that the mean heights for each phase represent at least 125 years and it is well known that height may change very rapidly in response to nutritional factors. For example, there was a rapid fall in the mean height of English men during the eighteenth century in the wake of the industrial revolution (Nicholas and Steckel 1991). By contrast, at the end of the nineteenth century, the mean height of males increased over the course of two or three decades following improvements in nutrition.[4] Such rapid changes in height are beyond the scope of the examination of human remains. It seems barely credible that the state of nutrition did not fluctuate sufficiently at Barton to affect final achieved height – for example during the great famines at the start of the fourteenth century. Even here, however, a caveat has to be entered, and it is that if the diet improves following a period of inadequacy during the phase of active growth, then, providing the epiphyses have not fused, individuals may 'catch up' and achieve a final height that may not be markedly different from those whose nutrition was always adequate. This is certainly beyond detection in the skeleton.

The Effects of Age

Among the contemporary population there is a strong negative relationship between height and age. This is partly because the elderly appear to lose height due to disc degeneration or kyphosis of the spine, especially noted in elderly women with osteoporosis. The young have grown substantially taller than their parents in recent years, almost certainly as the result of changes in nutrition; for example, the mean height of 25-year-olds has risen by *c.* 4 cm during the last thirty years. A cross-sectional study of height will, therefore, reflect the nutritional status of each age-group during their period of maximum growth.

The mean heights of the Barton assemblage were analysed by age, with the results shown in Table 11.

Table 11: Mean height of the Barton assemblage, by age

	Male			Female		
Age (years)	*Mean (m)*	*SD*	*Range*	*Mean (m)*	*SD*	*Range (m)*
15–	1.68	0.08	1.51–1.84	1.57	0.06	1.58–1.70
25–	1.70	0.04	1.62–1.80	1.59	0.05	1.50–1.70
35–	1.70	0.05	1.59–1.78			
45+	1.71	0.06	1.54–1.83	1.59	0.03	1.52–1.66

Table 12: Barton: relationship between mean age at death and height

	Male			Female		
Height (m)	*Mean age*	*SD*	*n*	*Mean age*	*SD*	*n*
1.50–1.59				29.4	11.0	54
1.60–1.64	33.7	33.7	26	29.4	10.4	36
1.65–1.74	35.2	11.1	102			
1.75+	39.0	11.1	38			

If there is a trend in the data it is towards an increase – not a decrease – with age. In neither sex, however, is the difference significant ($F = 1.72$, $p = 0.16$ for males; $F = 0.98$, $p = 0.38$ for females) and so is most likely to have arisen by chance.

This analysis exemplifies one of the problems that arises in an historical cross-sectional study and which tends to obscure differences that may well exist in the study population. In a modern cross-sectional study, a secular change in height could be noted as all the 20-year-olds, all the 35-year-olds, all the 50-year-olds (and so on) would have been born in the same year; including males from the age of 20 to 70 would enable the investigator to determine whether there were changes in height over the 50 years covered by the study. In an historical study population, such as this one at Barton, the 20-year-olds may have been born at any time during the period that the cemetery was in use; or if phasing were possible, at any time during the period covered by each phase. Each age group, therefore, comprises individuals born in different years and, probably, subject to different nutritional and environmental experiences. The effect is, thus, virtually to obliterate the possibility of finding secular trends such as the one investigated in this section.

The Relationship between Height and Longevity

Although there are some opinions to the contrary, it is generally agreed that longevity is directly related to height (Dublin *et al.* 1984; Waaler 1984)[5] and an earlier analysis by Juliet Rogers and her colleagues suggested that this was also the case at Barton (Gunnell *et al.* 2001). In that study, the length of long bones from 490 individuals showed that the likelihood of dying before the age of 30 decreased as long bone length increased as compared with those dying before the age of 45. The differences, however, were very small and only reached statistical significance for the length of the humerus and a so-called combined bone index. A re-analysis of the data here using height determined only from femoral length has been unable to confirm these earlier results. In the case of the males three sub-divisions of height were made, <1.65 m, 1.65–1.74 m, and 1.75+ m. These divisions were made to provide reasonable numbers in each group. The mean age at death was determined by summing the precise age of death (30, 33 and so on) where given, and the lowest age where this was reported as a range (25–34, for example). Although it is certain that the actual age at death could lie anywhere within the range, and that it might have been more reasonable to take the mid-point of the range for the estimation of the mean, the purpose of the exercise was not to determine the 'true' mean, but merely to compare the mean so derived between groups. Using the same procedure for each group should ensure that no bias was introduced into the calculation.

The results for the males are given in Table 12 and, although they show a trend for increasing age at death with increasing height, the difference is not statistically significant ($F = 2.10$; $p = 0.13$), nor is the difference between the two extreme groups ($t = -1.74$; $p = 0.087$).

So far as the females were concerned, because there were so few with both their age and their height known it was possible to define two groups only, those between 1.50–1.59 m, and greater than 1.60 m. The mean age at death, calculated in the same way as for the males, was exactly the same, as may also be seen from Table 12.

These data, unlike those from the earlier Barton study, provide no evidence to support the view that taller people experienced a more favourable expectation of life. The previous study was also certainly in error by using a variety of bones to estimate height, as discussed earlier in this chapter; had the results been valid there is no reason why the relationship between height and longevity should not have been demonstrated consistently for each bone and the re-analysis presented here seems more likely to represent the true state of affairs at Barton.

The reason for the disparity with results from modern studies may be because the relationship is a relatively recent phenomenon, but unfortunately the numbers were too few to permit an analysis by phase and this matter must remain unresolved at present.

The Growth of Children

Modern children do not grow at a uniform rate but experience two growth spurts, the first during the first two years of life, and the second which begins when they enter puberty. Growth is more rapid during the first than during the second year of life but thereafter slows down until puberty.[6] There are no generally agreed formulae by which to determine the height of children but use can be made of long bone measurements to study the rates of growth.[7] For the present purposes we have relied on the length of the femoral shaft and the results from 190 children up to the age of 15. The rate of growth is greatest during the first two years and thereafter is steady and shows no sign of a pubertal growth spurt, at least up to the age of 15 (Fig. 39). The rate of growth in the first two years is 4.8 cm/year and 1.6 cm/year for the remaining years. These data suggest that puberty was delayed beyond the age of 15 in these children and that they continued to grow later than modern children; this agrees with the suggestion to this effect made some years ago by Tanner (1981).

Some further evidence for this comes from a comparison of the femoral length of the Barton children with modern data from children of the same age. The data from Barton were compared with those published by Maresh (1955), who determined the length of long bones in American children from radiographs. The Barton data were standardized by dividing the measured femoral lengths by those of children of the same age from Maresh's data, to obtain a ratio which we have called δ_1. If the femoral lengths are equivalent in both sets of children then $\delta_1 = 1$. It can be seen from Fig. 40, however, that in the great majority of cases (87.4%) the values for the Barton children are less than unity. There is much more variation in the values of δ_1 in the younger children, and most of the values greater than unity are in the children aged one or two years; this might suggest that some at least were underaged. Nevertheless, these data confirm those which we published earlier for a smaller group of children from the Barton assemblage (Goode *et al.* 1993).

The observation that children in the past grew more slowly than their modern counterparts is by no means unique to Barton: indeed, it seems to be noted for virtually all groups of children studied and is almost certainly an accurate reflection of the true state of affairs (Bogin 1999). Since these children died young, it is possible that they are not typical of *all*

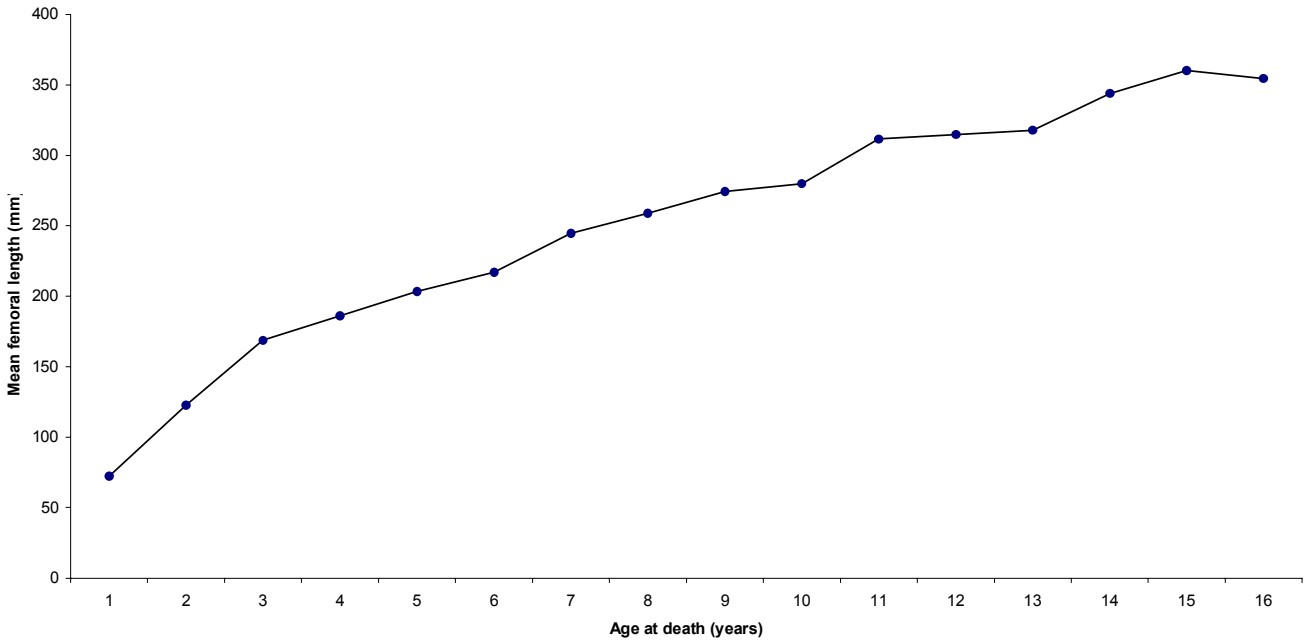

Fig. 39: Mean length of femoral shaft of infants and juveniles at Barton. There is an apparent rapid increase in femoral length in the first two years of life, but no evidence of a rapid increase at puberty

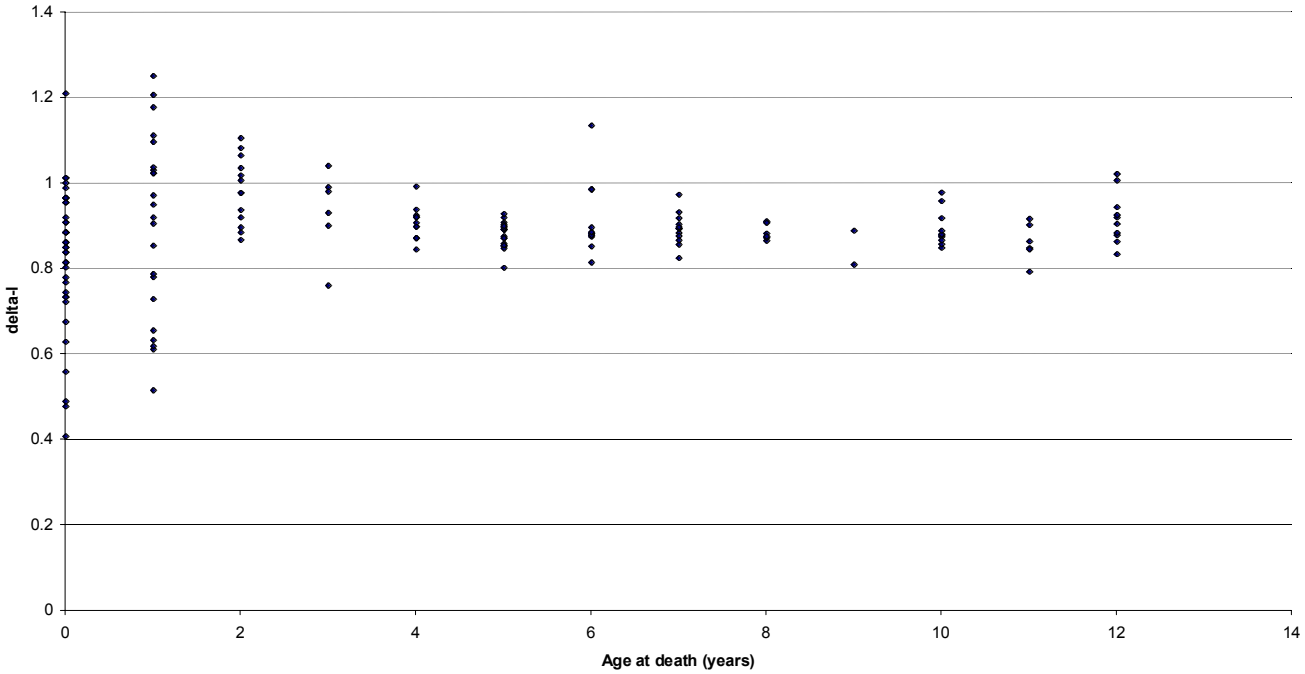

Fig. 40: Femoral length of infants and juveniles at Barton compared with a modern standard. The ratio of standard to actual length (δ_I) is generally less than unity, suggesting that these children were smaller than a modern population of the same age

Table 13: Barton: cranial index, by sex

		Dolichocephalic (< 75)	*Mesocephalic (75–)*	*Brachycephalic (80–)*	*Hyperbrachycephalic (85+)*
Male	n	27	41	12	8
	(%)	(30.7)	(46.6)	(13.6)	(9.1)
Female	n	21	38	17	4
	(%)	(26.3)	(47.5)	(21.3)	(5.0)

Table 14: Barton: cranial index, by sex and period

			Dolichocephalic (<75)	*Mesocephalic (75–)*	*Brachycephalic (80–)*	*Hyperbrachycephalic (85+)*
Male	Early	n	14	18	4	3
		(%)	(35.9)	(46.2)	(10.3)	(7.7)
	Late	n	10	23	5	3
		(%)	(24.4)	(56.1)	(12.2)	(7.3)
Female	Early	n	11	17	9	1
		(%)	(28.9)	(44.7)	(23.7)	(2.6)
	Late	n	10	18	8	2
		(%)	(26.3)	(47.4)	(21.2)	(5.3)

children, and it is likely that at least some died of chronic or acute illnesses that may have adversely affected their growth.[8] An analysis of the Barton data which compared long bone lengths in children with and without so-called stress markers[9] found no difference between the two groups (see Chapter 8). If one assumes that these stress markers are indeed indicative of chronic illness, then it seems to have had no effect on the growth of the skeleton. A more likely explanation, however, is that these markers are unrelated to chronic illness since there has never been any clinical evidence to support what has become widely accepted as fact. The degree to which these patterns of growth may be generalized to the children who survived into adulthood at Barton, therefore, must remain unresolved.

Metric Indices

Of the various indices that can be derived from skeletal measurements, the three that were calculated here were the cranial, femoral and tibial indices.

Cranial index

The cranial index is determined from the maximum length and maximum breadth of the skull. The breadth is divided by the length and the result multiplied by one hundred to give the index. An index of 100 describes a perfectly round skull; the lower the index, the longer the skull. The cranial index assumed great importance in the early years of the twentieth century when it was used in an attempt to define race, and to trace the migration of different peoples. There have apparently been changes in the mean cephalic index from the Neolithic period onwards and this was one reason why the early physical anthropologists were so interested in it (Brothwell 1981, 87 *et seq*.).

At Barton, 168 adult skulls were sufficiently intact for both measurements to be taken and the resultant indices, divided into the four conventional categories, are shown in Table 13. The largest group for both males and females lay within the mesocephalic range and the slight differences there were between the sexes were most probably due to chance. The distribution of the cranial index in the early and late groups was examined to determine whether it had changed over time (Table 14). There was some shift among the males from dolichocephaly to mesocephaly in the later period but this difference was not statistically significant (p = 0.72). The female distribution shows no change worthy of comment in the two periods.

Femoral index

This index describes the shape of the proximal femoral shaft, the measurements used for its calculation being taken just distal to the lesser trochanter. The index has apparently increased in value over time; that is to say, the shaft of the femur has tended to become more rounded. Various hypotheses have been put forward to account for the shape of the femur – mechanical stress, squatting, and mineral and vitamin deficiency – but none seems entirely satisfactory, especially since it is not uncommon to find that the index differs between the two femora.

Table 15: Barton: femoral index, by sex

		60–	*70–*	*80–*	*90+*
Male	n	19	138	102	29
	(%)	(6.6)	(47.9)	(35.4)	(10.1)
Female	n	37	121	76	17
	(%)	(14.7)	(48.2)	(30.3)	(6.8)

A considerable number of femora were measured and a total of 539 estimations of the femoral index could be made, 288 in males and 251 in females. The results are shown in Table 15. The male femora tend to be more round than the female and the differences noted in the table are highly significant (χ^2 = 11.4; p = 0.01). When the index was examined in those buried pre-1500 and post-1500, substantial changes were noted in the distribution of the index (Table 16). In the later period there is a marked shift in both sexes towards a high index; that is to say, the shape of the femoral shaft has become more round, with almost a quarter of the males and just over ten per cent of the females having an index in excess of 90. The differences pre- and post-1500 are highly significant for both males and females (χ^2 = 46.7; p = 0 and χ^2 = 38.3; p = 0, respectively). The differences between the male and female values in the early period are significant (χ^2 = 9.59; p = 0.02), but not in the late period (p = 0.13).

Tibial index

The tibial index describes the shape of the tibial shaft at the level of the distal edge of the nutrient foramen which, however, does not occupy a constant position along the shaft of the bone and the utility of this index is open to doubt. As with the femur, a number of factors have been proposed to account for the shape of the tibia, including pathological change, muscular action and persistent squatting. Over 400 tibial shafts were measured, 239 from males and 218 from females, and the distribution of the index is shown in Table 17. There is a noticeable difference between the sexes, the males tending to have flatter tibial shafts than the females, and this difference is also statistically significant (χ^2 = 8.51; p = 0.01). The distribution pre- and post-1500 is shown in Table 18. The tendency towards flattening of the tibia in males is confined to the early period and the differences in the early and late distrib-

Table 16: Barton: femoral index, by sex and period

		Early				*Late*			
		60–	*70–*	*80–*	*90+*	*60–*	*70–*	*80–*	*90+*
Male	n	16	97	46	5	1	21	37	19
	(%)	(9.8)	(59.1)	(28.0)	(3.0)	(1.3)	(26.9)	(47.4)	(24.4)
Female	n	29	77	28	8	3	25	45	9
	(%)	(20.4)	(54.2)	(19.7)	(5.6)	(3.7)	(30.5)	(54.9)	(11.0)

Table 17: Barton: tibial index, by sex

		60–	*70–*	*80+*
Male	n	95	122	22
	(%)	(39.7)	(51.0)	(9.2)
Female	n	59	131	28
	(%)	(27.1)	(60.1)	(12.8)

utions is significant in the males (χ^2 = 6.11; p = 0.47). The female distributions do not differ significantly (p = 0.09), nor do the differences between the male and female distributions either pre- or post-1500 (p = 0.11 and 0.70, respectively).

Left and right differences

In many instances, measurements were available for both left and right femora and tibiae and where indices for both sides were calculated they were examined to see if there were any systematic differences. The ratio of the indices was obtained by dividing the left value by the right, and the results were plotted as shown in Figs. 41 and 42: the former shows the results for the femoral index. In about 60% of the cases the ratio was between 0.95 and 1.05. In the remaining 40% of cases, the shape of the left and right femoral shafts varied by more than 5%, although very few varied by more than this. In slightly more of the males, the right femoral shaft was larger than the left but this is almost certainly a chance observation; there is no such trend with the females. The distribution of the tibial shaft indices (Fig. 42) is very similar to that of the femoral indices, but the trend towards larger right tibiae is rather more marked than for the femora although, again, fails to reach the conventional level of statistical significance.

Weight in Young Adulthood

Body weight at the age of 18 or 19 has been found to be highly correlated with the maximum diameter of the femoral head in modern populations (Ruff *et al.* 1991). The regression equation to determine weight from the femoral head diameter[10] has been used here with the results shown in Table 19. Both overall mean weights – 81.6 kg for the males and 58.9 kg for the females – seem unrealistically high and one must suppose that the regression equation is not suitable for application to this particular assemblage.[11] When examined by phase, there is no difference between the mean weight of either sex, pre- and post-1500. Again, the estimated weights are surely not representative of the true weights of the Barton individuals: nevertheless, the conclusion that the mean weight has not changed over a very long period should be reliable, if somewhat surprising.

Laterality of Measurements and Sexual Dimorphism

It is by no means unusual to find that the maximum length of the limb bones and the maximum diameter of the femoral and humeral heads varies between left and right, but at Barton no difference between the means of left and right measurements was greater than 2% in either sex; measurements differing by approximately 2% were the ulnar length in females, and the length of the clavicle in males.

Most of the measurements taken on the skeleton show some degree of sexual dimorphism, being larger in males than in females, and indeed some of these differences are used for determining sex when all else fails. To see which of the measurements taken at Barton showed most dimorphism, a sexual dimorphism index was derived as follows:

Table 18: Barton: tibial index, by sex and period

		Early			*Late*		
		60–	*70–*	*80+*	*60–*	*70–*	*80+*
Male	n	55	80	5	22	59	7
	(%)	(39.3)	(57.1)	(3.6)	(25.0)	(67.0)	(8.0)
Female	n	31	80	4	21	49	9
	(%)	(27.0)	(70.0)	(3.4)	(26.6)	(62.0)	(11.4)

Table 19: Barton: weight at age 18 of males and females, by period

	Early		*Late*		*Total*	
	Mean weight (kg)	*SD*	*Mean weight (kg)*	*SD*	*Mean weight (kg)*	*SD*
Males	81.3	7.9	81.7	9.1	81.6	8.4
Females	58.5	7.8	58.7	7.0	58.9	7.7

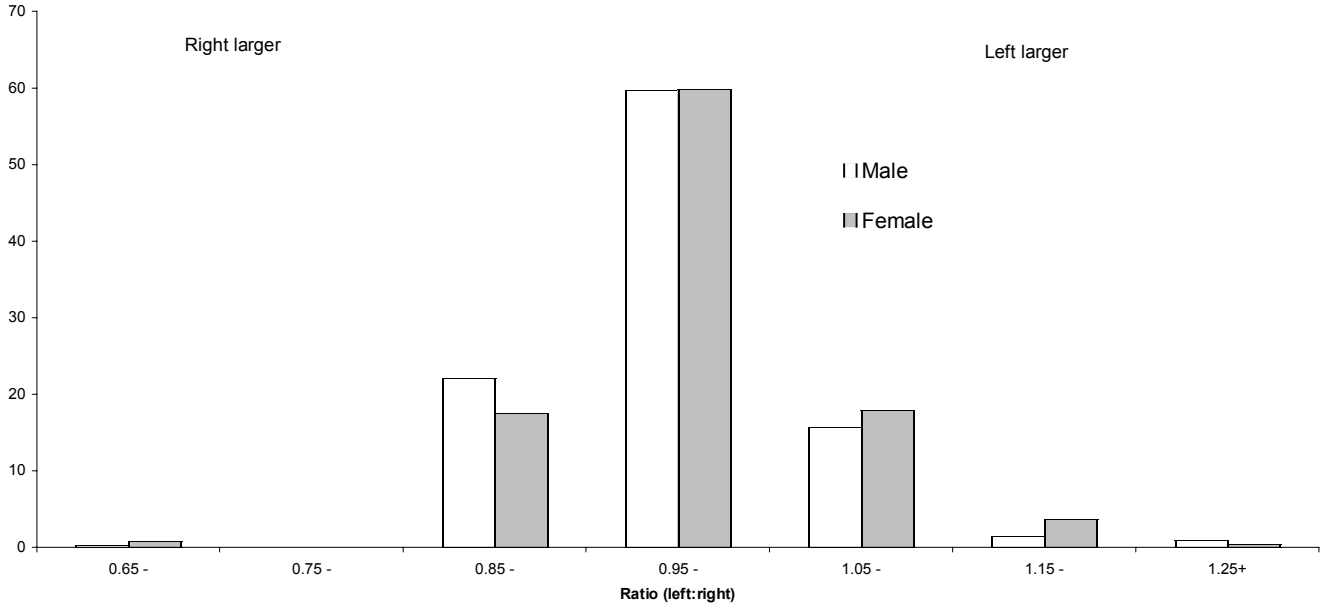

Fig. 41: Distribution of differences between left and right femoral indices, by sex

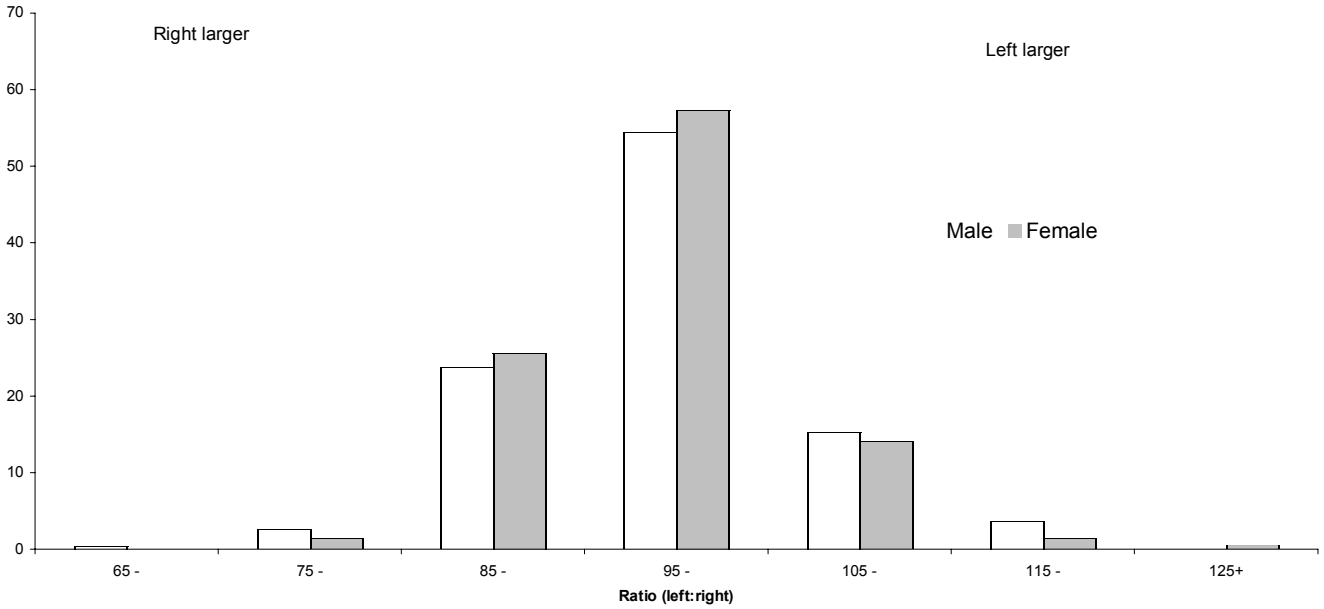

Fig. 42: Distribution of differences between left and right tibial indices, by sex

$$DMI = \frac{male\ length - female\ length}{Female\ length}$$

where DMI = dimorphic index.

The higher the value of the index, the more dimorphic the measurements. As can be seen from Table 20, the most dimorphic measurements were the maximum diameter of the head of the humerus, the maximum length of the glenoid and the maximum diameter of the femoral head, confirming their usefulness in assessing the sex of the skeleton if it cannot be determined by other means.

Table 20: Barton: values of dimorphic index for different measurements

Measurement	*Value of DMI*
Maximum diameter of head of humerus	17.1
Maximum length of right glenoid	16.7
Maximum diameter of head of femur	14.0
Maximum length of left glenoid	13.9
Maximum length of left ulna	11.0
Maximum length of left radius	10.5
Maximum length of right radius	10.0
Maximum length of right clavicle	9.7
Maximum length of right ulna	9.6
Maximum length of left clavicle	9.5
Maximum length of left humerus	9.2

Only indices with a value greater than 9 are shown

5. NON-METRIC CHARACTERISTICS

There are a number of non-metric – or discontinuous – characteristics in both the skull and the post-cranial skeleton. They are dichotomous variables; that is to say, they are either present or absent, and there can be no intermediate position. A great number have been described throughout the skeleton (Finnegan 1978) and they have been used – particularly those found in the skull – in an attempt to measure genetic differences between different populations (Berry and Berry 1967; Berry 1974; Hanihara *et al.* 2003). This assumes that these characteristics are directly under genetic control and this is by no means certain. Nor is it known to what extent they may be determined or modified by environmental factors.

Some of the non-metric traits are very trivial – the form of the supra-orbital foramen, the presence of a parietal foramen, and the presence of certain foramina within the orbit, for example – and data have only been collected here for the twelve cranial and five post-cranial traits which seemed to be the most significant.

Cranial Non-Metrics

The cranial non-metrics examined were those in which there were extra ossicles in certain of the cranial sutures – sagittal, coronal, lambdoid and occipito-temporal – or at the junction of sutures – asterion, bregma and lambda – and metopism, os inca and palatine, and mandibular tori.

Ossicles

These small bones, sometimes referred to as wormian bones (after the Danish anatomist Ole Worm; 1588–1654), may occur in any of the sutures although they are most commonly found in the lambdoid suture. They are most usually understood to be normal variants – perhaps genetically determined – but they are associated with a number of genetic disorders[1] and they may also result from trauma, including cranial deformation (O'Loughlin 2004). The presence of bones at sutural junctions is likely to be genetically determined (Hanihara and Ishida 2001a). The os inca is to be distinguished from the ossicle at the lambda by virtue of its greater size. As the name suggests, it is found commonly in New World populations (Hanihara and Ishida 2001b) and its aetiology is not understood.

Metopism

This refers to the persistence of the mid-line suture in the frontal bone into late childhood or adulthood;

Table 21: Crude prevalence of cranial non-metric traits at Barton

		Crude prevalence (%)	*Juvenile*	*Male*	*Female*
Metopism	Prevalence	3.7	5.6	3.2	3.6
	95% CI	2.7–5.1	2.9–10.2	1.9–5.6	2.1–6.2
Bregmatic bone	Prevalence	0.4		0.5	
	95% CI	0.2–1.1		0.1–2.0	
Coronal wormian bones	Prevalence	0.8	1.4	0.5	0.9
	95% CI	0.4–1.6	0.4–4.9	0.1–2.0	0.3–2.5
Sagittal wormian bones	Prevalence	0.9	1.3	0.5	0.6
	95% CI	0.4–1.7	0.4–4.6	0.1–2.0	0.2–2.0
Bone at lambda	Prevalence	4.9	7.0	2.3	6.5
	95% CI	3.6–6.6	3.9–12.5	1.2–4.5	4.3–9.7
Lambdoid wormian bones	Prevalence	19.5	28.6	18.0	17.4
	95% CI	16.7–22.6	20.8–37.8	13.9–22.8	13.3–22.4
Os inca	Prevalence	0.4	1.1		0.6
	95% CI	0.2–1.0	0.3–3.8		0.2–2.0
Occipito-temoral wormian bones	Prevalence	1.5	1.4	1.9	
	95% CI	0.7–3.1		0.5–4.1	0.8–4.9
Asterionic bone	Prevalence	3.7	5.3	5.0	2.3
	95% CI	2.4–5.7	1.8–14.4	2.8–8.7	1.0–5.4
Parietal notch bone	Prevalence	3.4	3.6	2.8	4.3
	95% CI	2.2–5.4	1.0–12.1	1.3–6.0	2.3–8.1
Maxillary torus	Prevalence	0.4		1.1	
	95% CI	0.1–1.2		0.4–3.2	
Mandibular torus	Prevalence	0.3		0.9	
	95% CI	0.1–0.9		0.3–2.5	

normally it disappears during the first or second year of life. The presence of this metopic suture is likely to be genetically determined and it shows considerable geographical variation (Hanihara and Ishida 2001c).

Tori

There are four of these bony prominences in the skull, but only two – the mandibular and palatine – were considered here. The mandibular torus occurs on the lingual surface of the mandible; it is usually bilateral and found mainly in the molar and premolar areas. The palatine torus is a longitudinal protrusion in the midline of the palate. Both tori show racial, geographical and sexual variation (Chohayeb and Volpe 2001) and both environmental and genetic factors are thought to be involved in their aetiology (Seah 1995).

The prevalence of the cranial non-metrics is shown in Table 21. Most are uncommon, with a prevalence of less than 5% except for the presence of ossicles in the lambdoid suture. There are some differences between juveniles, and between males and females, but none is statistically significant. The crude prevalence of these traits has remained constant over time as may be seen in Table 22, the only difference being in the prevalence of the parietal notch bone which has increased from 1.1% to 6.9% in the late period. This difference is significant at the 5% level but, since it is the only difference noted, it is unlikely to be of biological significance.

Post-Cranial Non-Metrics

Many post-cranial non-metrics have been described but several are trivial, and only six were examined: os acromiale, septal aperture, supracondylar process, vastus notch, squatting facets, and bipartite patella. There were no cases of bipartite patella and so the results reported here relate only to the first five traits.

Os acromiale

The distal end of the acromion has a separate ossification centre and when this fails to unite with the proximal acromion – which usually occurs in early adulthood – the non-united fragment is referred to as an os acromiale. It may be unilateral or bilateral and may be a developmental anomaly, although there is considerable evidence that it may also result from trauma or be related to occupational factors.[2] The condition is sometimes associated with pain and there is a relationship with osteophytic lipping (Edelson *et al.* 1993), tears of the rotator cuff and with the shoulder impingement syndrome (Mudge *et al.* 1984).

Table 22: Crude prevalence (%) of cranial non-metrics at Barton, by period

		Early	*Late*
Metopism	Prevalence	2.8	5.8
	95% CI	1.7–4.6	3.6–9.2
Bregmatic bone	Prevalence	0.4	0.7
	95% CI	0.1–1.4	0.2–2.7
Coronal wormians	Prevalence	0.9	0.4
	95% CI	0.4–2.2	0.1–2.1
Sagittal wormians	Prevalence	0.9	1.1
	95% CI	0.4–2.1	0.4–3.2
Lambdoid bone	Prevalence	5.1	3.9
	95% CI	3.5–7.4	2.1–7.1
Lambdoid wormians	Prevalence	20.1	16.0
	95% CI	16.5–24.3	11.7–21.4
Os inca	Prevalence	0.5	
	95% CI	0.2–1.5	
Occipito-temporal wormians	Prevalence	1.6	1.7
	95% CI	0.6–3.9	0.6–5.0
Asterionic bone	Prevalence	3.9	2.8
	95% CI	2.2–6.8	1.2–6.4
Parietal notch bone	Prevalence	1.1*	6.9*
	95% CI	0.4–3.2	4.0–11.7
Maxillary torus	Prevalence	0.5	
	95% CI	0.1–1.7	
Mandibular torus	Prevalence	0.2	0.8
	95% CI	0–1.0 0.2–2.8	

*Significant at 5% level

Table 23: Crude prevalence (%) of post-cranial non-metric traits at Barton, by sex

		Crude prevalence	*Juvenile*	*Male*	*Female*
Os acromiale	Prevalence	2.7		4.7	0.6
	95% CI	1.5–4.6		2.7–8.3	0.1–3.2
Septal aperture	Prevalence	4.6	1.6*	4.0	7.0*
	95% CI	3.4–6.2	0.5–4.5	2.4–6.5	4.6–10.6
Supracondylar process	Prevalence	1.1	0.5	1.5	1.2
	95% CI	0.6–2.0	0.1–2.6	0.7–3.2	0.5–3.0
Vastus notch	Prevalence	2.8	3.7	2.5	1.8
	95% CI	1.6–4.8	0.7–18.3	1.1–5.7	0.6–5.1
Squatting facets	Prevalence	3.5		2.2	5.8
	95% CI	2.3–5.4		1.0–4.7	3.3–10.1

*Significantly different at 5% level

Table 24: Crude prevalence (%) of post-cranial non-metric traits at Barton, by period

		Early	*Late*
Os acromiale	Prevalence	2.4	3.0
	95% CI	1.1–5.1	1.2–7.4
Septal aperture	Prevalence	5.7	3.5
	95% CI	4.0–8.0	1.9–6.6
Supracondylar process	Prevalence	1.0	1.1
	95% CI	0.5–2.2	0.4–3.3
Vastus notch	Prevalence	2.1	2.9
	95% CI	0.9–4.9	1.1–7.3
Squatting facets	Prevalence	3.4	3.6
	95% CI	1.8–6.3	1.7–7.2

Septal aperture

The septal aperture is a hole in the bone which separates the olecranon and coronoid fossae at the distal end of the humerus. This piece of bone is always thin and easily damaged, especially during excavation or cleaning of the skeleton, but the true aperture is easily differentiated from post-mortem damage by having a smooth or rolled edge. Its significance does not seem to be well understood.

Supracondylar process

This is a spur of bone that projects from the anteromedial aspect of the distal shaft of the humerus. It is usually found about 50 mm above the medial epicondyle and is directed down towards the elbow joint and may form an accessory origin for the pronator teres muscle (Terry 1921). The process is often connected by a fibrous band to the medial epidcondyle and the median nerve, and the brachial artery may sometimes pass through the arch so formed where it may be compressed, giving rise to symptoms in the hand or arm. In this form, it is the homologue of the supracondyloid foramen found in many animals.

Vastus notch

The vastus notch is one form of the so-called patella partita which is a normal variant; in the living it is sometimes mistaken for a fracture of the patella on X-ray.[3]

Squatting facets

These are indentations at the distal end of the tibia which are considered to be due to hyper-extension of the ankle-joint during squatting. They have considerable antiquity, having been found in Neanderthal skeletons (Trinkhaus 1975), but their relationship to squatting has not been proven and it is likely that they represent normal variants in the anatomy of the tibia rather than owing their presence to environmental or behavioural factors; their prevalence in the modern population does not seem to have been recorded.

The prevalence of these post-cranial traits is shown in Table 23. None has a crude prevalence greater than 5% and there is only one marked difference, and that is between the prevalence of septal aperture in juveniles and in females. The prevalence in females is more than four times greater than in the juveniles and could

suggest that the condition continues to develop in later life. The difference between males and females, and between juveniles and males, however, is not significant, so it would be unwise to place much emphasis on these findings.

No differences can be seen in the prevalence of these characteristics in the early or late skeletons (Table 24), which tends to suggest that they are relatively stable and probably not much affected by external factors.

6. PALAEOPATHOLOGY I: INTRODUCTION AND OSTEOARTHRITIS

Introduction

The integrity of the skeleton is normally controlled by the twin processes of resorption and formation which are synchronous, so that the form and mass of the skeleton remain relatively constant throughout much of life. During late adult life, however, bone mass starts to decline; the rate of loss increases with advancing age and may be particularly rapid in females after the menopause (see Chapter 10).

The cells responsible for resorption and formation are the osteoblast and the osteoclast, respectively. Bone metabolism is a complex phenomenon and a large number of factors are involved in controlling the action of the bone cells. These factors include hormones, such as parathyroid hormone, vitamin D, calcitonin and oestrogen; growth factors, including insulin-like growth factors (IGF), fibroblast growth factors (FGF) and platelet-derived growth factor (PDGF); cytokines, including those of the interleukin (IL), tumour necrosis factor (TNF) and colony stimulating factor (CSF) families; and bone morphogenetic proteins (BMP) (Favus 2003).

Bone has a limited capacity to respond to pathological insults and the end result is either to form more bone, or lose bone, and sometimes both. This means that although pathological bone is usually easy to recognize, providing the observer has a thorough knowledge of the range of normal variation, it is not always easy to determine the underlying pathology and arrive at a definitive diagnosis. There are some conditions in which pathognomonic features are present and this renders the diagnosis straightforward, but pathognomonic signs are not common. Diagnosis in palaeopathology is made more difficult because so much of the information on which the clinician or pathologist may rely is not available to the palaeopathologist. For example, the clinician will rely on a medical history from the patient, a family history, a clinical examination, radiology, and the results of supplementary biochemical, haematological, immunological or bacteriological tests, while the pathologist will have soft tissue to examine for evidence of cellular change. The palaeopathologist will almost always have to rely solely on the gross examination of the skeleton – which is often incomplete – and, perhaps, an X-ray. Recently some other diagnostic tools have become available, albeit on a limited basis, the most important of which is the extraction of bacterial DNA from bone to confirm a diagnosis of tuberculosis, leprosy or brucellosis, for example (Zink *et al.* 2002). When one bears in mind that with all the aids at their disposal, clinicians make errors in diagnosis in a substantial number of cases,[1] it is hardly credible that palaeopathologists will be able to do better.

For these reasons, we have suggested earlier that in cases where there are no pathognomonic signs, palaeopathologists should rely on operational definitions, as is done in epidemiological studies (Rogers *et al.* 1987; Rogers and Waldron 1989). The examination of a skeletal assemblage is, in effect, a cross-sectional epidemiological study, and so the application of epidemiological techniques seems entirely apposite. To date, however, almost no operational definitions have been universally agreed, and it is also unusual for those who report on human remains to indicate the criteria they use in making diagnoses. Those who read bone reports will soon find that, although many bone specialists seem very secure in their diagnoses, they frequently have little clinical validity. This has two effects: firstly, the relationship between palaeopathological and clinical diagnosis is uncertain and, secondly, it is difficult – if not impossible – to make reliable comparisons between the results of different studies.

Causes of Death

Most human diseases affect soft tissues and most deaths are caused by soft tissue diseases. Primary bone disease is, therefore, relatively uncommon in the general population and so the palaeopathologist is not able to give a broad-brush account of the health of a past population, but must perforce survey a much smaller canvas. It is rare that the cause of death can be determined from the skeleton, except, for example, in cases of severe head trauma, or execution by beheading, or when there are signs of the spread of malignant disease to the skeleton (see Chapter 12). Young women may sometimes be found with a fetus in the pelvis, the death presumably having been caused by an obstetric accident or perhaps from an intra-uterine infection. There were four such occurrences at Barton, one of whom (sk. 1356, F3641) was Mary Goy, aged 36 when she died. She was buried with an eight-month fetus, and so presumably she died from complications of labour, possibly an uncontrollable bleed. The three other women, two aged 25–34 at the time of death (sk. 1178, F1489 and sk. 2145, F7121) and the other (sk. 405, F3064) unassigned an age, were all buried with a fetus *in situ*; two of the three were apparently full-term infants and so death must have occurred during labour (Fig. 43). In the third case, the fetus was aged seven months and death may have been due to an intra-uterine infection, or perhaps the complications of premature labour.

Osteoarthritis

The most common diseases in the skeleton are those that affect the joints and the teeth, and of the joint

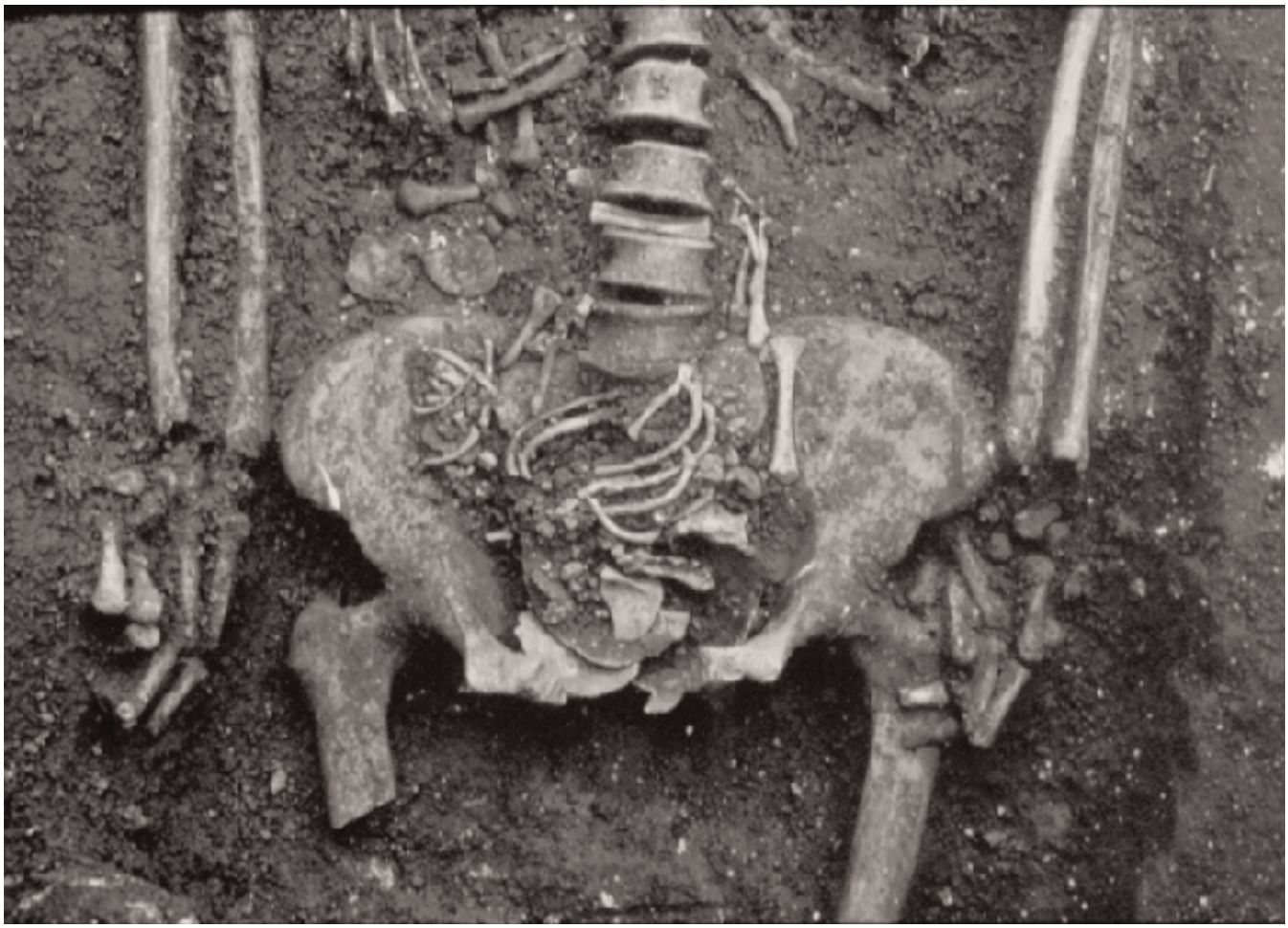

Fig. 43: Skeleton of female (sk. 2145) buried with the skeleton of a full-term fetus in situ. *Death was probably due to some complication of pregnancy or labour. Photo: Warwick Rodwell*

diseases, the most common by far is osteoarthritis (Dequeker and Dieppe 1998; Kraus 1997). Osteoarthritis is primarily a disease of articular cartilage which breaks down as the disease progresses. The breakdown of the cartilage is divided into three phases:

1 Enzymatic breakdown of the cartilage matrix. The metabolism of the cartilage-forming cells (chondrocytes) is affected leading to an increase in the enzymes, including metalloproteinases, that break down the matrix. The chondrocytes produce enzyme inhibitors but in insufficient quantities to counteract the proteolytic effect.
2 The cartilage breaks down into fibrils and the surface becomes eroded leading to a release of collagen fragments and proteoglycan – a constituent of cartilage – into the joint space.
3 The breakdown products induce an inflammatory response in the synovial membrane leading to the production of cytokines such as IL-1, TNF-α and metalloproteinases (Iannone and Lapadula 2003) which can either diffuse into the cartilage and directly destroy it, or lead to the production of further proteolytic enzymes. In time these events give rise to compensatory overgrowth of bone in an attempt to repair the joint.

The changes that occur in the joint are well understood and include:

1 The formation of new bone around the joint margin, referred to as marginal osteophyte;
2 The formation of new bone on the joint surface;
3 Pitting on the joint surface, in some cases communicating with sub-chondral cysts;
4 Alteration in the joint contour; and
5 Eburnation on the joint surface.

Of all these changes, eburnation is by far the most significant so far as the palaeopathologist is concerned. It results when areas of the articular cartilage are completely lost and the articulating ends of the bone come into direct contact and rub on each other giving rise to polishing on the joint surface. The eburnated surface is sometimes scored or grooved in the direction of movement of the joint (Fig. 44), presumably because débris within the joint gets between the two opposing joint surfaces (Rogers *et al.* 1993).[2] Eburnation can be taken as pathognomonic of osteoarthritis and this sign has been used to diagnose the condition here.[3]

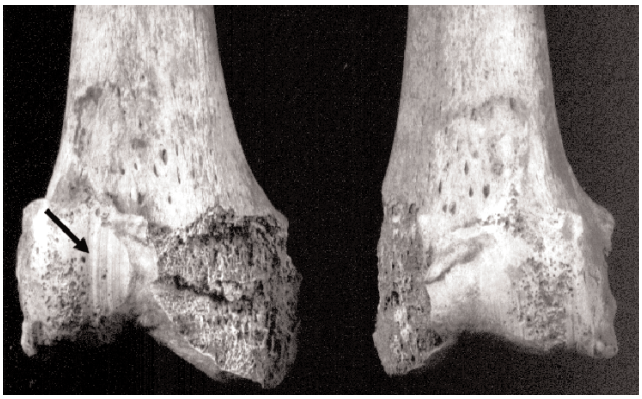

Fig. 44: Osteoarthritis of the patello-femoral joint in a female of unknown age (sk. 10). Grooving (arrow) is clearly seen on the right joint surface. Eburnation, marginal osteophyte and pitting on the joint surface are also clearly seen and there is considerable post mortem *damage. Photo: Juliet Rogers*

Precipitants of osteoarthritis

The cause of osteoarthritis (OA) is not known with certainty but there are a number of precipitants (Sowers 2001) that are acknowledged to be important (see Fig. 45). Osteoarthritis is highly age-related and the prevalence and incidence increase markedly with age. It is also rather more common in females than in males, especially in older women; there is a strong correlation between obesity and osteoarthritis of the knee and the hand (and a much weaker correlation with OA of the hip); there are some racial differences in the expression of the disease; a genetic predisposition is important; and movement is a *sine qua non* for the development of OA. Joints that do not move do not get OA. This has led many authors to try to determine the occupation of skeletons with OA but it takes no great feat of logic to realize that this is an effort that has not the slightest chance of success;[4] this, of course, does not stop them making the attempt.

The factors noted above are important in what is sometimes referred to as primary OA; that is, the variety in which there is no known cause. Following fractures to bones, especially where healing results in alterations in the architecture of the joints above or below the fracture, OA may develop several years later. Where the fracture goes into the joint, or where trauma to the joint damages intra-articular structures, then OA is almost inevitable. Osteoarthritis with a known cause such as this is sometimes referred to as secondary OA, but this term is not used so frequently these days.

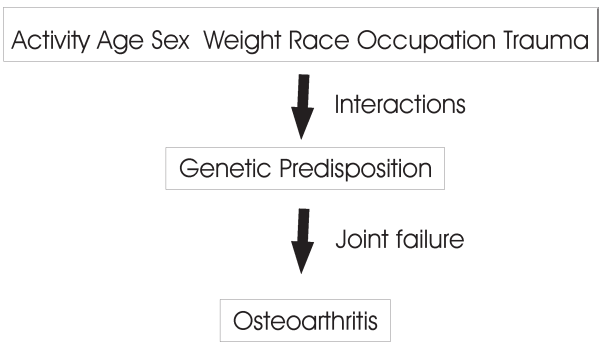

Fig. 45: Model to show development of osteoarthritis. One or more of the precipitants interacts in those with a genetic predisposition to produce joint failure, manifest by the changes which are recognized as osteoarthritis

Natural history of osteoarthritis

Osteoarthritis may affect a single joint (monoarticular) or many joints (polyarticular) and there may be a massive formation of new bone (hypertrophic) or, at the other extreme, very little (hypotrophic); this last type is more common in older females. The changes seen in OA may reverse, but this is very uncommon and usually occurs only in small joints. Normally the condition is irreversible but it does not necessarily progress in severity. There is little correlation between the morphological changes and the symptoms that the patient experiences, and palaeopathologists should resist making clinical judgements on the basis of the appearances of the joint, as they have no means of validating their conclusions. Osteoarthritis is rare under the age of 40 but the incidence increases markedly thereafter, as does the prevalence. Osteoarthritis should not be used as a criterion for ageing the skeleton as it will then be impossible to calculate age- and sex-specific prevalence rates.[5]

In modern clinical practice OA is said most commonly to affect the knee, the hip, the hands, and the facet joints of the spine. This is probably due to the fact that OA at these sites is more painful than elsewhere. In the skeleton, however, this is not the case as will be discussed later. The ability to see all the joint surfaces in the body is one of the few advantages that palaeopathologists have over their clinical colleagues.

Erosive osteoarthritis

In osteoarthritis the production of bone is the predominant feature but there is an inflammatory component affecting the synovial membrane and the joint capsule, perhaps contributing to the pain the patient may experience. There is also a rare variant of OA in which there are erosions on the joint surface. This variant characteristically affects the hands of middle-aged women (Cobby *et al*. 1990) and is discussed further in Chapter 7.

The diagnosis of osteoarthritis

Clinicians make the diagnosis of OA on the basis of pain and swelling in a joint and on a clinical sign known as crepitus, which is a crackling felt in a hand placed over an arthritic joint when it is moved.

Radiologists use the presence of joint space narrowing, of marginal osteophyte, and of sclerosis (Gupta *et al.* 2004). The articular cartilage that caps the ends of the articulating bone is not radio-opaque so that when a joint is X-rayed, there appears to be a gap between the ends of the bones and this is referred to as the joint space. With the onset of OA, the cartilage is destroyed and the joint space appears to narrow. Sclerosis is the name given to a dense white line at the ends of the articulating bones and is the radiological analogue of eburnation.

Few of these criteria can be applied to the skeleton and we rely here on the appearance of eburnation to make the diagnosis. This practice is by no means universally applied and other authors may diagnose OA solely on the basis of marginal osteophyte around a joint. This is a mistaken way to proceed as at many joints marginal osteophyte may be found as a completely independent phenomenon. If there is some resistance to relying only on the presence of eburnation, then it is acceptable to use any two of the four changes described above, but, however diagnosed, the criteria should be clearly stated.

Osteoarthritis at Barton[6]

There was a total of 390 skeletons with osteoarthritis at Barton. Of these 205 were male, 143 female, and 42 for whom no sex could be ascertained. The substantial excess of male cases is unusual and is particularly marked in the early (medieval) period, when there were 121 males and 70 females with OA; in the later (post-medieval) period, the corresponding numbers were 61 and 53, respectively. The difference in the sex ratio in the early period is statistically significant ($p < 0.05$)[7] but in the later period it is not, even though there are approximately a fifth more males affected than females. In the modern period, and in most skeletal assemblages, it is usual to find a slight excess of *females* with osteoarthritis and it is difficult to explain why this is not the case at Barton. Assuming that the observations are valid, the most likely explanation is that the females engaged in different, or more strenuous activities in the early period than they did later on. The other explanation – that it is related to age differences between the males and females – is not plausible since, although there is a higher proportion of younger women than men in both periods, there is no significant change in the age structure in the two periods, as can be seen in Table 25.

One problem with the interpretation of age-related changes is that many of the skeletons with OA could not be assigned an age; this is most noticeable with the post-medieval females, where the great majority fall into the unknown age category. There is no *a priori* reason to suppose that the ages of these individuals would not have been similar to those which were assigned an age, and it is reasonable to assume that the ages are not biased in any way which would render the data in Table 25 invalid. The number of young individuals with OA is relatively high, certainly higher than would be expected nowadays, and may reflect the fact that people started to undertake strenuous activities at an earlier age.

Table 25: Age structure of males and females with osteoarthritis at Barton, by period

	Age (years)	*Male* *n*	*Female* *% of known age*	*n*	*% of known age*
Early	25–	24	27.9	16	32.7
	45+	62	72.1	33	67.3
	Unknown	35		21	
Late	25–	10	25.6	3	33.3
	45+	29	74.4	6	66.6
	Unknown	22		44	

Sites affected

One interesting feature of OA is that by no means all the synovial joints in the body are affected equally; the joints affected in the Barton assemblage are shown in rank order in Table 26. The number of sites involved (638) is much greater than the number of individuals with OA since in many cases the disease affected more than one joint.[8] For the period as a whole, the great majority of individuals – more than three-quarters for both sexes – had only one or two sites affected. With each increase in the number of sites, there were fewer individuals, with only a single female having the maximum of seven sites affected.

There is little difference in the distribution of the number of sites affected between males and females; nor is there very much difference in the distribution in the early and later males in the assemblage, although slightly fewer have one or two sites affected in the earlier period (73.5%, compared with 79.2%). By contrast, the females show a marked tendency for the earlier group to have fewer sites (one or two) affected

Table 26: Rank order of number of cases of osteoarthritis at Barton, by site

Site	*Male*	*Female*	*Unknown sex*	*Total*
Spine	100	84	10	194
Hand	49	51	10	110
ACJ	48	28	3	79
Foot	27	22	4	53
Elbow	24	15	2	41
Wrist	22	11	7	40
Knee	12	24	3	39
Hip	22	12	1	35
SCJ	16	4	0	20
Shoulder	8	4	1	13
TMJ	5	7	0	12
Ankle	1	1	0	2

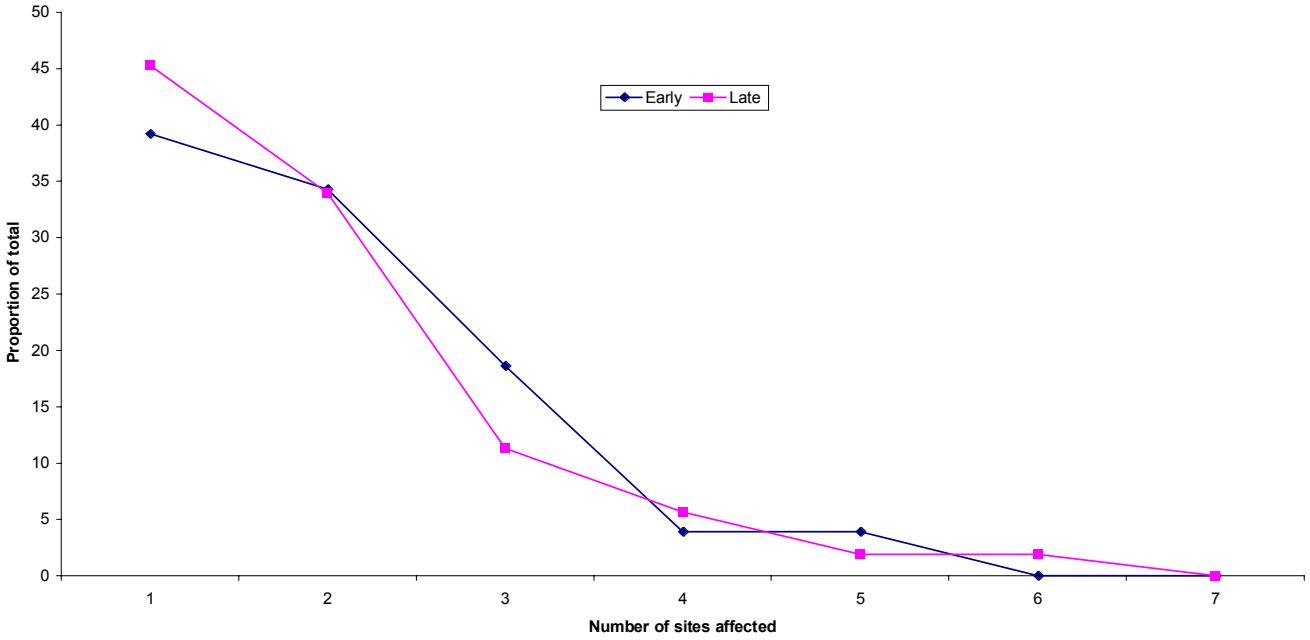

Fig. 46: Number of sites affected by osteoarthritis in males at Barton, by period

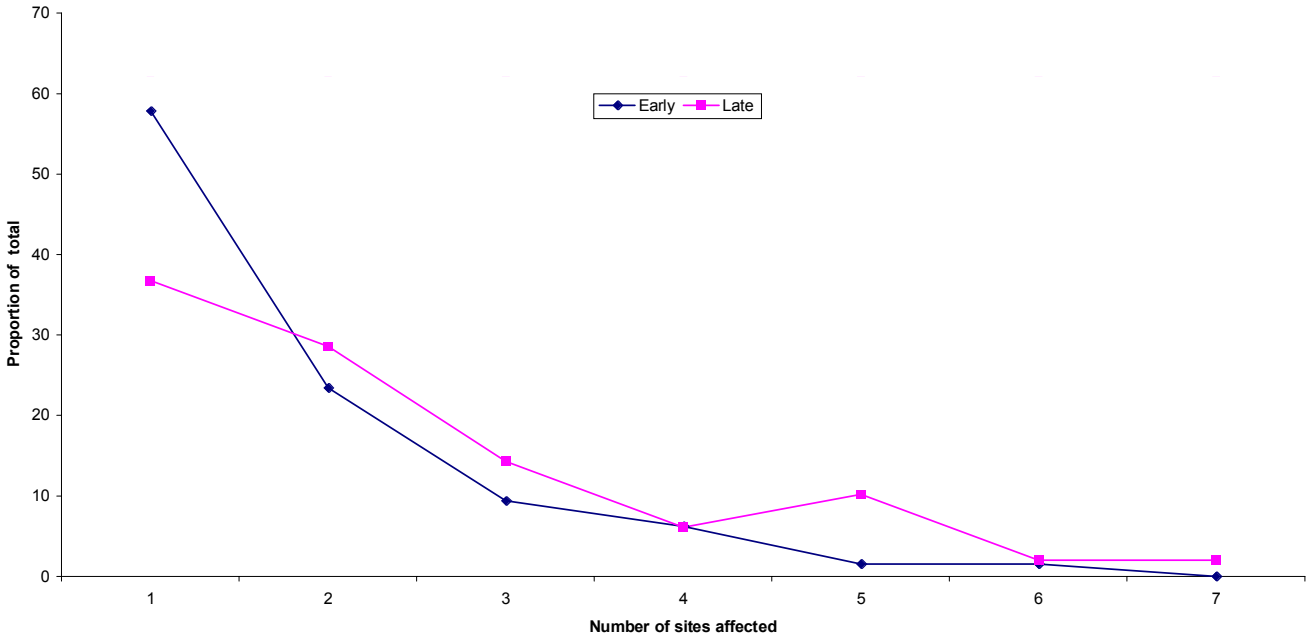

Fig. 47: Number of sites affected by osteoarthritis in females at Barton, by period

than the later (63.5% compared with 81.3%): see Figs. 46 and 47. The fact that the majority of individuals had only one or two sites affected in the later period conforms to the modern pattern, at least in patients with symptomatic disease (Cushnaghan and Dieppe 1991).

Individual sites

Of the various joints, the facet joints of the spine were, by a considerable margin, the most commonly affected, followed by the joints of the hand and the acromioclavicular joint (acj). This is slightly surprising since at virtually every other archaeological site that we have examined, the acj and the spine are affected about equally, with the hand being the third site most commonly affected. There is very little variation in the rank order between males and females (Table 26) except that in the females, OA of the knee is fourth in rank order compared to ninth in the males. The number and laterality of sites affected are shown in Table 27. There is no general trend towards one side rather than the other being affected, except with OA of the elbow in males where the right elbow is affected twice as

Table 27: Number and laterality of sites affected by osteoarthritis at Barton, by sex

Site	*Male*				*Female*			
	Left	*Right*	*Bilateral*	*Total*	*Left*	*Right*	*Bilateral*	*Total*
tmj	1	1	3	5	2	2	3	7
acj	8	12	27	47	6	12	8	26
scj	5	8	3	16	2	1	1	4
Shoulder	3	3	2	8	0	3	0	3
Elbow	7	15	2	24	6	5	4	15
Wrist	9	12	1	22	3	5	3	11
Hand								
Thumb base	5	3	1	9	3	15	4	22
Carpus				10				10
cmc				2				3
mcp				21				22
pip				14				18
dip				9				20
Hip	6	10	6	22	3	5	3	11
Knee								
pf	1	3	4	8	4	6	9	19
Lateral compartment	0	1	0	1	0	3	0	3
Medial compartment	0	5	0	5	1	3	0	4
Ankle	1	0	0	1	0	0	1	1
Foot								
mtp1	7	6	4	17	9	9	4	22
Sub-talar joint	1	1	0	2	0	1	0	1
Other joints in foot	2	3	0	5	1	0	0	1

tmj = temporo-mandibular joint
acj = acromio-clavicular joint
scj = sterno-clavicular joint
cmc = carpo-metacarpal joint
mcp = metacarpo-phalangeal joint
pip = proximal inter-phalangeal joint

often as the left, and with the thumb base in females, where the right is affected much more frequently than the left; both differences were statistically significant.

Some of the differences between the sexes were noteworthy; OA of the acj and scj were significantly more common in males than females, whereas for the thumb base, dips and patello-femoral joint the converse was the case.

Temporo-mandibular joint: Osteoarthritis of the tmj was not common at Barton and almost equally distributed between the sexes. The disease seems to occur most often in cultures that use their teeth as tools (Roberts-Thompson and Roberts-Thompson 1999) and it has also been found to be related to excessive tooth wear (Griffin *et al*. 1979; Hodges 1991).[9] Bilateral disease was common but there was otherwise no trend towards laterality.

Acromio-clavicular joint: Osteoarthritis of the acj is often found in association with rotator cuff disease, but it also occurs commonly as a separate entity (Peetrons *et al*. 2001; Martinoli *et al*. 2003) and this is also one of the joints affected by the arthropathy that is sometimes a complication in patients with chronic renal disease (Shih *et al*. 1993). It is extremely common in skeletal remains, and so it was at Barton. The condition was significantly more common in males, in the majority of whom it was bilateral; it was slightly – but not significantly – more frequent on the right side in females.

Sterno-clavicular joint: This site is not commonly involved with OA although it has been noted in the older clinical literature (Arlet and Ficat 1958; Buckler 1955). At Barton it was substantially more common in males than in females, occurring less frequently on the left than on the right.

The spine: Within the spinal column, the facet joints in the lower cervical and lower lumbar regions are most frequently involved, while in the females the lower thoracic spine is also a common site of the disease, as may been seen from Fig. 48. This pattern is found in contemporary patients (Resnick and Niwayama 1988, 1503) and does not seem to have changed over time (Waldron 1991), perhaps because there is a very strong genetic component in the aetiology of the condition (Spector and McGregor 2004). When the disease occurs in the lumbar spine it is often associated with pain (Borenstein 2004), but there is some dispute about whether or not this is always the case with disease in the cervical spine (Marchiori and Henderson 1996; Peterson *et al*. 2003).

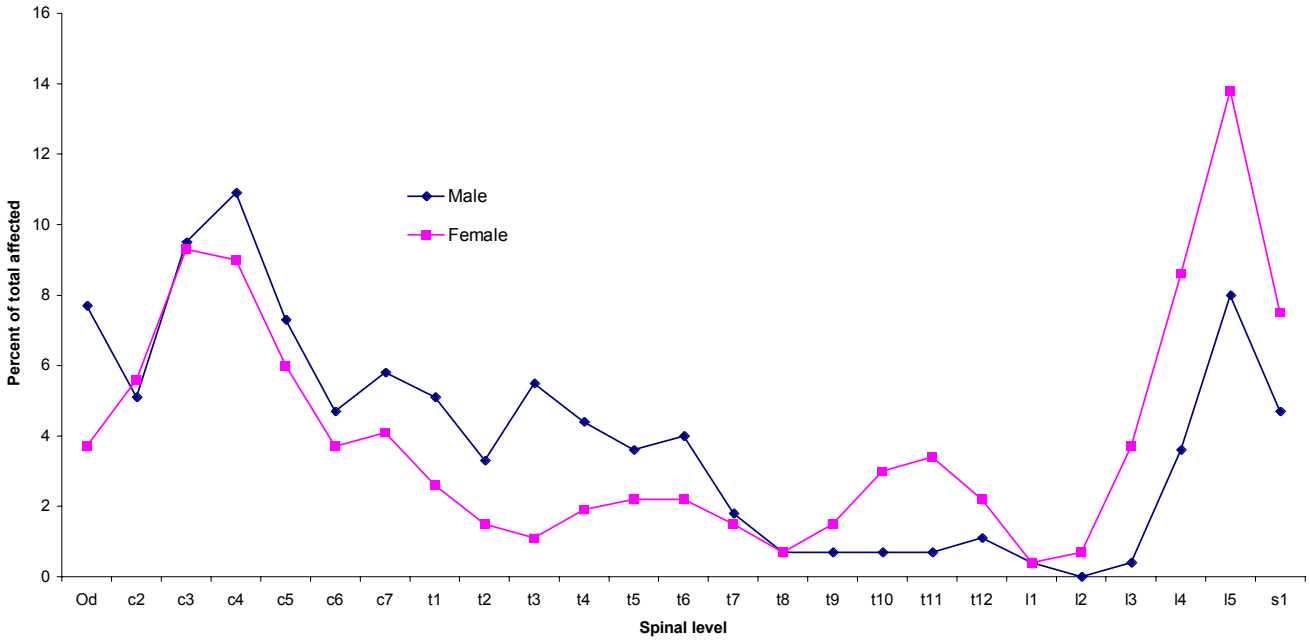

Fig. 48: Distribution of osteoarthritis of the spine in males and females at Barton

Shoulder: Osteoarthritis of the true shoulder joint – that is the gleno-humeral joint – is uncommon and tends to affect women more frequently than men (Nakagawa *et al.* 1999). The other structures which help to form the shoulder joint, most notably those of the so-called rotator cuff, are frequently affected by degenerative changes and these will be referred to later. Although the gleno-humeral joint was seldom involved at Barton there were twice as many males affected as females, which is unexpected given the modern evidence. Among the males with OA of the shoulder there was no obvious preference for right or left, but the right was affected in all three females with the condition.

Elbow: OA of the elbow is not common and when it does occur it is generally in the context of trauma, sometimes occupationally or sports related (see, for example, Bovenzi *et al.* 1987; Schmitt *et al.* 2001). It was, therefore, somewhat surprising to find so many cases in the Barton assemblage: 24 among the males and 15 among the females. The fact that more cases occurred in men than in women, and that the presumed dominant (right) arm was much more frequently affected, conforms to present-day experience (Doherty and Preston 1989) but does little to explain the apparent increase in the disease here. There was a single case (an adult male, sk. 1058, F1773) in whom the OA had developed following a fracture of the left radial head, a fracture that would have damaged the annular ligament.

Wrist and carpal bones: We make a distinction between the wrist joint and the joints between the eight carpal bones. The wrist joint is formed primarily by the distal end of the radius and the lunate and scaphoid, and OA here is commonly met with in clinical practice (Weiss 2004). At Barton there were twice as many male as female cases, one of which (sk. 1074, F4074) was secondary to a left Colles fracture. In both sexes there were slightly more cases affecting the right wrist.

Osteoarthritis of the carpal bones follows the general rule that the radial side of the hand is more often involved than the ulnar (Brown *et al.* 2003). In the Barton assemblage there were 27 instances of OA of the carpals, of which 10 involved the scaphoid, 6 the trapezoid and 4 the trapezium. Of those on the ulnar side, 3 involved the hamate, 3 the triquetral and one the pisiform.

Hand: The propensity for the radial side of the hand to be preferentially affected by OA is well known and is illustrated by the Barton data. The thumb base and the dips were common sites for the disease and substantially more common in females than males, as is the case nowadays (Kessley *et al.* 2003). The metacarpo-phalangeal joints were also commonly affected, with an equal number of cases in males and females; of these joints, the first three bear the brunt of the disease as expected (Table 28).

Table 28: Osteoarthritis of the metacarpo-phalangeal joints at Barton
Distribution by number of cases

	Males	*Females*
mcp1	9	11
mcp2	5	8
mcp3	10	1
mcp4	2	2
mcp5	1	1
Total	27	23

mcp = metacarpo-phalangeal joint

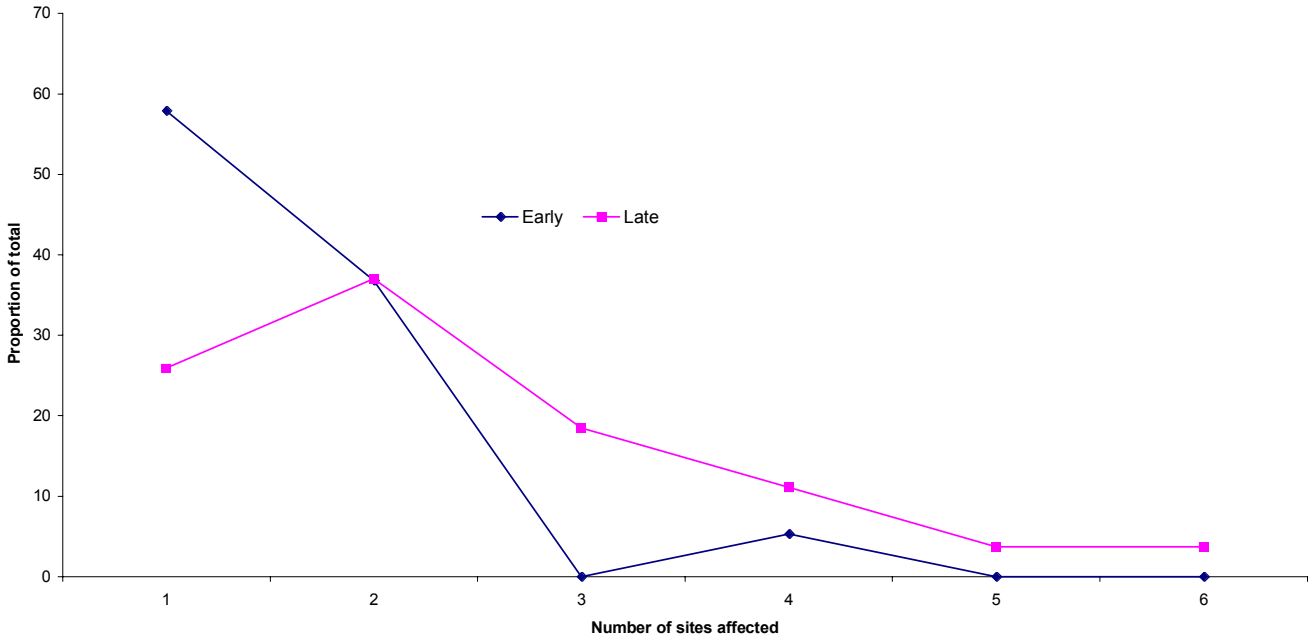

Fig. 49: Number of sites affected in the hands, by period

There is some change in the number of sites affected within the hand over time. Prior to 1500 the disease was more likely to involve only a single site, whereas after 1500 the trend is for more than a single site to be involved. This is particularly noticeable in the females from the site (Fig. 49). This change in the distribution of OA within the hand has been commented on before and probably reflects differences in nutrition and body weight (Waldron 1995). OA of the hand is one of the sites which is highly correlated with obesity – the other being OA of the knee[10] – and it seems likely that an increase in body weight in recent times has been at least partly responsible for the changing pattern in the expression of the disease in the hands (Sayer *et al.* 2003; Coggon *et al.* 2001).

Knee: The knee is a compound joint comprising the patello-femoral and medial and lateral tibio-femoral joints. In the majority of the 39 cases at Barton, it was the patello-femoral joint that was affected and there was no strong evidence of preferred laterality; bilateral disease was common and there were several cases with grooving. The importance of the patello-femoral joint in modern clinical practice was suggested only comparatively recently (McAlindon *et al.* 1992); OA of this joint is especially common in modern women, as at Barton.

The medial tibio-femoral joint was affected more often than the lateral and tended to affect the right side more often than the left; in the five males with OA of the medial compartment only the right was involved. The lateral compartment showed a strong laterality with the right side being affected in all four cases found. The distribution of OA in the various compartments of the knee joint is shown by period in Fig. 50, from which it can be seen that the expression of the disease barely changed throughout the entire period.

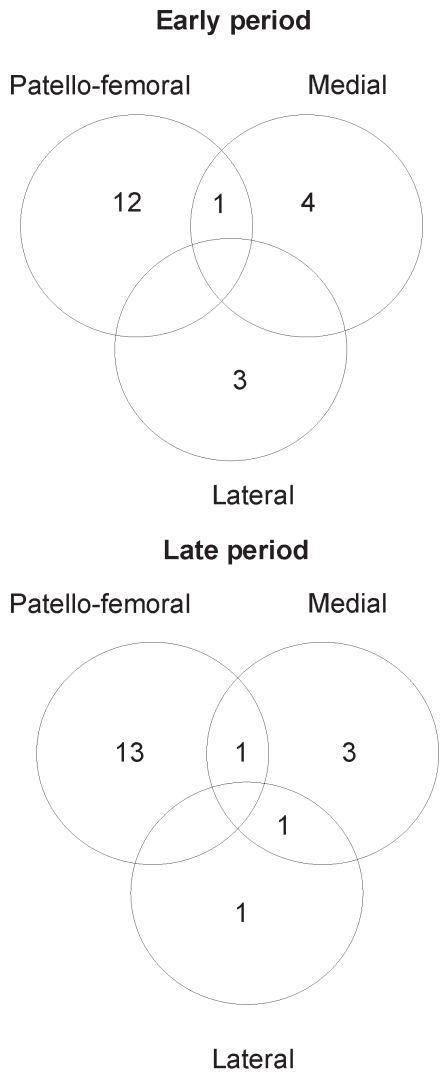

Fig. 50: Distribution of osteoarthritis in different compartments of the knee, by period

Fig. 51: Female skeleton aged at least 45 years at time of death (sk. 70) with osteoarthritis of the hip. A large marginal osteophyte has developed which would have impaired movement at the hip. Marginal osteophyte and pitting on the joint surface are clearly seen in the acetabulum. Photo: English Heritage

Fig. 52: Osteoarthritis of the left hip in an adult female (sk. 1710) with developmental dysplasia of both hips. The acetabulum is greatly reduced in size and a large pseudarthrosis has developed on the ilium which shows typical signs of osteoarthritis. Photo: English Heritage

Hip: Whereas OA of the knee was more common in females than males, the converse was the case as far as the hip was concerned. There was a moderate but not significant tendency for the right side to be more often affected than the left, and bilateral disease was common. In two female cases (sk. 70, F347 and sk. 2801, F7635) the osteophyte around the acetabulum was so extensive that movement at the hip joint was considerably restricted (Fig. 51).

In three cases, one male and two female, the OA was secondary to other pathology of the hip. One adult female (sk. 1710, F4822) had developmental dysplasia of both hips and both were arthritic (Fig. 52); in the case of a young female (sk. 553, F1332) the disease was unilateral and consequent upon a slipped femoral epiphysis, while in the third case, an adult male (sk. 528, F3481) had developed OA in the context of Perthes' disease.

Earlier studies have shown that OA of the hip and the knee reversed their frequency after the medieval period. In the early period, OA of the hip was more common than that of the knee, whereas the converse was true in the post-medieval period; moreover, OA of the medial compartment was considered to be of very recent origin (Waldron 1995; Rogers and Dieppe 1994). These findings conform to modern clinical observations, but at Barton there was no such trend, and OA of the knee was more common than OA of the hip in both periods. The reason for the discrepancy with the previous studies is not clear and the results of some later studies give no support one way or the other, one refuting (Crubezy *et al.* 2002) and one confirming the original observations (Inoue *et al.* 2001); this is certainly an area that merits further study.

Ankle: This joint is very rarely the site of OA in the absence of injury (Huch *et al.* 1997) and so it proved at Barton with only two cases, one male and one female; the male case (sk. 1910) was secondary to osteomyelitis in the distal tibia and foot (see Chapter 8).

Foot: In the foot, OA is restricted almost exclusively to the first metatarso-phalangeal joint and it is more common in women than in men (Brandt 1988; Solomon *et al.* 1975). The pattern at Barton was similar to the modern pattern, although the female excess was slight and not significant; there was no trend towards laterality. There were three cases in which the sub-talar joint was affected, five in which other bones of the tarsus were affected, and a single case in which there was OA in the joint between the third and fourth metatarsals.

Generalized osteoarthritis

Generalized OA (GOA) is considered to be a separate entity that particularly affects elderly women (Cooper *et al.* 1996). Its cause is not known but is has been suggested that it may be hormonally mediated (Spector and Champion 1989), or have an auto-immune basis (Doherty *et al.* 1990), or be an evolutionary problem (Hutton 1987). Using the Ulm definition of the disease, that is, a case in which there is OA of the thumb base and either the pips (proximal inter-phalangeal joints) or the dips (distal inter-phalangeal joints) (Gunther *et al.* 1998), there was a total of ten cases: eight female and two male at Barton. Eight were from the late period. There is no general agreement on the definition of GOA and it is often taken to include OA of at least one large joint (Vignon 2000). Of the ten

Table 29: Barton: cases of generalized osteoarthritis*

Sex	*Age*	*Phase*	*Site*								
			acj	*scj*	*Spine*	*Shoulder*	*Elbow*	*Wrist*	*Knee*	*Hip*	*Foot*
Female	25–	A			✓						
Female	U/K	A	✓		✓		✓	✓			
Female	U/K	A			✓						
Female	U/K	A	✓		✓		✓				
Female	U/K	A			✓						
Female	U/K	A/B			✓						
Male	45+	A/B			✓			✓		✓	✓
Female	U/K	B				✓	✓		✓	✓	
Male	45+	C/D		✓	✓						
Male	U/K	C/D					✓		✓		

*Cases with OA of the thumb base and either the pips or dips
U/K = unknown age at death

putative cases, two (one female and one male) also had OA of the knee, and two (again one of each sex) had OA of the hip. The female case had OA of the hip *and* the knee. In all ten cases, at least one other joint (or set of joints) was involved: see Table 29. It is interesting, but perhaps not surprising that eight of the ten also had OA of the facet joints of the spine. What is perhaps more interesting, given the relative paucity of the disease, is that four of the cases also had OA of the elbow. It is worth commenting that only two had OA of the acj, given how common disease at this site was.

Prevalence of Osteoarthritis

The crude prevalence of OA at the various sites is shown in Table 30 and follows the rank order distribution (Table 26) very closely, as would be expected.[11] In the majority of cases the prevalence appears to have increased in the post-medieval period, almost three times in the case of OA of the elbow and the hip, and has doubled or almost doubled in the same time for OA of the shoulder and the knee. The crude prevalence is not a very suitable measure for comparative purposes since it takes no account of the age and sex distribution of the populations and so any inferences drawn from the data in Table 30 must be treated with caution.[12] Similarly, these data cannot be directly compared with modern prevalence data which are generally based on radiographic findings and which most often quote age- and sex-specific rates.

Table 30: Crude prevalence (% and 95% CI) of osteoarthritis at Barton, by period

Site	*Early*	*Late*	*Total*
Spine	13.1 10.9–15.6	18.4 14.7–22.8	14.5 12.8–16.5
acj	12.4 9.4–16.2	13.5 9.0–19.7	13.6 11.1–16.6
Hand	11.8 9.3–14.9	15.8 11.8–20.9	12.5 10.5–14.9
Wrist	6.3 4.3–9.2	5.6 3.1–9.7	6.1 4.5–8.2
Elbow	3.4 2.1–5.5	9.9 6.6–14.7	5.2 3.8–7.0
Knee	4.3 2.8–6.6	7.2 4.6–11.1	5.0 3.8–7.0
Foot	4.5 3.1–6.6	6.0 3.8–9.3	4.6 3.5–6.0
Hip	2.4 1.4–4.1	6.7 4.2–10.4	4.1 2.9–5.6
scj	3.6 2.1–6.1	2.5 1.0–6.2	3.4 2.2–5.2
Shoulder	1.5 0.7–3.2	3.2 1.5–6.8	1.9 1.1–3.3
tmj	0.7 0.3–1.5	1.1 0.4–2.8	0.9 0.5–1.5
Ankle	0 0–0.6	0.6 0.2–2.1	0.2 0–0.6

Rotator Cuff Disease

The shoulder joint is extremely complex, allowing for a very great range of movement (Petersilge *et al.* 1997). The gleno-humeral joint is very shallow and it is stabilized *inter alia* by the tendons of four muscles that insert into the greater tuberosity of the humerus[13] and by the long head of the biceps muscle which runs in the front of the joint in the bicipital groove (Cone *et al.* 1983). The four muscles all act to rotate the humerus, and the combined tendinous structure around the shoulder joint is referred to as the rotator cuff.

Disease of the rotator cuff is extremely common and is a frequent cause of shoulder pain. It seems to be a true degenerative disease (Hashimoto *et al.* 2003) which increases markedly with age and is often associated with over-use of the shoulder, such as occurs in a number of different occupational groups (Hagberg and Wegman 1987; Frost and Anderson 1999) and it is also sometimes found to be a complication of os acromiale (Park *et al.* 1994).

Fig. 53: Head of right humerus of female aged 45 or more at the time of death (sk. 7). Changes typical of rotator cuff disease were seen on the insertions of subscapularis, supraspinatus and infraspinatus. Photo: English Heritage

Inflammation in the tendons of the rotator cuff muscles will cause changes in the acromion, the coracoid process (Ogata and Uhtoff 1990) and the acj (Hardy *et al.* 1986), and in the bicipital groove if the tendon of the long head of the biceps is involved (Murtagh *et al.* 2000). In addition, if a tear develops in the subscapularis tendon, the action of the deltoid muscle may cause the head of the humerus to become displaced upwards to impinge on the under surface of the acromion, where it may become eburnated with continued use; this is referred to as the impingement syndrome. Although rotator cuff disease (RCD) is not, strictly speaking, a form of osteoarthritis, osteoarthritic changes may form part of the expression of the disease and this is the reason for considering it here.

Table 31: Number of cases of rotator cuff disease at Barton, by sex and laterality

Sex	*Left*	*Right*	*Bilateral*	*Total*
Male	15	11	25	51
Female	4	17	13	34
Sex unknown			1	1

Rotator cuff disease is diagnosed in the skeleton by the presence of new bone and/or pitting on the insertions of the muscles of the rotator cuff, or new bone formation in the bicipital groove (Fig. 53). Impingement can be diagnosed by the presence of eburnation on the superior pole of the head of the humerus or on the under surface of the acromion. Using these criteria, a total of 86 individuals were found to have RCD at Barton, 34 females and 51 males, and a single case in a skeleton that could not be assigned a sex (Table 31). The disease is more often bilateral in males than in females, while in the latter the right shoulder is much more frequently involved. Although the difference in distribution in the females did not quite achieve the conventional level of statistical significance, the pattern of involvement may reflect differences in the activities carried out by the two sexes.

7. PALAEOPATHOLOGY II: OTHER JOINT DISEASES

The joint diseases may be simply categorized into those in which the predominant feature is the production of new bone (the proliferative types) and those in which loss of bone is the most notable characteristic (the erosive types). A simple 'family tree' of the joint diseases is shown in Fig. 54.

Osteoarthritis is the archetypical proliferative joint disease but there are a substantial number of erosive arthropathies, including a form of erosive OA which provides a link between the two types. The type specimen of the erosive arthropathies is rheumatoid arthritis (RA) which was first described clinically in 1800 in an MD thesis by Augustin Landré-Beauvais.[1] The disease had been noted in nine patients in the Salpêtrière Hospital in Paris but was said to differ in a number of ways from gout, the name by which joint diseases were then generally known. It was much more common in women, had a chronic course, and involved many joints from the outset. Landré-Beauvais knew that he was describing a previously unrecognized entity but believed that it was, nevertheless, a novel form of gout, and he called it primary asthenic gout (goutte asthénique primitive). The late appearance of rheumatoid arthritis in the clinical literature has led some to believe that it was truly a new disease, but this is now known not to be so since cases have been found in human remains which antedate Landré-Beauvais's description.

The proximate cause of RA is not known exactly although it is generally referred to as an auto-immune disorder, but whether auto-immunity is a primary or secondary phenomenon is not clear. A genetic factor is thought to be involved but since only a small number of identical twins are both affected the genetic effect is relatively minor and other factors are more important (Svendsen *et al.* 2002). About 90% of patients show the presence of HLA-DR4/DR1 (Ebringer and Wilson 2000) and many cells and cytokines are involved to produce inflammation and proliferation of the synovial membrane which is the major pathological event in the condition.

One feature of RA is the presence in the serum of an autoantibody which reacts with the Fc portion of IgG. This antibody is referred to as rheumatoid factor (RF) and is present in the majority of patients with RA. When RF was discovered it was found that patients with many of the other erosive arthropathies, which had frequently been assumed to be variants of RA, did not have RF in their serum and so these came to be known as the sero-negative arthropathies.[2] The most important of these are ankylosing spondylitis, psoriatic arthropathy, reactive arthropathy (Reiter's syndrome), and enteropathic arthropathy.[3]

The sero-negative arthropathies have a number of features in common but the unifying pathology is inflammatory change in entheses. They are also linked strongly to the MHC Class I allotype which is known to regulate inflammatory cell activation thresholds. The HLA-B27 allotype in particular appears to allow inflammation to arise when in combination with other unknown genetic factors.[4] The presence of HLA-B27 is most strongly associated with ankylosing spondylitis

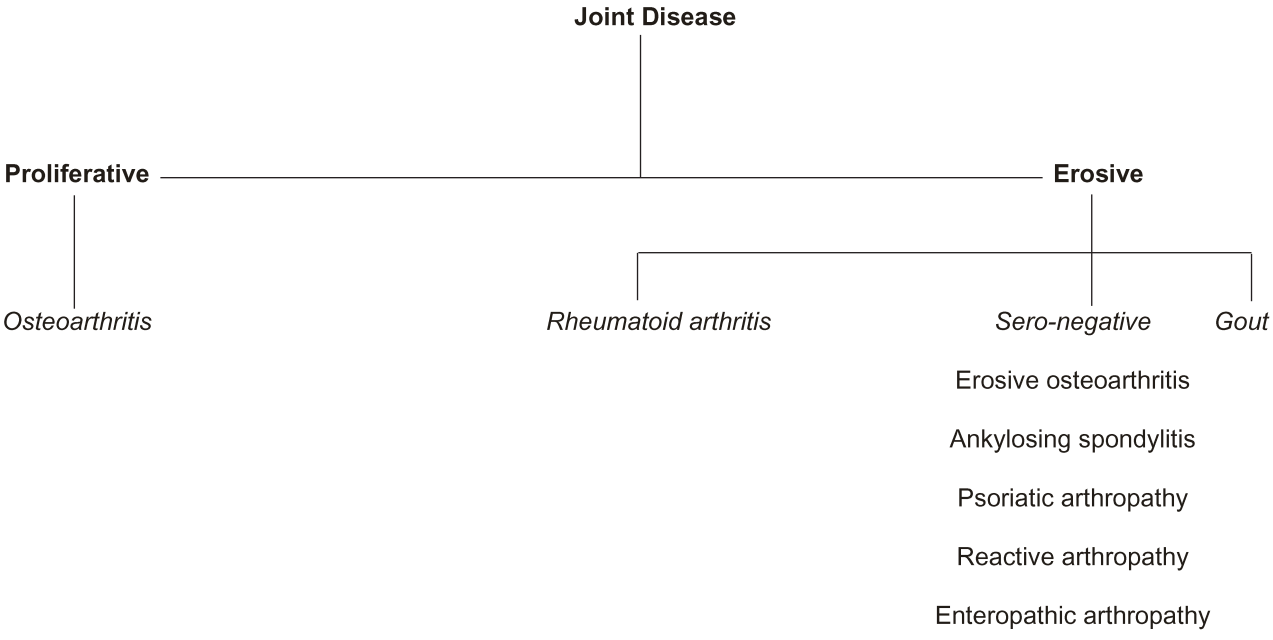

Fig. 54: Family tree of the different joint diseases

(AS) and about 90% of patients with the disease have the antigen, and the arthritis that accompanies chronic inflammatory bowel disorders presents particularly in patients who have the antigen. HLA-B27 is present in about half the cases of reactive arthropathy and those with psoriatic arthropathy (PSA) (Gladman 1998).

The sero-negative arthropathies share similarities in the distribution of the lesions in that the spine and the sacro-iliac joints are invariably involved. On this account they are also commonly known as the spondylo-arthropathies (Kataria and Brent 2004).

Seven skeletons were found with erosive joint changes at Barton but not all could be given exact diagnoses; in addition there were three skeletons with erosive OA and a further ten with gout.

Erosive Osteoarthritis

In some patients with OA the inflammatory component of the disease is sufficient for erosions to occur in the dips and pips. Nowadays erosive changes occur in about a third of patients with OA (Verbruggen and Veys 2000; Cavasin *et al*. 2004) but they have been described very infrequently in human remains (Rogers *et al*. 1991). This may be because the erosions have been overlooked, but it may also reflect changes in the expression of the disease. The erosions[5] begin in the centre of the joint, and the dips may assume a so-called gull-wing appearance which is a helpful diagnostic criterion (Greenspan 2003).

There were three skeletons (sk. 7, F90; sk. 64, F338 and sk. 716, F7767) in which the changes in the small joints of the fingers were consistent with EOA (Fig. 55). Two of the three cases were female, one aged at least 45 at the time of death. Neither of the other cases was assigned an age and one could not be assigned a sex (Table 32).

In three further skeletons with OA, erosions were found which did not conform to the appearance of EOA or with any of the categories of sero-negative arthropathy. The first (sk. 458, a female of unknown age) had OA of the spine and hands and also had erosions with undercut edges in the right scaphoid, and the left capitate and trapezium. The second (sk. 649, F7714, a male aged 25–34) had OA of the feet and hands and erosions in the medial compartment of the right tibia and the left first mtpj. In the final case (sk. 899, F7870, a female of unknown age) there was OA of the odontoid peg with an erosion immediately below the joint surface (Fig. 56). These cases may be referred to as OA with erosions, but they cannot be considered as true instances of EOA.

Fig. 55: Proximal and middle phalanges from a female of unknown age (sk. 716) with erosions (one arrowed) and marginal osteophyte, most probably caused by erosive osteoarthritis. Photo: English Heritage

Fig. 56: Axis vertebra from a female of unknown age showing eburnation on the articular surface of the odontoid peg (sk. 899). An erosion (arrowed) is seen immediately below the joint surface. Photo: English Heritage

Rheumatoid Arthritis

Rheumatoid arthritis has a prevalence rate in the contemporary population of approximately 1% but in developing countries, at least, it appears to be becoming less common. The prevalence increases with age, and women are affected more frequently than men (Kvien 2004). The suggestion that it was a modern

Table 32: Cases of erosive osteoarthritis at Barton, by phase

Skeleton number (context)	*Phase*	*Sex*	*Age*	*Sites affected*
64 (F338)	B	Unknown	Adult	dips, right wrist
7 (F90)	C	Female	45+	dips, superior pole right humerus (RCD)
716 (F7767)	C/D	Female	Adult	Right thumb base, pips

disease raised the possibility that it might be related to an environmental factor that is now of less importance in its aetiology than formerly. Although cases have been found in human remains of considerable antiquity (Waldron *et al.* 1994; Hacking *et al.* 1994), it seems probable that the disease was less common in the past; and it is also possible that it did not progress as far, given that the mutilation which may occur in the hands in advanced cases could hardly have been overlooked by earlier physicians.

The distinguishing feature of RA in the skeleton is the presence of symmetrical marginal erosions in the small joints of the hands or the feet. The disease starts in the proximal joints – the pips and the metacarpo- or metatarsal-phalangeal joints – and as it progresses other joints may become involved but the sij is rarely affected, and when it is, the lesions are seldom extensive. In severe cases, the mcpjs may sublux (that is, become partially dislocated) and the fingers are often deviated towards the ulnar side of the hand and in time the patient may become severely disabled (Scott *et al.* 2003). An important feature for diagnosing RA in skeletal material is the absence of new bone formation such as occurs in the sero-negative arthropathies. The bone around affected joints may be osteoporotic and in very badly damaged joints eburnation may be found when the articular cartilage has been destroyed; the presence of other signs will not lead to any confusion between OA and RA, at least by experienced palaeopathologists and those who do not interpret their observations in the light of pre-existing expectations.

There were only three skeletons (sk. 342, F1244; sk. 817, F7834 and sk. 1622, F4649) in which the lesions found seemed best to fit a diagnosis of RA; two were male and one female, and all were from the early period (Table 33). In two cases the diagnosis was tentative because the skeletons were so poorly preserved. The only female (sk. 342, F1244) was represented by just the right arm and hand, but there were marginal erosions on the first, second and fourth mcpjs with no new bone present; the third mcpj was missing (Fig. 57).

In one of the males (sk. 817, F7834) only about 15% of the skeleton had survived; all that was present were the left hand, proximal left humerus and the rib cage. Marginal erosions were found affecting the pips, and on X-ray RA seemed to be the most probable diagnosis.

The second male (sk. 1622, F4649) was in a much better state of preservation but all the bones of the feet were missing except for the right calcaneus. Marginal

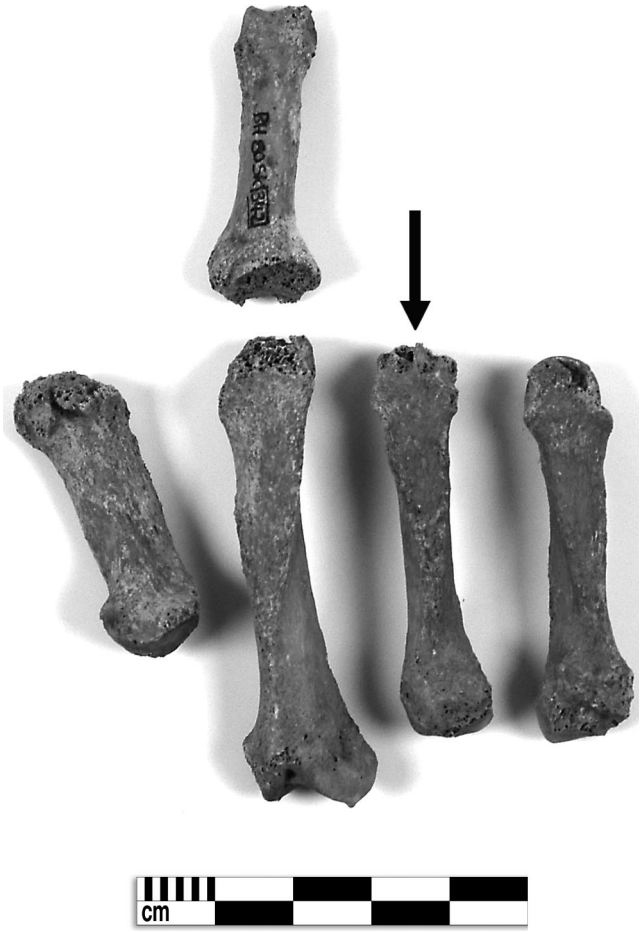

Fig. 57: Right first, second, fourth and fifth metacarpals from a female of unknown age (sk. 342). All but the fourth metacarpal have marginal erosions on the head (one arrowed) consistent with rheumatoid arthritis. Photo: English Heritage

erosions were found on the first and second right mcpjs, on the dips and around the head of the left humerus (the right humeral head was missing). There was ulnar deviation of the first three fingers on the right (Fig. 58), and the X-ray changes were most suggestive of RA.

One reason why RA may not be diagnosed very frequently in the skeleton is that the diagnosis cannot be made if the hands and feet are missing, and there were almost 900 skeletons at Barton to which this applied; it is probable that skeletons that are found with non-proliferative erosions around larger joints may actually have had RA, but without the small joints of the hands

Table 33: Cases of rheumatoid arthritis at Barton, by phase

Skeleton number (context)	*Phase*	*Sex*	*Age*	*Distribution of lesions*
342 (F1244)	C	Female	Adult	Marginal erosions on MCPJs of right hand. Only right arm has survived
817 (F7834)	C/D	Male	Adult	Marginal erosions on left PIPs; only 15% of skeleton present
1622 (F4649)	C/D	Male	Adult	Marginal erosions on DIPs, right MCPJs I and II, and left proximal humerus. Ulnar deviation of first three fingers of right hand

Table 34: Cases of erosive arthropathy at Barton, by phase

Skeleton number (context)	*Phase*	*Sex*	*Age*	*Distribution of lesions*
1347 (F4536)	A	Male	45+	Fused spine, sijs and four ribs. Ankylosing spondylitis.
1937 (F7009)	A	Male	45+	Left talus and calcaneum fused; other tarsals fused with second and third metatarsals; right tarsals fused with second and third metatarsals; new bone and erosions present. Erosive arthropathy, unknown aetiology
1995 (F5111)	B	Male	45+	Erosions on right mtpjs and mcpjs with spiky new bone. Reactive arthropathy
2711 (F7561)	C/D	Female	45+	Widespread erosions hands and feet; cup-and-pencil lesions in hands; skip lesions in spine. Psoriatic arthropathy
345 (F1243)	D	Male	Adult	Cup-and-pencil erosions hands and feet; erosions of sijs; fusion of tarsus and carpus and some digits. Psoriatic arthropathy
893 (F1784)	E	Unknown	Adult	Only right foot and both distal tibiae and fibulae present. Bilateral subtalar fusion; right calcaneus and cuboid fused; calcaneal spurs and periosteal new bone on right tibial shaft. Reactive arthropathy
1851 (unknown)	-	Unknown	Adult	Poorly preserved with only about 50% of skeleton present. Erosions of right first mtpj; dip expanded and eroded. Reactive arthropathy

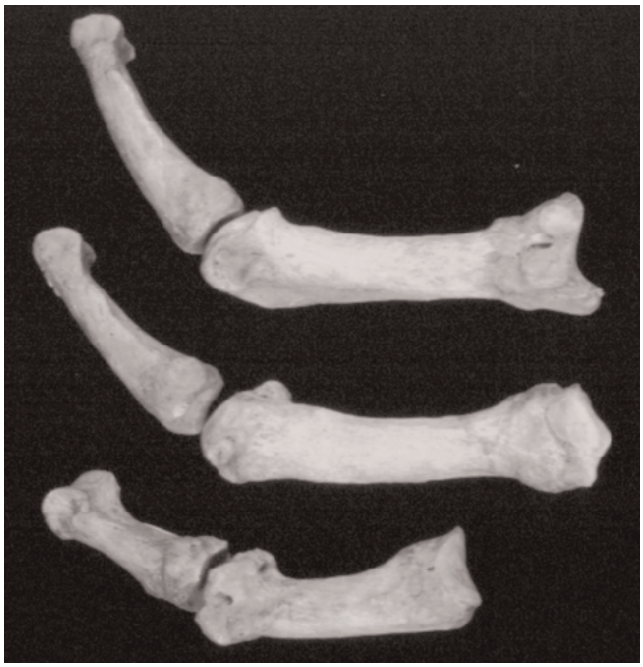

Fig. 58: First three right metacarpals and proximal phalanges from a male of unknown age (sk. 1622). The contour of the metacarpal heads and the bases of the phalanges has been altered in such a way as to suggest that there was ulnar deviation during life caused by rheumatoid arthritis. Photo: Juliet Rogers

and (or) feet, the diagnosis cannot be confirmed.[6] Given that the bones around joints affected by RA are liable to be fragile because of osteoporosis, there is likely to be a bias against detecting RA in skeletal assemblages and this will reduce the apparent prevalence. At Barton, the crude prevalence in the early skeletal assemblage is 0.70% (95% CI 0.24–2.05%)[7] which is the same as prevails nowadays. This does not provide any evidence for the hypothesis that RA was less frequent in the past than it is today.

Ankylosing Spondylitis (AS)

This is the most straightforward of the sero-negative arthropathies to recognize in the skeleton and it is a disease of considerable antiquity, cases having been described at least as far back as ancient Egypt (Elliot Smith and Wood Jones 1910). The disease affects men more than women and the prevalence nowadays is slightly in excess of 1%.

The pathological changes begin in the sijs where they are bilateral and symmetrical and finally produce fusion of the joints (Bennett *et al.* 2004). Inflammatory change in the anterior longitudinal ligament of the spine results in spinal fusion, which begins in the lower lumbar region and may then proceed inexorably upwards, sometimes involving the entire length of the spine. In the thoracic region, the ribs may fuse to the spine if the disease produces inflammatory change in the costo-vertebral and costo-transverse joints.

There was only a single case of AS at Barton (sk. 1347, F4536), a male of at least 45 at the time of death (Table 34). The entire spine was fused, both sijs were fused, and the first left rib was fused to the manubrium. There was a solitary erosive lesion, on the right navicular at the margin of the joint with the talus. This skeleton came from the late period and the crude prevalence is thus 0.28% (95% CI 0.05–1.57%).

Psoriatic Arthropathy (PSA)

Psoriasis is a skin disease which affects about 1% of the population and of these, a variable proportion – between 5% and 42% – develop joint disease (Gladman and Brockbank 2000). Although the association between psoriasis and joint disease was recognized in the early part of the nineteenth century (O'Neill and Silman 1994), the arthropathy was not separated from RA until the 1960s. The changes in the sijs in PSA are often unilateral or asymmetrical: there

Fig. 59: Fragments of calcified arteries found with skeleton 345. Photo: Warwick Rodwell

Fig. 60: Left foot from adult male skeleton (sk. 345) with extensive fusion of the tarsus and changes in the distal inter-phalangeal joints. The bone was extremely fragile and has deteriorated since exhumation and examination. The changes are considered to be most characteristic of psoriatic arthropathy. Photo: Juliet Rogers

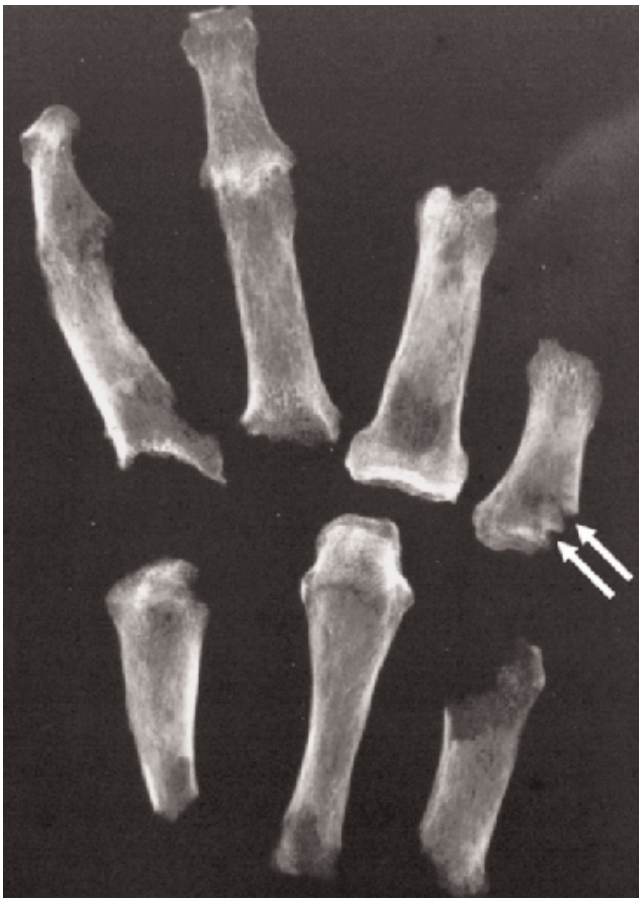

Fig. 61: X-ray of metacarpals and phalanges from the same case as in Fig. 60. Ankylosis of the proximal inter-phalangeal joints is evident and many erosions are present, best seen on the base of the first proximal phalanx (arrowed). Photo: Juliet Rogers

is often some spinal fusion, with normal regions intervening between the areas of fusion, so-called skip lesions. The extra-spinal lesions are asymmetrical, with the dips, pips and mtpjs being preferentially affected (Gold *et al.* 1988; Brockbank and Gladman 2002). Although PSA is a chronic progressive disease in the majority of patients (Kane *et al.* 2003), the changes in the skeleton are often not very marked and a number of variants have been described (Veale *et al.* 1994), including a severe, mutilating form in which the inter-phalangeal joints are destroyed leading to ankylosis and telescoping of the digits. The proximal end of the middle phalanx is often widened, while the distal end of the middle phalanx is resorbed to present what is known radiologically as a 'cup-and-pencil' sign. This severe form of PSA – arthritis mutilans – has some of the appearances of leprosy and it is possible that some of the cases of leprosy in the palaeopathological literature may actually have been PSA.

There were two skeletons at Barton with changes that were best explained by PSA. The first was an adult male (sk. 345, F1243);[8] the skeleton was virtually complete and when found in the grave had flexed knees. It was extremely fragile and difficult to recover, and even

Table 35: Crude prevalence of erosive arthropathy at Barton, by period

	Early period		*Late period*	
	Prevalence (%)	*95% CI*	*Prevalence (%)*	*95% CI*
Rheumatoid arthritis	0.70	0.24–2.05		
Ankylosing spondylitis	0.28	0.50–1.57		
Psoriatic arthropathy	0.47	0.13–1.70		
Reactive arthropathy	0.47	0.13–1.70	0.34	0.06–1.89

when inspected in the ground, preservative had to be used to consolidate the bone. In addition to the skeleton a handful of fragments of calcified arteries were recovered (Fig. 59). There were widespread lesions throughout the skeleton. The fourth and fifth lumbar vertebrae and the ninth, tenth and eleventh thoracic were fused, and the right first rib was fused to the manubrium. The medial condyle of the left humerus was missing and a pseudarthrosis had been formed. Erosions were present in the distal radius, around the right wrist, and the right lunate was fused to the distal radius. Erosions were also present around the knee and on both sijs, and both feet were extensively fused with erosions affecting the distal inter-phalangeal joints (Figs. 60 and 61). Marginal erosions were present on all the extant carpal bones and on all the other joints of the hands, and typical cup-and-pencil erosions were present in the hands and the feet. Erosions were also evident at entheses and sites of ligament insertion.

The combination of sacro-iliitis, skip lesions in the spine, widespread erosions and cup-and-pencil changes in the hands and feet leave little doubt that this is a true case of PSA. It is not clear what caused the lesion in the left elbow but they are unlikely to be connected with the other changes in the skeleton.

The second skeleton (sk. 2711, F7561) was a 45-year old female with spinal fusion affecting the second and third cervical and first and second lumbar vertebrae. There were inflammatory changes in the sijs and pepper-pot erosions affecting both wrist joints, erosions affecting all the small joints of the hand, the tarsal bones and the mtp joints in both feet; the phalanges of both feet were missing. There were also erosions at the junction of the manubrium and body of the sternum with undercut edges and remodelled trabeculae. The changes in this skeleton were best explained by PSA and, as with the case above, radiography confirmed the diagnosis.

Both cases would fall within the polyarticular subgroup (Koo *et al.* 2001), and both were found in the early phase of the cemetery; the crude prevalence of the disease in this sector of the assemblage is 0.47% (95% CI 0.13–1.70%) which overlaps that of RA given above (Table 35). It would be a surprise indeed if the prevalence of these two diseases had truly been the same in the early years at Barton (Savolainen *et al.* 2003), and we must suppose that the similarity is an artefact of small numbers.

Reactive Arthritis (Reiter's Syndrome)

In 1916 Hans Reiter described a combination of urethritis, conjunctivitis and arthritis in a young German army officer who had had a bout of bloody dysentery. Reiter (1916) considered that the changes were an unusual form of syphilis. A similar combination of symptoms was described in the same year in four soldiers by Fiessinger and Leroy (1916), but the sobriquet Reiter's syndrome has persisted in the English literature[9] although the term reactive arthritis (ReA) is now generally preferred.

The syndrome is associated with urethral infections, venereal or non-venereal, and with gastrointestinal infections particularly with *Shigella*, *Salmonella*, *Cambylobacter* and *Yersinia* species (Rees *et al.* 2004). The condition existed well before its first clinical description and arthritis associated with venereal disease was common during the nineteenth century in London, accounting for 3% of admissions to three of the largest London hospitals (Storey and Scott 1998).

Reactive arthritis may be present in the living with a number of different clinical syndromes (Amor 1998), but in the skeleton it is characterized by asymmetric marginal erosions which preferentially affect the small joints of the feet. As expected, the sijs are involved and there are skip lesions in the spine. The formation of periosteal new bone is more common in ReA than in PSA and fluffy new bone may be found around the calcaneum and on the metatarsal shafts, and sometimes on the tibias. The changes in ReA are usually not as destructive as those in PSA but there is a rare form in which there is subluxation and deformity of the mtp joints; this is sometimes known as Launois's deformity (Csonka 1965).

Reactive arthritis was considered to be present in three skeletons at Barton, one each from the early and late periods, and one which was unphased. The early case (sk. 893, F1784) was in an adult skeleton of unknown sex which was represented only by the distal tibias and fibulas and some of the tarsal bones. There was bilateral sub-talar fusion and the right calcaneus and cuboid were also fused. The fusion was associated with spiky new bone and calcaneal spurs, and there was periosteal new bone on the right tibial shaft. This combination of features was very suggestive of ReA and the diagnosis was confirmed on X-ray.

The case from the late period was a virtually complete skeleton from a male of 45 (sk. 1937, F7009). The joint surfaces of the right first mtpj was expanded with marginal erosions and the right first mcpj was similarly affected. At both sites the lesions were accompanied by rough, spiky new bone and although there were no other stigmata of ReA, the X-ray appearances suggested this was the most likely diagnosis.

The final case was an adult that could be neither aged nor sexed; the skeleton (sk. 1851, context unknown) was in poor condition and only about half survived. Erosions were present around the right first mtpj and the dip was expanded and eroded. Following radiography, ReA seemed the most probable diagnosis.

The crude prevalence of ReA in the early and late periods was 0.47% (95% CI 0.13–1.70%) and 0.34% (95% CI 0.06–1.89%), respectively, showing no significant difference in occurrence over time.

Uncategorized Erosive Arthropathy

One further skeleton (sk. 1937, F7009) from the late period had an erosive arthropathy that could not be categorized. The skeleton was that of a male of 45 and was in poor condition, with only about half being present.

Both feet were extensively fused. On the left, there was sub-talar fusion but most of the calcaneus was missing. The remainder of the tarsal bones were fused together, and the second and third metatarsals were fused to the tarsal. The first proximal phalanx was fused at about sixty degrees to its metatarsal, and the foot looked as though it were in high heels (Fig. 62).

On the right side the talus and calcaneus were missing, but all the other tarsal bones were fused with the second and third metatarsals. None of the phalanges was present but there was new bone on the distal joint surface of the first metatarsal, suggesting that this joint was involved in the process and may have progressed to ankylosis as the contra-lateral joint had done. The only changes in the hands were that one pip and one dip were fused; the carpal bones were normal and there were no other changes apart from OA of the pips and dips. Radiography did not help in the diagnosis in this case.

Fig. 62: Left foot from a male of at least 45 years of age at death (sk. 1937) with an undefined erosive arthropathy. The tarsus is extensively fused and the second and third metatarsals are fused to the tarsus; the first proximal phalanx is fused at about 60° to the first metatarsal. Photo: English Heritage

Gout

Gout results from a defect in purine metabolism which causes high levels of uric acid to accumulate in the blood. The symptoms of gout occur when crystals of monosodium urate deposit in intra- or extra-articular tissues and induce inflammatory changes in them (Pascual and Pedraz 2004). Acute gout is characterized by attacks of excruciating joint pain which may last several days. In a proportion of individuals, chronic gout develops in which collections of urate crystals and inflammatory tissue form tophi which produce erosions in and around joints, often with a characteristic overhanging edge, referred to by radiologists as a Martel hook; the overhanging edge represents bone which has formed over the tophus. The erosions have a punched-out appearance and a sclerotic border (Monu and Pope 2004). Individuals with chronic gout often have kidney disease which is caused by the deposition of urate crystals in the substance of the kidneys; if untreated it may lead to renal failure and death.
Changes in the skeleton do not occur until late in the disease and in about half the cases the first mtp joint is affected. Other sites commonly affected include the wrist, elbow and knee; the shoulder, scj and sijs are less commonly involved, and the hip and spine rarely.

Gout is known from antiquity and became particularly common in the eighteenth century, when lead poisoning was widespread (Ball 1971). The prevalence of primary gout[10] is less than 1%, but there is a strong genetic component and about a fifth of those with a

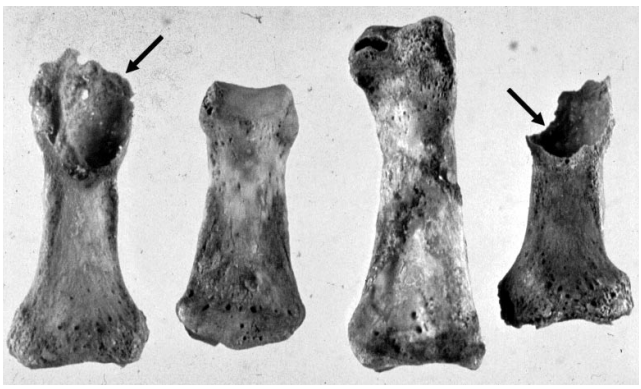

Fig. 63: Proximal phalanges of the hand from a skeleton of unknown age and sex (sk. 665). Two show very considerable distal expansion (arrowed) most probably caused by gout. Photo: Juliet Rogers

Table 36: Cases of gout at Barton, by phase

Skeleton number (context)	*Phase*	*Sex*	*Age*	*Location of lesions*
117 (F405)	B	Female	Adult	Right navicular, cuneiform, tmtj, mtpj
665 (F1527)	B	Unknown	Adult	Ankles, subtalar joints, most joints of both feet
1673 (F4653)	B/C	Male	Adult	Left acj; no hands or feet present
2180 (F5256)	B/C	Unknown	Adult	Right calcaneus, cuboid, navicular; both fifth mtpjs; two pips
122 (F410)	C	Female	25–34	Left third mcpj; tmtjs; first mtpj
499 (F3462)	C/D	Female	45+	Both acjs and scjs; right wrist, mcpjs and two pips
280 (F1210)	D	Unknown	Adult	Right acj, left fifth mtpj
1047 (F3154)	D/E	Male	45+	Many joints of both hands; right ankle, many joints of both feet
1766 (F4835)	E	Male	Adult	Both ankles, many joints of both feet; both wrists and many joints of both hands
1870 (F3987)	E	Male	25–34	Left first, second and third mtpjs; 3 pips

Table 37: Crude prevalence of gout at Barton, by period

	Crude prevalence (%)	*95% CI*
Early	0.94	0.38–1.96
Late	0.70	0.49–1.96

family history develop the disease, perhaps because of a mutation in the uromodulin gene (Bleyer *et al.* 2003).

Ten individuals were identified at Barton with lesions typical of gout; six were from the early period, two from the late period and two from the intermediate B/C phase. Four males and three females were identified while the three remaining cases could not be assigned a sex (Table 36). Skeleton 665 (F1527) from the late period exemplified the features found in gout. The margins of the tibial compartment of the knee, the ankle, sub-talar joint, calcaneo-cuboid joint, left and right cuneiforms, right first mtp and tarso-metatarsal joints displayed erosions with sharp margins and undercut edges. There were similar lesions on one carpometacarpal and one mcp joint and extensive involvement of three pips (Fig. 63). The other skeletons had similar lesions in typical sites and in addition two (sk. 499, F3462 and sk. 1673, F4653) had lesions of the acj which is rarely involved clinically. Skeleton 499 (F3462), a female of 45, also had lesions on the superior surface of a marginal osteophyte of a thoracic vertebra as well as being affected at more conventional sites.

The crude prevalence of gout in the early and late periods is shown in Table 37, from which it can be seen that there has been no significant change over the period of use of the cemetery.

Bunions

Bunions do not properly belong with the joint diseases but they are considered here because their appearance is sometimes confused with gout. In contemporary societies bunions most often result from wearing shoes that are too narrow (Coughlin and Thompson 1995) and the end result is that the big toe is displaced at the first mtp joint in the direction of the other toes, a condition that is known as hallux valgus;[11] The condition is much more common in females than in males. The bursa overlying the joint subsequently becomes inflamed and may cause pressure deformities on the medial side of the head of the first metatarsal. The lesions, which involve the juxta-articular area of the metatarsal head, are not strictly erosions; the cortex is usually intact and there are no overhanging edges to the lesions as there are in gout.

There were only three cases of hallux valgus at Barton (sk. 18, F206; sk. 788, F7813 and sk. 1887, F5008): all were from the early period, two females aged 45 or more at the time of death, and the third was an adult of unknown sex. The fact that so few cases were noted at this site perhaps attests to the sensible design of the footwear throughout the period, when indeed it was worn at all.

8. PALAEOPATHOLOGY III: INFECTIOUS DISEASES

Infectious diseases would certainly have been a major health problem for the population of Barton throughout the entire period that the cemetery was in use. Childhood infections such as measles, chickenpox and mumps would probably have been common and would have exerted a significant morbidity and mortality. Water-borne infections would also have been common and we have already suggested that gastro-intestinal infections may have been the cause of death of many of the children found in the assemblage. Both children and adults may have died from typhoid fever, and poliomyelitis would also have been present from time to time, as most likely would smallpox, especially in the later part of the period. There is evidence from the parish records for an outbreak of plague in 1593, in which year there was a huge increase in mortality in the town (see Chapter 15), and the town would most certainly not have escaped the ravages of the Black Death.

Most of the infections that affected the population would have left no evidence on the skeleton, however, either because the infection was in the soft tissue or the gut, or because the individuals died too soon in the course of the disease for the skeleton to become involved. Some of the cases of reactive arthropathy mentioned in the previous chapter are likely to have been the result of chronic gut or urinary tract infections and a small number of skeletons showed evidence of polio; these are discussed below.

There are relatively few infections that primarily affect the skeleton. Those that do include osteomyelitis, tuberculosis, syphilis and leprosy. Some others may affect bone as a secondary phenomenon including, for example, poliomyelitis and infections in soft tissues that overlie bones in which the periosteum is stimulated to form new bone. Lesions of this kind include varicose ulcers overlying the distal tibia, and chronic infections of the facial sinuses.

Osteomyelitis

Osteomyelitis is an infection of the bone and bone marrow which results in inflammatory destruction of bone. Many organisms may cause osteomyelitis but at least half the cases are caused by *Staphylococcus aureus* which is a bacterium that produces localized skin infections such as boils. The infecting organisms gain access to the bone by one of three methods: by spreading through the blood stream from an infection elsewhere (haematogenous spread); by direct spreading from adjacent infected soft tissues; and as the result of trauma.

The disease is commonest in children in whom haematogenous spread is the most usual mode of infection (Carek *et al*. 2001). The bacteria tend to settle out in the looped vessels that supply the growing ends of the long bones; the bacteria settle like silt in the loop of a meandering river. On this account the infection is commonest at the sites of rapid bone growth, particularly the bones around the knee, and the disease is commonest in children between the ages of 3 and 15.

The bacteria gain entry to the bone marrow which acts as a splendid culture medium and a great deal of pus is produced when the infectious agent is *S. aureus*. The infected bone may swell and new bone is laid down by the periosteum; it may form a thick collar around the bone which is known as an involucrum. Small pieces of bone may become detached from their blood supply and die, acting as a nidus for bacteria; these pieces of dead bone are known as sequestra and can usually only be demonstrated by X-ray. Finally, sinuses or cloacae are formed through which the pus drains to the outside from the infected bone and bone marrow. The combination of an involucrum (or periosteal new bone), cloacae and sequestra is absolutely characteristic of osteomyelitis, allowing the diagnosis to be made with complete confidence. The disease may last for many years and death may result from spread of the infection to other organs including the brain, the meninges, or the kidney.

Although the long bones are most commonly affected, osteomyelitis may occur in the vertebra and in the joints. Vertebral osteomyelitis is more common in adults than in children and is also known as discitis. Joints may become infected as the result of trauma or by spread from an adjacent infected bone. Cases of osteomyelitis of the long bones, the vertebrae and the joints were all seen at Barton, in approximately equal numbers.

Sixteen individuals were identified with osteomyelitis of the long bones, and in all but two cases, the femur, tibia or fibula were affected; in the remaining cases the ribs (sk. 1678, F4645) or radius (sk. 2799, F7632) were involved. Of the sixteen cases, ten were male, four female and two were juveniles. Of the eleven cases to which an age could be assigned, two were twelve-year-old children, two were aged between 15 and 24, three between 25 and 34, one between 35–44, and the remaining three were at least 45 years old at the time of death (Table 38).

One of the twelve-year-olds (sk. 1010, F7949) had a typical lesion in the right distal tibial diaphysis. The bone was expanded with a deposit of fine periosteal new bone on the surface of the bone. Cloacae were present at the epiphyseal margin and on the articular surface of the diaphysis. Much more florid changes were present in the other twelve-year-old (sk. 2159, F7471). An irregular deposit of new bone covered the whole shaft of the left femur except for the distal posterior third (Fig. 64). Multiple cloacae were visible, especially in

Fig. 64: Left femur from a 12-year-old child (sk. 2159) with florid osteomyelitis. There is a massive involucrum with multiple cloacae (two arrowed). Part of the shaft of the femur is shown towards the left of the picture with the context number written on it. Photo: English Heritage

Table 38: Cases of osteomyelitis at Barton, by age and sex

Skeleton number (context)	*Phase*	*Sex*	*Age*
4 (F84)	A	Female	Adult
156 (F3022)	A	Female	45+
307 (F7962)	A	Male	Adult
445 (F3420)	A	Male	17
1678 (F4655)	A/B	Male	25–34
57 (F997)	B	Male	Adult
1010 (F7949)	B/C	Unknown	12
2159 (F7471)	B/C	Unknown	12
2251 (F7154)	B/C	Male	45+
2252 (F7165)	B/C	Male	15–24
2754 (F7599)	D/E	Male	25–34
2785 (F7624)	D/E	Female	25–34
896 (F1491)	E	Female	Adult
1109 (F4085)	E	Male	Adult
1910 (F5032)	E	Male	45+
2799 (F7632)	E	Male	35–44

the posterior part of the proximal femoral shaft and a sequestrum was also visible; the X-ray changes confirmed this as a typical case of osteomyelitis, and this child's death was most likely to have been due to the disease.

One of the adults (sk. 1910, F5032) presented with a swollen right tibia which had an expanded grainy and striated surface but no cloacae were evident. The right ankle was involved in the process and there was a great deal of new bone in and around the inter-tarsal joints and the inter-metatarsal joints. The tibio-talar joint was affected with an eburnated and grooved articular surface. Osteoarthritis of the ankle joint is extremely uncommon without some predisposing factor, which in this case was the adjacent infection and the septic arthritis.

Six of the cases were from the early period and six from the late, with four from the intermediate Phase B/C. The crude prevalence of osteomyelitis for the early period was 0.39% (95% CI 0.18–0.86); for the late period it was 0.82% (95% CI 0.38–1.78). Although the prevalence in the late period is approximately twice that of the early period, the difference is not statistically significant ($p > 0.05$) and is almost certainly a chance finding.

Septic Arthritis

Fifteen skeletons were noted with septic arthritis. The causative organisms of this disease are similar to those causing osteomyelitis (Weston *et al.* 1999). The disease may present with destructive lesions on the joint surface, florid new bone formation, sometimes on the joint surface, and ankylosis of the joint. In some cases there is evidence of trauma to the joint, which presumably antedated the infection, and there may be signs of osteoarthritis because the normal mechanics of the joint have been altered. The age and sex of the cases, and the joints that were affected, are shown in Table 39.

Skeleton 329 (F3370) was one of the four examples of septic arthritis of the elbow. The joint was completely fused with extensive new bone formation, and cloacae could be seen. The elbow had been fractured and there was also a Colles fracture of the left wrist.

In skeleton 2169 (F7477) the right hand was involved. The right trapezoid was fused to the second metacarpal and the third, fourth and fifth metacarpals were fused to the hamate, capitate, triquetral and scaphoid, forming two separate blocks (Fig. 65). Radiography confirmed the diagnosis of a chronic joint infection, perhaps resulting from an infection in the hand.

The changes in skeleton 1091 (F3514) were unusual: the articular surface of the left first mtpj was obliterated with much flattening and expansion, giving rise to a 'hammer head' appearance. Radiographically, the changes seemed best explained by septic arthritis that had occurred during childhood; the first metatarsal was shortened with a fused sesamoid and a small proximal phalanx.

Seven of these cases were from the early period and three from the late, with four from Phase B/C and one

Table 39: Cases of septic arthritis at Barton, by age and sex

Skeleton number (context)	*Phase*	*Sex*	*Age*	*Joint affected*
170 (F3026)	A	M	45+	Right wrist
1992 (F7036)	A	M	Adult	Right ankle
1344 (F4530)	A/B	F	Adult	Right hip
329 (F3370)	B/C	M	Adult	Left elbow (following fracture)
439 (F3416)	B/C	M	Adult	Left hand, wrist
1505 (F4751)	B/C	F	20	Left hip (probably acquired during infancy)
1586 (F4768)	B/C	F	Adult	Right elbow
1178 (F1489)	C	F	25–	Right sij
775 (F7809)	C/D	U/K	Adult	Right mid-tarsal joints
230 (F1177)	D	U/K	10	Right elbow
1015 (F4040)	E	M	35–	Right elbow
1091 (F3514)	E	M	25–	Left mtpj
1918 (F5040)	E	M	25–	pipjs
2577 (F7406)	E	M	Adult	pipjs
2169 (F7477)	0	F	35–	Right wrist and carpus

sij = sacro-iliac joint
mtpj = metatarso-phalangeal joint
pipjs = Proximal inter-phalangeal joints

Fig. 65: Right hand of a female aged 25–34 at death (sk. 2169). The second metacarpal is fused to the underlying trapezoid and the third to fifth metacarpals are fused to the carpals forming two blocks. The most probable cause was septic arthritis. Photo: English Heritage

unphased. The crude prevalence in the early period was 0.46% (95% CI 0.22–0.95) and in the late period, 0.41% (95% CI 0.14–1.20), indicating that the frequency of this condition had not altered over time.

Discitis

Discitis is a special form of osteomyelitis and in the present day is primarily a disease of adults, with most patients being over 50 years of age at diagnosis; men are affected about twice as often as women. Again, *S. aureus* is the most common infectious agent (Jevtic 2004).

Table 40: Cases of discitis at Barton

Skeleton number (context)	*Phase*	*Sex*	*Age*
1330 (F7291)	A	Male	17
2692 (F7543)	A/B	Female	25–34
701 (F7755)	C/D	Unknown	8
2494 (F4945)	C/D	Female	25–34
779 (F1446)	D	Unknown	5
758 (F7795)	D/E	Unknown	Adult
867 (F1483)	D/E	Male	45+
1295 (F3262)	D/E	Male	15–24
1877 (F3996)	D/E	Female	25–34
1103 (F4082)	E	Male	45+
1312 (F3272)	E	Male	17
2594 (F7420)	E	Male	45+
2775 (F7614)	E	Unknown	Adult

The disease is recognized in the skeleton by the presence of focal erosions in the end plate of one or more vertebrae, most usually in the thoracic or lumbar regions. It is not always easy to differentiate discitis from metastases, or sometimes from very large or serpiginous Schmorl's nodes. In each instance, discitis was diagnosed in the Barton assemblage only on the basis of radiographic findings. Thirteen cases were identified, ranging in age from a five-year-old child to three adults aged over 45 years; males slightly outnumbered females (Table 40). As may be seen from this table, all but two of the cases came from the early period and although the crude prevalence was more than twice that in the late period, the difference again was not statistically significant. The crude prevalence for the early period was 1.05% (95% CI 0.59–1.88%), and for the late period: 0.44% (95% CI 0.12–1.60 %).

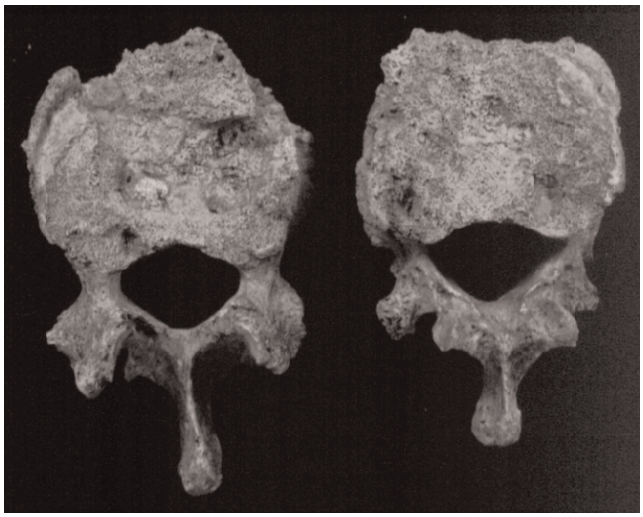

Fig. 66: Eleventh and twelfth thoracic vertebrae of a young man (sk. 1312) showing extensive changes in the end plates consistent with discitis. Photo: Juliet Rogers

Typical changes of discitis were shown by a male aged between 17 and 24 (sk. 1312, F3272) in whom three vertebrae were involved, the eleventh and twelfth thoracic and the first lumbar (Fig. 66). The inferior end plate of T11 was deformed by a crescent-shaped lesion with a scalloped appearance situated posteriorly. On T12 the lesions also affected the inferior end plate which had an uneven 'billowy' surface with deep erosions cutting into it. The vertebral body of T12 showed some inflammatory changes, with a coarse uneven deposit of new bone and extensive pitting. These changes were especially prominent on the right-hand side. On L1 the superior end plate was involved, with a major defect with scalloping and sinuses connecting to the spinal canal. The radiograph showed typical signs of infection.

A skeleton of a 17-year-old male (sk. 1330, F7291) had changes affecting the lower five thoracic and the first lumbar vertebrae, which appeared to be due to tuberculosis. All the vertebrae had scalloping of the anterior bodies and on T8 and T12 the end plates had similar lesions. Radiographically, however, the appearances were typical of an infective process but it was not thought that they were caused by tuberculosis.

Tuberculosis (TB)

Tuberculosis in past populations would have been caused by one of two organisms, *Mycobacterium tuberculosis* or *M. bovis*.[1] The former affects only humans and is primarily a disease of the lungs. It is spread by the propagation of infected droplets which are inhaled and which then set up an immune response which, in most people, becomes dormant. After a variable period, the disease may be reactivated and spread throughout the lungs and to other parts of the body, including the skeleton. *M. bovis* causes disease in a wide range of animals including, most importantly for the human economy, cattle (Montali *et al.* 2001). Humans contract bovine TB by drinking infected milk or eating infected dairy products. The bacteria are taken up from the gut into the lymphatic system and spread from there to the lymph nodes and to other organs, again including the skeleton. Neither organism has a particular propensity for the skeleton although in some older texts and bone reports there may be a reference to the fact that bovine TB *is* more likely to affect bone; this is not true.

The number of infected individuals in whom the skeleton is affected is very variable but always a minority (Liyange *et al.* 2000). In half of those with skeletal TB it is the lumbar spine that is affected. Any other bone may become infected, however, and there is a form in children in which the fingers are involved (Yoon *et al.* 2001); generally not more than one site is affected in any individual. Within the affected vertebrae destructive lesions are formed, almost always only in the anterior part of the vertebra and without a significant amount of new bone formation, and no cloacae. As the disease progresses, the substance of the vertebral body may be completely replaced with tuberculous material and this will lead to collapse and fusion of adjacent vertebrae. The collapse causes a sharp angulation of the spine, so-called angular kyphosis of Pott's disease,[2] which is very characteristic of tuberculosis.[3]

Tuberculosis may affect joints, in which case they become fused but without the formation of any new bone. The large joints such as the hip and the knee are commonly involved and so is the wrist (Malaviya and Kotwal 2003).

Fig. 67: Part of thoracic spine from a male aged 35–44 at death with typical changes of tuberculosis (sk. 1454). The bodies of T7 and T8 are fused, with that of T8 having almost completely disappeared. Photo: English Heritage

Only one skeleton at Barton (sk. 1454, F3727) was identified with changes that could definitely be ascribed to TB. This was a male aged 35 from the intermediate Phase A/B. The skeleton was virtually complete and had a typical Pott's spine. The anterior portions of the bodies of the sixth, seventh and eighth thoracic vertebrae are eroded and T7 and T8 are fused together. The body of T7 is much diminished and that of T8 has almost completely disappeared (Fig. 67). No other changes were present in the skeleton apart from an erosion situated on the lesser tuberosity of the left humerus, which was not related to the TB.

One other skeleton (sk. 2427, F5389), a female from the early period, aged between 17–24, had changes in the hip which might possibly have been caused by TB. The acetabulum was obliterated and the femoral head was grossly abnormal, appearing only as a small peg. No other changes were noted in the skeleton and it was not possible to come to a definitive diagnosis in this case.

There appear to be remarkably few cases of TB at Barton, given that it was a common disease in the past. In the latter part of the period covered by the phase from which the single case derived, tuberculosis was very prevalent. In the London *Bills of Mortality,* consumption – which would have been predominantly caused by TB – accounted for approximately a fifth of all deaths in non-plague years,[4] and the disease became increasingly common during the eighteenth and early nineteenth centuries. It would be surprising if the population of Barton had been immune from the general upsurge in the disease, and either the disease was particularly virulent so that those infected died quickly, or the expression of the disease may have been different, with many fewer individuals going on to develop skeletal lesions. Had it been possible to conduct either DNA or mycolic acid studies on a sample of the Barton assemblage it is likely that markers of the disease would have been found in some apparently normal skeletons, as has been found elsewhere (Zink *et al*. 2003).

Poliomyelitis

Poliomyelitis is caused by an RNA virus that is spread via the faeco-oral route or through contaminated water. The virus replicates in the throat and intestinal tract and then invades the lymphatic system and the blood and finally, in about five per cent of patients, infects the central nervous system. The motor cells in the spinal cord may be destroyed and the muscles supplied by these nerves then become paralysed. If the muscles of respiration are affected, death will ensue unless artificial respiration can be given (Adams *et al*. 2001). In young children with paralysed limbs, the bones will fail to develop normally and will be shorter and more slender than those of the unaffected limbs.[5]

There were five skeletons at Barton with marked inequality of limb or other bones, which seemed likely to have been caused by polio, three from the late period and two from intermediate Phase B/C (Table 41).

Table 41: Possible cases of poliomyelitis at Barton

Skeleton number (context)	*Phase*	*Sex*	*Age*	*Affected area*
1423 (F3707)	A	F	Adult	Left leg
1425 (F3683)	A	M	28	Right leg
1964 (F5102)	A	M	Adult	Left side; spinal scoliosis
1409 (F3694)	B/C	M	35–45	Right leg; very slight lumbar scoliosis
1505 (F4751)	B/C	F	20	Left side

Table 42: Measurements (in mm) from four cases of poliomyelitis at Barton

Skeleton number (context)	*FeLL*	*FeLR*	*FeD1L*	*FeD1R*	*FeD2L*	*FeD2R*	*TiLL*	*TiLR*	*TiD1L*	*TiD1R*	*TiD2L*	*TiD2R*
1409 (F3694)	424	401	23	23	27	27	350	327	31	21	22	18
1425 (F3683)	450	394	21	14	21	19	347	320	32	17	20	15
1505 (F4751)			15	23	26	29						
1964 (F5102)	445	476	28	23	29	30	366	395	24	31	18	27

FeL = maximum length of femur
FeD1 = medio-lateral diameter of proximal femoral shaft
FeD2 = antero-posterior diameter of proximal femoral shaft
TiL = maximum length of tibia
TiD1 = antero-posterior diameter of tibial shaft at level of nutrient foramen
TiD2 = medio-lateral diameter of tibial shaft at level of nutrient foramen
Suffix L = left and R = right

Fig. 68: Both femora from a young man with poliomyelitis (sk. 1425). The right femur is shorter and much more gracile than the left and there is a degree of coxa valga. It is likely that he contracted polio before puberty. Photo: English Heritage

The characteristic changes in the skeleton of an individual affected by polio are exemplified by those seen in sk. 1425 (F3683), a young man of between 17 and 25 when he died. All the bones of the skeleton were gracile, but those on the left appeared within the normal range. The right lower limb bones, however, had thin shafts with a marked curvature of the femur and fibula (Fig. 68). There was a marked increase in the angle between the femoral neck and the shaft (coxa valga), which is a typical finding in a neuromuscular disorder. The left patella was larger than the right and both acetabula were shallow and the left sciatic notch was rather feminine in shape. Some abnormalities were also noted in the spine. The majority of the bodies of the thoracic and lumbar vertebrae were 'fish shaped', that is, they had a prominent waist and an increased height. It is not unusual for curvature of the spine to develop under these circumstances where there is a substantial discrepancy between the length of the legs. The discrepancies in the lengths of the affected limb bones in the four cases in which comparisons could be made are shown in Table 42.

Although other possible causes of these changes should be considered, such as trauma or congenital disease, polio seems much the most likely.

Chronic Sinusitis

Chronic sinusitis is an extremely common condition in modern life and accounts for a substantial morbidity, especially in areas where atmospheric pollution is high (Anand 2004). It generally has no serious complications although it may exacerbate pre-existing asthma and, rarely, result in brain abscesses or meningitis. Any of the sinuses in the head may become infected but it is the maxillary sinuses that are most frequently involved; almost three-quarters of all sinus infections are caused by three organisms, *Streptococcus pneumoniae*, *Haemophilus influenzae* and *Moraxella catarrhalis* (Kennedy 2004).

In the skeleton, sinusitis is recognized by the presence of periosteal new bone on the floor of the sinus. It is not reported very often in human remains because the new bone will be visible only: if the sinus is broken, so that its interior can be observed; if X-rays of the skull are taken routinely; or if the sinuses are explored with an endoscope, as Calvin Wells did some years ago (Wells 1977). Neither of these ancillary investigations was routinely undertaken at Barton and so sinusitis was noted only when the facial bones were broken; this report of the few skeletons with the condition should not be taken as anything like a definitive account of the true prevalence of the disease.

Seven skeletons were noted to have changes within the sinuses that were probably due to chronic sinusitis. These included plaques of new bone, sometimes smooth and sometimes irregular. The changes were noticed – with one exception noted below – only because the facial bones were broken to permit visual inspection of the infected sinuses; in most cases it was

Fig. 69: Left frontal bone from a male aged at least 45 years at the time of death (sk. 2274). There is a depressed area above the orbit with a number of openings into the frontal sinus which subsequently became infected. The lesion almost certainly resulted from a blow to the head. Photo: English Heritage

Table 43: Cases of chronic sinusitis at Barton

Skeleton number (context)	*Phase*	*Sex*	*Age*	*Infected sinus*
1496 (F4607)	A	M	35–45	Left maxillary
1984 (F7037)	A	F	35–45	Left maxillary
2668 (F7534)	A/B	M	45+	Right maxillary
2013 (F7070)	B/C	F	Adult	Left maxillary
2274 (F7183)	B/C	M	45+	Left frontal
1551 (F3796)	C/D	Unknown	45+	Left maxillary
1026 (F4047)	E	F	45+	Right maxillary

not possible to see into the other facial sinuses nor, since the post-cranial skeleton was poorly preserved, could signs of infection be noted elsewhere. The list of skeletons with sinus infection is shown in Table 43.

The appearances in one of the seven (a male of at least 45 years of age at death: sk. 2274, F7183) was different from the rest. Above the medial end of the left orbit adjacent to the frontal sinus there was a 15 mm^2 linear depressed area with a linear opening superiorly and small perforations inferiorly (Fig. 69). There was a raised area of bone around the depression. A radiograph showed signs of infection of the left frontal sinus and it seemed likely that this was secondary to injury. The fact that there was a healed cut across the left coronal suture gave support to this conclusion.

Periostitis

More nonsense is written about periostitis than almost any other affection of the skeleton and it is clear that its causes are imperfectly understood by many who pontificate about it. The periosteum is a membrane that covers the entire surface of a bone except where it is covered by articular cartilage, the synovial membrane, or where it forms part of a non-synovial joint such as the pubic symphysis. The function of the periosteum is to form bone and it is by this means that bones enlarge circumferentially, a process that persists after the epiphyses have fused and are no longer able to grow in length. Whenever the periosteum is stimulated its only response is to lay down bone; this may occur following, for example, trauma, burns, bleeding, tumour or infection. The term 'periostitis' is applied to any new bone formed by the periosteum, no matter what the stimulus, although it implies an inflammation of the periosteum, and in much of the palaeopathological literature it is taken to imply infection, particularly when found on the skeleton of juveniles. As may be seen from Table 44, however, there are many non-inflammatory and non-infectious causes of periosteal new bone (pnb).[6] It would be better to use a more neutral description of the new bone found in skeletal remains, especially as in the majority of cases the aetiology will be completely unknown. We suggest it is better to use the term periosteal new bone (pnb) when referring to human remains since it is neutral and has no aetiological implications.

Table 44: Some causes of periosteal new bone formation

Physiological in infants
Infant cortical hypertrophy (Caffey's disease)
Infections
  osteomyelitis
  syphilis
Trauma
Venous stasis
  varicose veins
Haemorrhage
  scurvy
Rickets
Burns
Tumours
  primary – osteosarcoma
  secondary – metastases
Leukaemia
Hypertrophic osteoarthropathy
Fluorosis
Hypervitaminosis A
Neurofibromatosis
Thyroid acropachy
Some congenital conditions
  Menkes' syndrome
  Camurati–Engelmann disease
Overlying soft tissue lesions

Periosteal New Bone on the Ribs

Periosteal new bone on the inner surface of the ribs is a relatively common finding and it has been suggested that it might be caused by tuberculosis (Roberts and Manchester 1995). If a tuberculous lesion were in the periphery of the lungs and affected the visceral pleura, then it might stimulate the periosteum. In fact, any peripheral lesion of the lungs such as lobar pneumonia, and any disease of the pleura, including pleurisy, a pleural effusion, blood or pus in the pleural space might involve the periosteum and lead to the production of pnb. An attempt to determine the cause of pnb on the ribs could be made if it were possible to specify which ribs were involved and relate this to the pleural and lung markings.[7]

Seven skeletons at Barton were noted to have pnb on the ribs, five males and two children; three of the cases were from the early period and four from the late

Fig. 70: Rib fragment from a young male (sk. 1977) showing a thick layer of periosteal new bone on the pleural surface (arrowed). Photo: English Heritage

Table 45: Cases with periosteal new bone on the pleural surface of the ribs at Barton

Skeleton number (context)	*Phase*	*Sex*	*Age*
1476 (F3735)	A	Child	1 year
1502 (F4575)	A	Male	20–25
1977 (F5114)	A	Male	15–25
2627 (F7640)	A	Male	45+
774 (F7808)	C/D	Child	5
2193 (F7140)	C/D	Male	35–45
1922 (F5050)	E	Male	45+

(Table 45). In the case of the children, pnb was also noted on limb bones but the cause of the pnb could not be determined in either, or indeed, in any of the other cases. One of the adults (sk. 1977, F5114) had a layer of new bone at least 1–2 mm in thickness on sixteen rib fragments from both sides of the chest (Fig. 70). In addition, there was pnb distributed symmetrically on both humeri, femora, tibiae, distal fibulae, calcaneii and the left fourth and fifth metatarsals. Apart from the periosteal new bone, no other changes were noted on X-ray and the cause of this widespread periosteal reaction remains undetermined.

Periosteal New Bone at Other Sites

In total, 193 individuals were found with pnb at sites other than the ribs: 74 males, 41 females, 47 juveniles and infants, and 31 adults of unknown sex. The prevalence of pnb in the early and late periods is given in Table 46. The prevalence in the later juveniles and infants is significantly higher than in those from the early period, although the reason for this is unclear. None of the other differences is due to anything but chance.

Virtually all the pnb was confined to three bones, the tibia, fibula and femur; in ten juveniles, pnb was noted on the skull. Of the three lower limb bones, the tibia was far more often involved than the other two: pnb on the tibia accounted for 53.8% of the total, the fibula for 24.1% and the femur, 13.1%. This is the relative frequency noted at many other archaeological sites and so entirely conforms to expectation. On the tibia and fibula pnb is often noted on the distal ends of the shaft, frequently associated with the distal tibiofibular joint, suggesting that repetitive trauma of the ligaments binding the joints combined with repeated small bleeds may be the cause. Periosteal new bone is also commonly found around the lateral malleolus, which leads to the conclusion that it may have been the result of venous stasis in varicose veins, while the other common site is on the subcutaneous border, consistent again with repeated mild trauma.

Periosteal New Bone as a Stress Marker

Periosteal new bone is increasingly being seen by those who examine human remains as a stress marker. It is not always immediately obvious what the investigators mean by the term 'stress', except that it does not mean what is sending countless thousands of our contemporaries to seek counselling. The usual interpretation is that it refers to an insult during childhood, most often

Table 46: Crude prevalence of periosteal new bone at Barton, by period

		Males	*Females*	*Unknown adults*	*Infants and juveniles*	*Total*
Early	Prevalence	11.1	6.7	5.8	4.7	7.1
	95% CI	8.3–14.9	4.4–10.0	3.5–9.5	3.0–7.3	5.9–8.7
Late	Prevalence	15.5	6.9	6.7	12.0	10.5
	95% CI	10.8–21.7	4.0–11.6	3.5–12.7	8.0–17.7	8.3–13.1

an episode of infectious disease. There are a number of other stress markers including cribra orbitalia, taken as an indication of iron-deficiency anaemia; enamel hypoplasia, taken to indicate an episode of indeterminate illness during childhood; and Harris's lines which are radiologically dense lines usually found on the distal tibia, and taken to indicate a period of arrested growth. There is no clinical evidence that cribra orbitalia is related to iron-deficiency anaemia, and Harris's lines by no means always equate with illness. Periosteal new bone – as Table 44 shows – has many causes, and it is important to remember that in young children it can be physiological, although since the presence of pnb can present a pit into which the medically qualified can fall, it is perhaps excusable that those without such training may sometimes overlook this fact.[8]

If pnb and the other putative stressors have an adverse effect on the child, then one could hypothesise that: a) stressed children might die at a younger age than those who are not stressed, and b) that this might adversely affect their growth so that they are shorter than their non-stressed contemporaries of similar age. To test these hypotheses, all the children with pnb, cribra orbitalia and enamel hypoplasia were selected and compared with all children with none of these markers. The mean age at death was calculated and where limb bone measurements were recorded these were compared between the two groups. To utilize all the limb bone measurements, they were standardized with Maresh's 1955 data to obtain a δ_1 value as described in Chapter 3.

Mean Age at Death

A total of 47 children had pnb, 87 had cribra orbitalia and 10 had enamel hypoplasia. The first observation that could be made was that each condition seemed to occur independently of the other. Thus there was only a single child with all three markers, and six with two of the three (Fig. 71). The group of 'stressed' children thus totalled 137, of whom age was known for only 60. The 'non-stressed' group included a total of 282 for whom age at death was known, excluding fetuses which would not be expected to show any of the stress markers; there were none in the 'stressed' group. The mean age of death for the non-stressed and stressed groups was 5.7 and 5.5 years, respectively, a difference

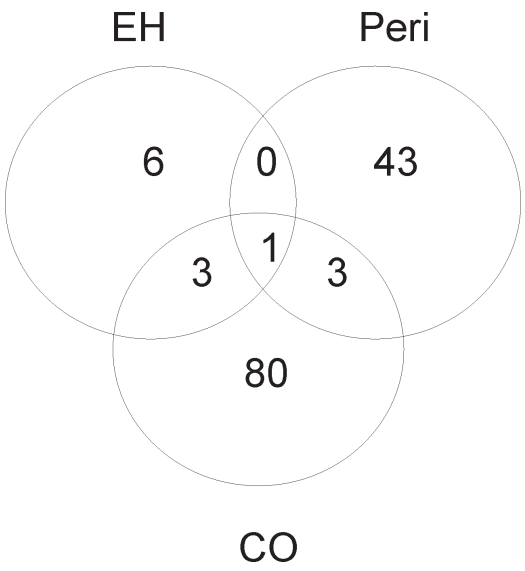

Fig. 71: Distribution of enamel hypoplasia (EH), cribra orbitalia (CO) and periostitis (Peri) among juveniles at Barton

that was, of course, not remotely significant ($t = 0.39$, $p = 0.698$).

Long Bone Length

When the values of δ_1 were compared for the two groups, it was found that 52% of the non-stressed group had a δ_1 value less than 1 (indicating that they were smaller for age than the reference group) compared with 55% of the stressed group. Again, these differences were not significant ($\chi^2 = 0.103$, $p = 0.748$). It is possible that, having recovered from the insult that affected them, the 'stressed' children may have caught up with their non-stressed counterparts; this would explain why they were not shorter.

Nevertheless, the results indicate that in this group of children, at least, the appearance of so-called stress markers in the skeleton did not result either in an earlier death, on average, or retardation of growth compared with children whose skeletons did not bear evidence of these markers. Their utility as indicating children in worse health or placed at some disadvantage thus seems extremely dubious; merely to say that their presence in the skeleton is sufficient indication, without some external validation, is perverse.

9. PALAEOPATHOLOGY IV: TRAUMA

Signs of trauma, whether deliberate or accidental, are commonly found on human remains. Fractures are by far the most frequent form of trauma found in assemblages of the general population but there may also be signs, such as cut-marks or the end results of soft tissue damage, that have subsequently ossified or left other marks on the bone. Some damage that is found on the skeleton is caused after death; during the period of burial the weight of the soil on the skeleton frequently results in the ribs fracturing, and the skull and pelvis being crushed. Bones may also be damaged by grave diggers or by subsequent building work. Nor are they always safe during excavation, and the pick and the trowel may leave their marks on the bones. It may be difficult to distinguish trauma that took place around the time of death, before any healing could have taken place, from damage suffered after burial and it is evident that some *peri-mortem* trauma will not be recognized. Breaks to the bone that have occurred during or after recovery can easily be recognized because the broken end of the bone will be a much lighter colour than the rest of the skeleton.

In this chapter an account will be given of broken bones, of wounding, and of some special forms of trauma, including spondylolysis and osteochondritis dissecans. Although they are not strictly evidence of trauma, Schmorl's nodes will also be considered here.

Fractures

Fractures are defined as a break in the continuity of bone, cartilage or both, almost always associated with soft tissue injury. There are several ways in which fractures can be categorized; they may be closed – when the skin over the fracture is not broken – or open, when it is. A simple fracture is one in which there are only two fragments and a comminuted fracture is one when there are more than two. Spiral, transverse, depressed and crush describe the appearance of the fracture and may also give an indication of the nature of the force that caused the fracture to occur. A stress fracture is one that occurs in a bone subjected to repeated loading and will be encountered in the section on spondylolysis; a pathological fracture is one that occurs in a bone which is already diseased: it may occur through the site of a secondary deposit of a malignant tumour, for example.[1]

The healing of a fracture follows an orderly series of events which enable those who examine human remains to tell whether it was of recent origin or not. The state of a fractured bone will also provide some information about the treatment that the injured person experienced since, for proper healing, the bone fragments must be re-aligned to approximately their normal anatomical position if they have become displaced – this is known as reducing the fracture[2] – and the bone must be immobilized for several weeks, usually with the application of a splint, and the injured limb must be introduced back into use gradually. Following a fracture of a femur, this whole process may take six to eight weeks, during much of which time the individual will be dependent upon others to care for him or her. The fact that a substantial number of fractures that are seen in assemblages of human remains are well healed and in good alignment suggests that the knowledge about bone setting was well established many hundreds of years ago and that individuals with this skill were to be found in most societies.

There are many complications of fractures, the most serious being death, usually as the result of damage to vital organs or to extensive blood loss. Other complications include infections, especially following an open fracture, non-union of the fracture, most often because the injured limb has not been immobilized, disability, due, for example, to the fractured bone not being reduced, or to nerve damage, and osteoarthritis which develops several years afterwards in a bone which is not properly reduced, or – almost invariably – if the fracture goes into a joint. Some of these complications can be detected in the skeleton but in most assemblages there are relatively few and most fractures seem to heal in good alignment and few become infected.

In the skeleton a fracture is usually very easy to diagnose, especially if it has not completely remodelled. A very old fracture may show as nothing more than a slight swelling on a seemingly intact bone but radiography will usually show evidence of the fracture line. Wherever a fracture was suspected at Barton its presence was confirmed by radiography and only diagnosed when there was positive radiographic evidence.

A total of 152 individuals had sustained 186 fractures between them. Of these, 99 were men, 36 were women and 15 were adults who could not be assigned a sex; there were only two children with evidence of fractures, both of the skull. This is not to say that some of the adults had not sustained their fractures during childhood because, unless the fracture occurred shortly before death, it is almost impossible to tell at what age it was sustained.

The first child with a skull fracture (sk. 2080, F7105) was between 2 and 3 years of age when it died. There was a depressed fracture approximately 50 × 50 mm in area, overlapping the left posterior parietal and the anterior occipital bones. The internal table of the skull was pushed in but the fracture was well healed and there was no other pathology in the skeleton. This type of fracture might have resulted from a blow or from the child falling onto a blunt object; it was certainly not implicated in its death.

Table 47: Distribution of fractures at Barton, by sex and anatomical site

*Anatomical site**	*Male*	*Female*	*Adult*	*Juvenile*	*Total*	*Per cent of total*
Skull	2	1	0	2	5	2.7
Spine	7	1	2	0	10	5.4
Rib	44	15	0	0	59	31.7
Clavicle	5	1	1	0	7	3.8
Humerus	3	1	0	0	4	2.2
Radius	7	5	1	0	13	7.0
Ulna	7	4	4	0	15	8.1
Metacarpals	13	3	2	0	15	8.1
Proximal phalanges	2	1	0	0	3	1.6
Femur	5	1	0	0	6	3.2
Patella	2	0	0	0	2	1.1
Tibia	12	3	2	0	17	9.1
Fibula	14	4	4	0	22	11.8
Metatarsals	4	0	2	0	6	3.2
Other foot bones	2	0	0	0	2	1.1
Total	129	40	17	2	186	100

*Fractures of the ribs and vertebrae have been counted as a single occurrence, irrespective of how many bones were broken

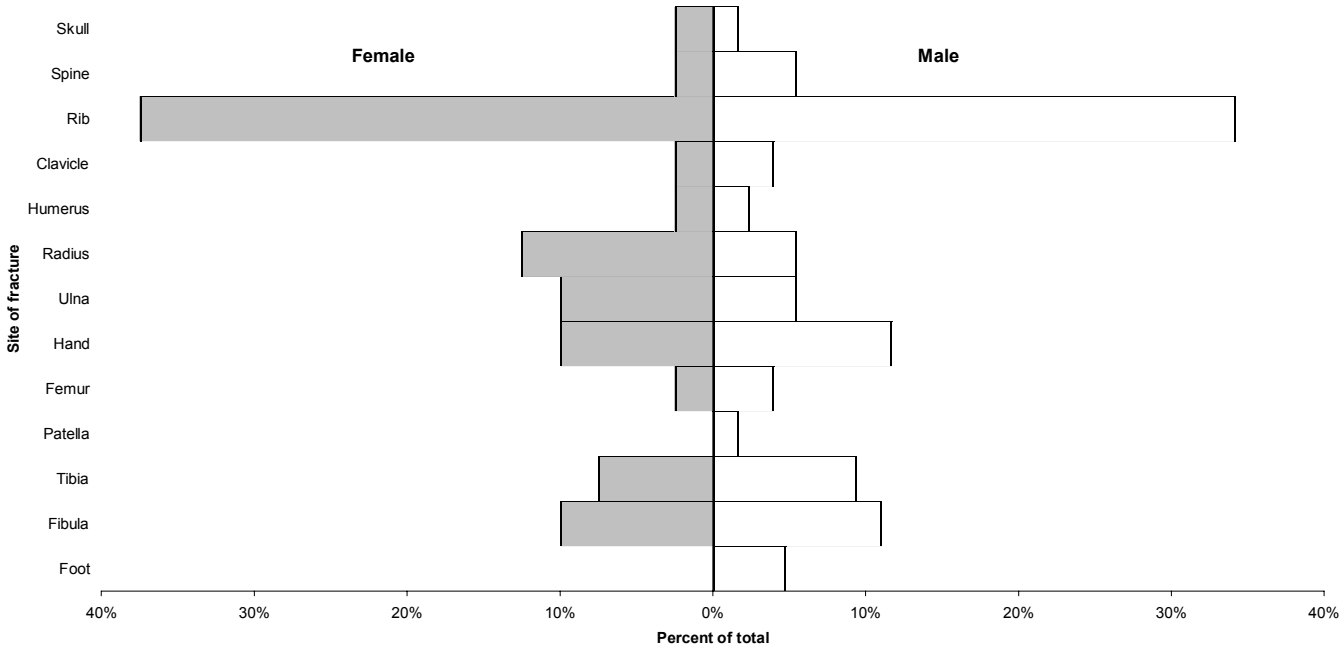

Fig. 72: Distribution of fractures at Barton, by sex

The second child (sk. 2302, F4977) was slightly older when it died, probably about 7 years of age. There was a small depressed area just supero-lateral to the right side of the glabella, and about 10 mm in length. On the endocranial surface of the skull what appeared to be a healed linear fracture ran along the line of the metopic suture, across the lesion visible from outside before deviating to the right for about 10 mm. The most likely explanation for this fracture – which was also well healed – was a blow to the head but, as with the other child, it did not seem to be the cause of death and the remainder of the skeleton was normal.

The most common fracture by far in the adults was a rib fracture, as is clear from Table 47 and Fig. 72. When listing the number of fractures, rib fractures have been counted only once no matter how many were involved. In fact of the 44 men with rib fractures, 15 had more than one broken: one man (sk. 1562, F3802) had a total of eight broken ribs including two that were still fusing, indicating that they had been broken shortly before his death. Of the 15 women with rib fractures, eight were single fractures only. Rib fractures are commonly the result of falls or brawls; in the latter case, usually from a kick when the individual is on the floor (Sirmali *et al*. 2003), but can also follow a bout of coughing on occasion. It is not difficult to suppose that some of the men at least – and perhaps some of the women too – injured their ribs in a fight.

The fibula was the second most commonly broken bone, the injury being most frequent at the distal end

Fig. 73: Bilateral, well healed fracture of the ulna in a male of at least 45 years of age at death (sk. 800). The fractures are remodelled and probably occurred several years before death. This individual also had numerous other fractures (see text for details). Photo: English Heritage

where it is susceptible to knocks on the ankle. It was seldom displaced unless it was involved with the tibia, as it was in four cases. The tibia and the bones of the forearm were relatively frequently broken, and so were the metacarpals. Skull fractures were uncommon, occurring less often than wounds to the head, as will be seen below. The distribution of fractures between the two sexes was very similar although, as shown in Fig. 72, women tended to sustain injuries to the radius and the ulna proportionately more often than men.

Although a few individuals had more than one fracture, there were only two cases (sk. 800, F3096 and sk. 1365, F3650) in which multiple fractures were noted. The first was a male of at least 45 at the time of his death. He had a linear fracture of his left parietal bone, a single rib fracture, well-healed and well-aligned fractures of both ulnae (Fig. 73), a fracture of the neck of the left humerus with the head of the humerus tipped back posteriorly, and a fracture of the fifth right metacarpal. In addition he also had injured his right first metacarpo-phalangeal joint and the proximal phalanx was fused to the head of the metacarpal. The fifth metacarpal is frequently broken during a fist fight (Kermad *et al*. 2002) and it is tempting to suppose that at least some of these injuries were sustained during a serious physical encounter. The second was a young man aged between 25 and 34 when he died. He had bilateral transverse fractures of the tibia, both of which had healed well and with minimal displacement. He also had an associated fibular fracture and bilateral fractures of the fifth metacarpal, both of which had healed with thickening and some shortening.

Among the men, the fifth metacarpal was the one most commonly broken; this was the case in five of the thirteen noted, and in two other cases, the fourth – the other bone of the hand frequently broken during a fist fight – was broken. This suggests that these injuries were due to the equivalent of a bar-room brawl. Two of the three metacarpal injuries in the women were also to the fifth metacarpal, possibly sustained when slugging their husbands returning home from a rowdy night out!

In most cases it is not possible to determine the nature of the accident that led to the fracture. There were three Colles fractures of the radius or ulna which are almost always the result of a fall onto an outstretched hand, however, and there were several crush fractures of the vertebrae, again the most likely result of a fall, although one thoracic vertebral fracture in an adult female (sk. 458, F3427) was related to osteoporosis.

Another type of fracture that is often caused by a fall is that which involves the tibial plateau, and there were four of these at Barton. This is not a common type of fracture and although the prevalence is not known exactly it probably does not account for more than 1% of all fractures (Anglen and Healy 1988); at Barton the prevalence was slightly more than 2%, so that it was rather more frequent than expected. Two of the fractures (sk. 66, F345 and sk. 2139, F5246) were type 3,[3] that is they involved depression in the lateral compartment without a split in the proximal tibia. In one case (sk. 66, F345), the skeleton was osteoporotic and the fracture was depressed about 10 mm below the margin of the lateral plateau (Fig. 74). A third fracture (sk. 213, F463) was type 2, in which a fracture of the lateral compartment is associated with a split in the tibia, and the fourth (sk. 2628, F7509), was a type 5, that is, there were split fractures in both the medial and lateral compartments.[4] Fractures of the tibial plateau are frequently associated with soft tissue injuries including the ligaments within and without the knee joint (Bennett and Browner 1994), and these lead to

Fig. 74: Tibial plateau fracture in a female aged between 25 and 34 at death (sk. 66). The fracture line is clearly seen in the lateral plateau and the joint surface is depressed by about 10 mm. Photo: English Heritage

osteoarthritis in a high proportion of cases (Honkonen 1995). In all the instances at Barton the fractures were well healed with minimal displacement and with no evidence of secondary osteoarthritis.

Prevalence of fractures

The crude prevalence of fractures at Barton was 10.1% (95% CI 8.6–11.7%). For males the prevalence was 15.4% (95% CI 12.8–18.4) and for females, 6.4% (95% CI 4.7–8.8%); this difference between the sexes is statistically significant. There are also differences in the prevalence of fractures between the early and late periods as can be seen in Table 48. In the late period, the prevalence of fractures increases in both sexes, although the differences are not significant and the male-to-female ratio is only significantly different in the early period. Whether these data are a reflection of a real increase in the frequency of fractures in the later period is hard to say; the relatively small numbers involved result in the wide confidence limits, again demonstrating how difficult it is to make definitive statements about health and disease in the past, even with what is – by osteological standards – a very large assemblage.

Table 48: Crude prevalence of fractures (% and 95% CI) at Barton, by period

	Early	*Late*
Male	12.8 9.8–16.5	18.7 13.6–25.2
Female	5.5 3.5–8.5	10.0 6.3–15.6

Complications of fractures

Given the potential for complications following fractures, remarkably few were evident among the Barton assemblage. There were three bones that had not united, one rib, one fibula and one clavicle; in each instance a pseudarthrosis – a false joint – had formed. Several fractured bones had healed with some displacement, but generally it was not very great. One exception involved a spiral fracture of the tibia and fibula that had fractured into the joint (sk. 81, F1008). The fracture had healed with considerable angulation and the ankle joint was greatly inverted: the individual would have found his or her mobility somewhat impaired and would have walked with a limp (Fig. 75).

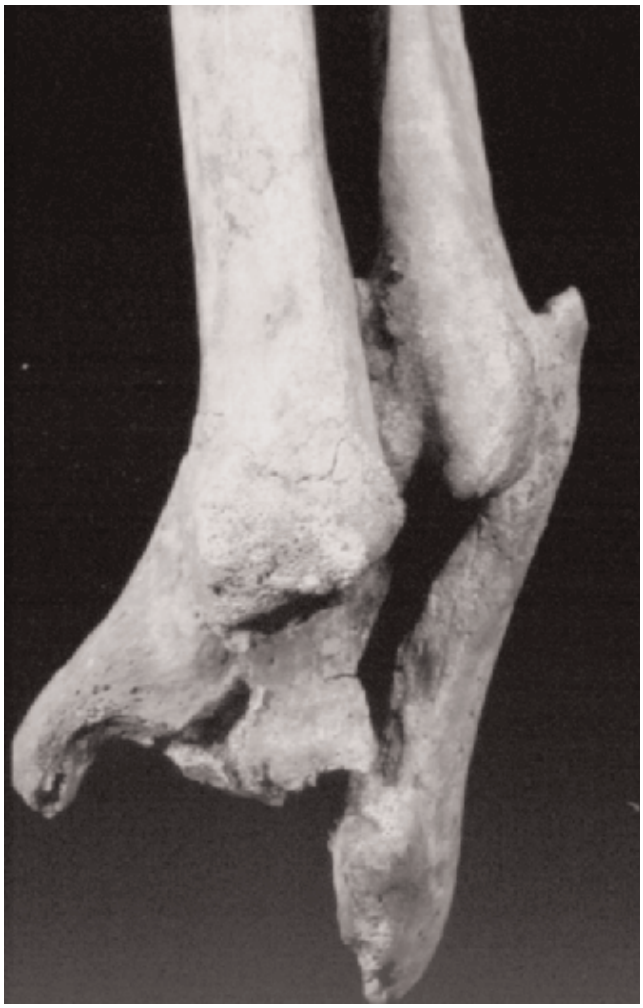

Fig. 75: Spiral fracture of the ankle in an adult of unknown sex (sk. 81). The fracture line extents into the ankle joint and there is considerable angulation. Photo: Juliet Rogers

Table 49: Percentage of shortening in some fractured bones at Barton

	Length of intact bone (mm)	*Length of fractured bone (mm)*	*% shortening**
Clavicle	143	125	12.6
Tibia	374	349	6.7
Femur	450	422	6.2
Radius	215	202	6.0
Clavicle	153	144	5.9
Radius	192	181	5.7
Ulna	245	236	3.7
Ulna	290	282	2.8
Radius	236	232	1.7
Ulna	250	246	1.6
Humerus	327	323	1.2
Tibia	396	393	0.8
Ulna	274	273	0.4
Tibia	363	362	0.3
Radius	249	249	0

*% shortening = (Normal length – Fractured length)/Normal length ×100

Despite the alteration to the normal architecture of the ankle joint, there was no osteoarthritis, probably because the individual had not survived sufficiently long after the injury. There was, in fact, only a single fracture which had developed OA, a Colles fracture with arthritic change in the wrist; this had occurred in an adult female (sk. 1420, F4565).

A number of the fractures had healed with some shortening, some of the metacarpals, for example. Where possible, the lengths of the fractured bone and the normal contralateral bone were compared; unfortunately this comparison could be made in only a small number of cases, either because the bones were too broken to measure or the normal bone was not present. The comparisons that could be made are listed in Table 49. The greatest degree of shortening was found with a clavicle; this is by no means surprising since fractures of the clavicle almost always result in shortening as there seems to have been no method of reducing a clavicular fracture known or used until comparatively recently. Fractures of the ulna or radius tend to show no shortening because, as explained above, the intact bone in the forearm acts to splint the broken one. In general, the data in the table support the notion that the fractures were often well treated with excellent results.

Pathological fractures

Very few fractures occurred in pathological bone. The vertebral fracture that was due to osteoporosis has already been mentioned. One other fracture typical of osteoporosis was found and that was a fracture of the femoral neck, in a male of unknown age (sk. 1479, F3738). The other pathological fractures were found in individuals with Paget's disease; a fracture of the radius in a male of at least 45 years of age (sk. 446, F1290) and one involved the proximal ulna, this time in a female of unknown age (sk. 1331, F4516).

Head Wounds

The two juveniles who had skull fractures were probably the victims of violence, even though there was no other evidence of ill treatment elsewhere in their skeletons, and even if they were deliberate injuries they did not lead to death. Eight adults, mostly from the earlier phases, also had injuries to the skull that were deliberately inflicted, the majority with edged weapons of some kind (Table 50).

Fig. 76: Head wound in an adult male aged 45+ at the time of death (sk. 179). The wound is slightly to the left of the midline in the frontal bone, and involved the internal table. It is well healed and remodelled indicating a long period of survival after the injury. Photo: English Heritage

Table 50: Cases of head wounds at Barton

Skeleton number (context)	*Phase*	*Sex*	*Age*	*Nature of head wound*
1775 (F3924)	B/C	M	45+	Depression 10 mm anterior to bregma with smooth margins
2196 (F7139)	B/C	M	45+	Healed cut *c.* 20 mm in length, above right orbit; begins 30 mm above centre of superior orbital rim and slopes upwards. Fracture of left femur; left Colles fracture
2359 (F7231)	B/C	M	25–35	Healed cut in left frontal *c.* 41 mm in length. Starts half way along coronal suture and continues medially
179 (F305)	C	M	45+	Healed cut extending from mid-frontal to bregma in sagittal plane just left of mid-line; 30 mm in length. Raised edge on left margin. Depressed fragment of internal table. Healing rib fracture
2352 (F7220)	C/D	F	25–35	Smooth healed depression on parietal, 10 mm lateral to sagittal suture and about two-thirds along from front of suture
2723 (F7568)	C/D	M	25–35	Healed cut parallel to superior margin of left orbit and *c.* 3 mm above it
362 (F1250)	D	M	25–35	Healed cut on left parietal running from junction of frontal and sphenoid to sagittal suture. Mid-way along, deeper area with rolled edges and with endocranial damage and displacement of fragment of internal table
800 (F3096)	D	M	45+	Healed cut 10 mm in length in left frontal at right-angles to coronal suture and 30 mm to left of bregma. Multiple fractures

Fig. 77: Young male (25–34) with a head wound in the left parietal (sk. 362). The injury was almost certainly made with a heavy weapon which displaced a flake of the internal table inwards. The wound is well healed and was probably sustained a long time before death. Photo: English Heritage

Of the seven men, six had healed cuts on the skull, mostly on the left side of the head suggesting that the attack on them had been perpetrated by a right-handed opponent. In two cases (sk. 179, F305 and sk. 362, F1250) the attack had been made with a heavy weapon which had fractured the internal table of the skull and displaced a fragment inwards (Figs. 76 and 77). The seventh man (sk. 1775, F3924) had a depressed healed fracture which most probably was the result of a blow on the head, and the only woman with a head wound

Fig. 78: Tibial injury in a child c. *ten years old at death (sk. 2551). The longitudinal cut (arrowed) is not completely healed and there is a periosteal reaction around it, suggesting that it was sustained not long before the child died. Photo: English Heritage*

(sk. 2352, F7220) also had a depressed fracture which seemed to be the result of a blow to the head.

It is interesting that all the injuries were well healed, indicating that these individuals had lived for some years afterwards and that none of the attacks had been responsible, directly or indirectly, for their deaths.

Post-cranial Injury

There was a single child of about ten years of age with a post-cranial injury (sk. 2551, F5439). There was an unhealed cut on the subcutaneous surface of the tibia about two-thirds of the way down the shaft (Fig. 78). The cut was partially healed with a periosteal reaction around it and with slight swelling of the shaft. The fact that the cut was not fully healed seemed to point to the fact that the injury had been sustained shortly before death, but there was no other injury in the skeleton and no indication of the cause of death. An X-ray of the injury provided no further information and it is impossible to be certain what caused it.

Table 51: Cases of dislocations at Barton

Skeleton number (context)	*Phase*	*Sex*	*Age*	*Site of dislocation*
1442 (F4583)	A/B	Female	15–24	Right shoulder with Erb's palsy
400 (F3402)	C/D	Juvenile	*c.* 13	Congenital dislocation of right shoulder
378 (F1558)	D	?Male	*c.* 17	Fracture dislocation of left elbow
1815 (F7985)	D/E	Male	Adult	Fusion of dip, with hyper-extension of terminal phalanx
1910 (F5032)	E	Male	45+	First toe with hyper-extension of proximal phalanx
2583 (F7395)	E	Male	45+	Right shoulder with osteoarthritis of glenoid

Fig. 79: Right glenoid of a young (15–24) female (sk. 1442). The glenoid appeared 'squashed' with a depressed area (arrowed) and most likely resulted from a shoulder dislocation at birth which would have given rise to an ulnar nerve palsy. Photo: English Heritage

Dislocations

Joints are said to be dislocated when there is a complete loss of contact between the two bone surfaces; where the loss of contact is only partial, the joint is said to be subluxed. Unstable joints such as the shoulder, or those subject to frequent injury, such as the finger joints, are commonly dislocated but can often be reduced and there may be no evidence that the dislocation ever took place (Browner *et al.* 1998).

There were six skeletons in which dislocation could be seen (Table 51). In a child who was about 13 at the time of death (sk. 400, F3402), the right glenoid was posteriorly displaced and this was most probably associated with a congenital displacement of the shoulder. A male aged 45 or more when he died (sk. 2583, F7395) had dislocated his right shoulder, probably on more than one occasion since he had developed osteoarthritis of the glenoid. Another skeleton with a dislocation of the shoulder was that of a young female (sk. 1442, F4583) who had a deformity of the right glenoid which appeared 'squashed', although the head of the humerus was normal (Fig. 79). There was a depressed area about 5 mm wide running horizontally across the area between the proximal two-thirds and the distal one-third of the glenoid. The radiographic appearance was characteristic of a shoulder dislocation taking place at birth and causing damage to the brachial plexus, resulting in an Erb's palsy.[5]

Another skeleton (sk. 378, F1558) had a fracture dislocation of the left radio-humeral joint. There was a uniform wedge fracture of the left ulna with posterior angulation and posterior dislocation of the head of the radius. A new articulation for the radius had been formed on the back of the humerus but, unfortunately,

Fig. 80: First metatarsal and proximal phalanx from an elderly male (sk. 1910). The superior surface of the metatarsal head had an impression that fitted the base of the phalanx, suggesting that the big toe had been dislocated and not been reduced. Photo: English Heritage

Fig. 81: Fused left elbow of an adult male (sk. 329). This most likely resulted from an injury with bleeding into the joint. The elbow was broken post mortem *and the new fracture line can clearly be seen. Photo: English Heritage*

the radius was missing and so no further details of changes which may have supervened could be obtained.

In one of the two remaining cases (sk. 1815, F7985), one of the finger joints had been dislocated, one distal inter-phalangeal joint was fused and the distal phalanx was hyper-extended showing that the dislocation had not been reduced. Nor was the dislocation displaced in the fourth case (sk. 1910, F5032), another elderly male who had dislocated his left first metatarso-phalangeal joints. The dorsal surface of the metatarsal head had an impression which corresponded to the base of the proximal phalanx, showing that it was hyper-extended although it had not fused to the metatarsal head, perhaps because the dislocation was relatively recent (Fig. 80).

Soft Tissue Injuries

Soft tissue injuries occur much more frequently than fractures or dislocations, but cannot be detected in the skeleton unless there are secondary signs from which the injury can be inferred. Such signs include fusion of joints in the absence of evidence of a fracture, or by the presence of what is frequently referred to myositis ossificans.

Evidence for soft tissue trauma was found in twelve skeletons and a brief description of these is given in Table 52. The cases were more or less evenly distributed between the sexes and almost all phases were represented; it must be remembered that they are only a fraction of those that will have occurred at the site and there is little point in calculating prevalences.

Two skeletons (sk. 329, F3370 and sk. 529, F3482) were found with elbows fused at least to a right-angle in the absence of any radiological evidence of a fracture. The most likely cause of this in these cases was an injury to the elbow with bleeding into the joint and subsequent bony ankylosis (Fig. 81). This was probably also the sequence of events in the three individuals with fused finger joints. The fingers are very often damaged, and with a penetrating injury there may be bleeding into the joint, again followed by ankylosis after the blood clot organizes. A couple of lesions on the tibia were considered to be soft tissue injuries or ossified haematomas on X-ray, while the only juvenile among the group (sk. 2694, F7681) seems to have damaged the outer border of the left hand.

The other evidence for trauma was in the shape of new bone, either in discrete plaques or as new bone attached to the femur or the ischium. This kind of new bone is almost always referred to as myositis ossificans in the palaeopathological literature. This is unfortunate as it leads to confusion with the inherited condition in which muscles or muscle groups may be entirely replaced by new bone,[6] and it is better to refer to it as myositis ossificans traumatica (MOT), or heterotopic ossification.

There were four skeletons with MOT, two having particularly extensive examples. With the first skeleton (sk. 1402, F4559) two large plaques of bone were found, one measuring 88 × 54 mm, and the second 47 × 30 mm, both approximately triangular in shape. It is possible that the smaller piece fitted onto the larger but this was by no means certain. A plaque measuring 85 × 75 mm, again approximating to a triangle in shape, was found with the second case (sk. 2210, F5282). The plaque was slightly concave and on this account it was first considered that it might be a pleural plaque,

Table 52: Cases of soft tissue trauma at Barton

Skeleton number (context)	*Phase*	*Sex*	*Age*	*Nature of injury*
1402 (F4559)	A/B	F	Adult	Large plaques of bone; myositis ossificans traumatica
2102 (F7442)	B	Unknown	Adult	Swelling in distal 2/3 of tibia; radiography suggest old soft tissue injury
329 (F3370)	B/C	Male	Adult	Fusion of left elbow at about 110°; no evidence of fracture
2694 (F7681)	B/C	Juvenile	*c.* 14	Left fifth metacarpal swollen and misshapen with an inferior bone spur
529 (F3482)	C/D	Male	15–25	Right elbow flexed at 90° and semi-pronated; no evidence of fracture
759 (F1438)	D	Male	Adult	Fused proximal and middle phalanx of hand
850 (F3105)	D	Female	45+	Fused proximal and middle phalanx of hand
2210 (F5282)	D	Male	35–45	Myositis ossificans traumatica
1134 (F3191)	D/E	Male	45+	Fused proximal and middle phalanx of hand
2503 (F7343)	D/E	Female	45+	Lesion on right tibial shaft, *c.* 31 × 45 mm; probably ossified haematoma
2539 (F7380)	D/E	Male	Adult	Myositis ossificans traumatica
1304 (F3264)	E	Male	Adult	Myositis ossificans traumatica

Fig. 82: Large plaque of heterotopic ossification; originally one piece but subsequently broken and most likely resulting from a bleed into a large muscle, perhaps the quadriceps femoris (sk. 2210). Photo: English Heritage

such as may be found in asbestosis; on X-ray, however, it seemed much more likely that it was heterotopic ossification (Fig. 82). Given the large size of all three plaques, they must have arisen in large muscles such as those of the thigh, following some extensive soft tissue damage and bleeding. There is no means of knowing precisely where they came from, however, or what injury led to their formation.

In the third case (sk. 2539, F7380), there was a large spur of bone overlying the right ischium, originating from above the acetabulum at the site of the origin of the reflected head of the rectus femoris muscle and damage to this muscle was most likely the cause. This skeleton also had a plaque of new bone (80 × 10 mm) on the subcutaneous border of the right tibia, almost certainly an ossified haematoma.

The final instance of MOT (sk. 1304, F3264) took the form of a wide, smooth plaque of bone arising from the lateral margin of the left femur. It had a sharp lateral edge and measured 20 × 10 mm and was probably the result of a bleed into the vastus intermedius muscle.

Osteochondritis Dissecans

Osteochondritis dissecans is characterized by fragmentation and possible separation of part of the articular surface. Radiologically, a well-demarcated area of increased radiolucency is noted surrounding the articular fragment, which in some cases may become completely detached. It is thought that the most likely origin of the lesion is some kind of trauma that results in a transchondral fracture but the exact aetiology is unclear (Yado *et al.* 2004). The lesions occur on the convex joint surfaces, the knee being the one most commonly affected, followed by the elbow, talus, hip and other joints (Williams *et al.* 1998). Those with the condition may be completely unaware of it, although in some cases the affected joint may be painful and swollen and limited in movement.

Twenty-eight individuals were noted with the lesion, 13 males, 11 females and 4 adults of unknown sex. The knee was affected in fifteen cases, the elbow in nine and there were three single cases affecting the talus, temporo-mandibular joint and femoral head. The lesion in the femoral head was located on the superior pole and measured *c.* 30 × 20 mm. It had occurred in an adult male (sk. 528, F3481) in the context of Perthes' disease,[7] which is a form of avascular necrosis of the femoral head.[8] This often leads to flattening and collapse of the femoral head and is frequently followed by secondary osteoarthritis in adulthood. The radiographic appearances of the lesion in the head of the femur strongly suggested that it was an area of osteonecrosis secondary to Perthes' disease.

Table 53: Crude prevalence of osteochondritis dissecans (% and 95% CI)at Barton, by sex and period

Site of lesion	*Male*		*Female*	
	Early period	*Late period*	*Early period*	*Late period*
Knee	1.1 0.4–3.3	0.7 0.1–3.8	1.3 0.4–3.7	2.2 0.8–6.3
Elbow	0.7 0.2–2.7	1.6 0.4–5.5	0.4 0.1–2.3	1.6 0.4–5.7

In the contemporary population, the medial condyle of the knee is most often affected and this was also the case for the Barton assemblage. Of the fifteen cases, eight involved the medial condyle, four the patello-femoral joint and the remaining three, the lateral condyle.

The prevalence of osteochondritis dissecans of the knee and of the elbow is shown, by sex and by period, in Table 53. It can be seen from this table that the frequency of the condition is similar in both males and females, and in the early and late periods such differences as Table 53 demonstrates are not significant.

Slipped-Capital Femoral Epiphysis

This refers to shifting or tilting of the growth plate of the femoral head, with displacement of the femoral head on the femoral neck. It occurs commonly between the ages of 8 and 17 years, and boys are more often affected than girls. Trauma is thought to play a part in its aetiology but it has also been found that those with the condition are more obese than those without (Poussa *et al.* 2003). The sequelae of the condition may include pain, alterations in gait and osteonecrosis (Song *et al.* 2004; Kennedy *et al.* 2001); in some cases osteoarthritis develops in adulthood. Three individuals were identified at Barton with a slipped femoral epiphysis (Table 54). In each case the left hip was affected, but in one (sk. 1156, F3539) the condition was probably bilateral although the right hip was poorly preserved and it was not possible to be certain on this point. Only one of the individuals (sk. 2693, F7680) was a child at the time of time of death; both the others had survived into young adulthood and there was no evidence on their skeletons of the cause of their death.

Spondylolysis

Spondylolysis is the condition in which the vertebral arch is separated from the body as the result of a fracture, most frequently through the isthmus, the so-called pars inter-articularis fracture. It is generally assumed that spondylolysis is a stress fracture that is associated with learning to walk, although there may be contributory environmental or inherited factors as some families have a high prevalence of the condition.[9] Spondylolysis does not occur in children before they start to walk, nor is it found in adult patients who have never walked (Rosenberg *et al.* 1981). The prevalence is much higher in those who take part in sports that involve repetitive hyper-extension of the spine such as gymnastics, diving and wrestling (Bone 2004).

The fracture is usually bilateral and most commonly affects the fifth lumbar vertebra and is more common in males than females. Nowadays, it is often discovered as an incidental finding when the lumbar spine is X-rayed and most cases are asymptomatic (Logroscino *et al.* 2001). If the affected vertebral body slips forward on the one below, when the condition is then referred to as spondylolisthesis, it may give rise to back pain.

The condition is extremely easy to diagnose in the skeleton although there is no certain way in which to identify whether spondylolisthesis has occurred; cases have been noted, however, where the affected vertebrae have become fused when the spine also has DISH (Manchester 1982).

Forty-seven skeletons at Barton were found with spondylolysis, 29 males, 17 females and one adult of indeterminate sex. The crude prevalence is 3.8% (95% CI 2.9–5.0%) which corresponds well to the normal range of 4–6% found in the modern population (McTimoney and Micheli 2003). The prevalence was higher in males than in females, although not significantly so, and there was no change in the frequency of the condition over time (Table 55).

Table 54: Cases of slipped-capital femoral epiphysis at Barton

Skeleton number (context)	*Phase*	*Sex*	*Age*	*Side affected*
2693 (F7680)	A/B	Juvenile	12–13	Left
1156 (F3539)	C	Female	25–35	Left and possibly right also
2340 (F7234)	D/E	Male	15–25	Left

Table 55: Crude prevalence of spondylolysis (% and 95% CI) at Barton, by sex and period

Period	*Male*	*Female*
Early	5.4	3.5
	3.5–8.4	1.9–6.3
Late	4.1	4.1
	1.9–8.7	1.9–8.7
Total	5.2	3.4
	3.6–7.3	2.2–5.4

Table 56: Lumbar vertebrae affected by spondylolysis at Barton, by sex

Level of lesion (n)	*Male*	*Female*
L3		1
L4	5	3
L5	23	15
L6	2	1

Fig. 83: Spondylolysis of fourth and fifth lumbar vertebrae in an elderly female (sk. 2775). The detached laminae were not recovered. Photo: English Heritage

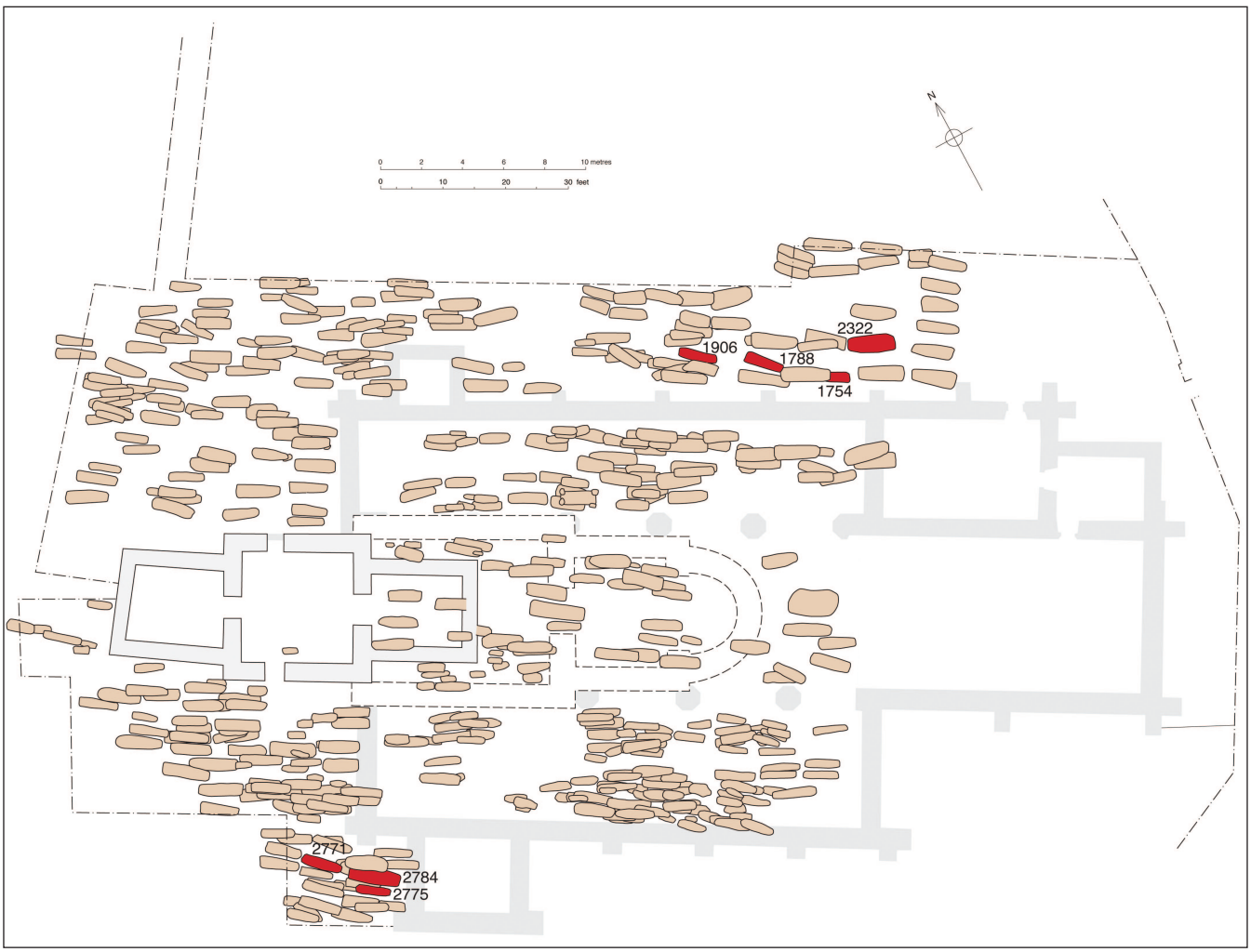

Fig. 84: Clusters of cases of spondylolysis in Phase E burials. Drawing: Simon Hayfield

The condition was bilateral in all but three cases, one female (sk. 2775, F7614) and two male (sk. 436, F3069 and sk. 671, F3092). As expected, the fifth lumbar vertebra was most commonly involved (Table 56) and in all but four instances, only a single vertebra was affected. Four skeletons presented with spondylolysis of two adjacent vertebrae, three females (sk. 2222, F7496; sk. 2692, F7543 and sk. 2775, F7614) and one male (sk. 2338, F4911). The fourth and fifth vertebrae were affected in three of the cases; the third and fourth in the remaining case (Fig. 83).

Given the high prevalence found in some families (Haukipuro *et al.* 1978), the cases of spondylolysis were plotted on the burial plans to see if any clustering occurred, the hypothesis being that individuals who were buried close together were likely to have been related. The only clusters found were restricted to Phase E (Fig. 84). Four cases were buried close to each other in the north-east corner of the churchyard and a cluster of three more cases was found in the south-west corner. Although one cannot rule out the possibility that these clusters occurred by chance, the alternative possibility, that they represent true family groupings, should not be entirely discounted.

Schmorl's Nodes

Schmorl's nodes are formed when part of the nucleus pulposus of the inter-vertebral disc herniates through the surrounding annulus fibrosus and produces an impression on the end plate of the adjacent vertebra. After death, the disc decomposes and the nodes are recognized by the negative impressions on the end plate which may vary both in size and shape. They are easy to diagnose and very common in the skeleton, occurring most frequently in the lower thoracic and lumbar regions. They are thought to be related to repetitive trauma to the spine, usually over a long period, although they may occur acutely (Fahey *et al.* 1998), and there is also a suggestion that they are the end result of avascular necrosis of the end plate and that the herniation is secondary (Peng *et al.* 2003); this is, however, still a minority view. The presence of Schmorl's nodes may be associated with pain (Hamanishi *et al.* 1994), but is usually asymptomatic.

Table 57: Crude prevalence of Schmorl's nodes (% and 95% CI) at Barton, by sex and period

Period	*Male*	*Female*
Early	19.6	9.6
	15.6–24.3	6.7–13.6
Late	19.6	3.7
	13.8–27.0	1.6-8.3
Total	24.3	8.9
	17.1–24.0	6.7–11.8

There were 160 skeletons with Schmorl's nodes among the Barton assemblage, 106 males, 43 females, 7 other adults and 4 juveniles; only 3 of the juveniles could be aged and all were between 12 and 13 when they died. As expected, the great majority of the nodes were in the lower thoracic and lumbar regions. The sex-specific prevalence for all phases and for the early and late periods is shown in Table 57. There is a significant difference in the prevalence between the sexes, the condition being much more common in males than in females, as expected. The male prevalence has not altered over time and, although the female prevalence has more than halved in the late period, the difference is not significant.

10. PALAEOPATHOLOGY V: METABOLIC DISEASES

There are a number of diseases that interfere with the normal metabolism of the skeleton; these include, for example, Paget's disease, rickets (vitamin D deficiency) and scurvy (vitamin C deficiency), but this chapter starts by considering osteoporosis, a condition in which the normal loss of bone with age is exaggerated.

Osteoporosis

Up until early adulthood the skeleton is in positive balance and sometime between about 30 to 40 years of age the maximum bone mass (MBM) is achieved. The absolute value of the MBM varies from one individual to another and is dependent on a number of factors, including sex, activity, diet and race. After MBM is achieved, the skeleton enters into negative balance and bone is lost with increasing age. Women lose bone at a faster rate than men at all ages but their rate of loss tends to increase further after the menopause, when the protective effect of oestrogen is lost. If the bone loss becomes too great – and especially if much trabecular bone is lost – there is a great risk of fractures occurring, particularly in bones in which the amount of trabecular bone is greatest, the vertebrae, femoral neck and distal radius. This state is referred to as osteoporosis, which can be defined as a systemic skeletal disorder characterized by low bone mass, and an increase in bone fragility and susceptibility to fracture. It is usually defined in terms of the bone mineral density (BMD) of the skeleton, measured most often in the lumbar vertebrae or the femoral neck (for further details see Favus 2003; Kanis 1994).

When the BMD is measured, individual readings are converted either to t or z scores. The t-score is the number of standard deviations that the reading is from the mean of young (25–30 year old) women; the z-score is the number of standard deviations from the mean of age-matched healthy women.[1] Osteoporosis is defined as a t-score that is greater than −2.5 and established osteoporosis is said to be present when the individual also has an osteoporotic fracture.[2]

Osteoporosis is currently a major public health problem and accounts for a considerable proportion of the health budget in countries with ageing populations. It is sometimes referred to as a modern plague, and it is of considerable interest to know how frequent it was in the past and whether it is actually increasing in incidence and prevalence, other than as a consequence of an increasingly elderly population. To date, however, there is no agreed way in which the condition can be diagnosed in the skeleton. Studies on the cortical thickness of the metacarpals (Mays 2000), on estimating BMD (Lees *et al.* 1993), and on directly measuring bone density (Brickley and Waldron 1998), have all been carried out on skeletal assemblages and show some changes with age in the expected direction, and radiographs may show thinning of the cortex of the limb bones or loss of traculae in the vertebrae, but no attempt has been made with any method to construct an operational definition of osteoporosis that can be used in palaeopathology. Instead, the diagnosis tends to rely on the subjective opinion of the bone specialist; in this opinion he or she may be guided by the apparent (or actual) weight of the bone, on the cortical thickness of broken limb bones, or the trabecular pattern of broken vertebrae, and the opinion may sometimes be supported by radiography.

In the present study, osteoporosis was diagnosed by the subjective feel or appearance of the bones, although in a single instance (sk. 458, F3427) there was a crush fracture of a thoracic vertebra which provided the only case of established osteoporosis.

There were 26 skeletons which were considered to have osteoporosis and, as expected, females predominated; there were 16 females, 7 males, and the remaining 3 cases were in adult skeletons that could not be assigned a sex. All the individuals who could be assigned an age were at least 45 years old at the time of death except for one female who was apparently aged between 25 and 34. It should be noted, however, that 18 of the skeletons could not be given an age, almost entirely because they were poorly preserved, perhaps as a consequence of the osteoporosis.

The prevalence of osteoporosis is shown in Table 58. There seems to have been a considerable increase in prevalence in females from the early to the late period, although it is not statistically significant. The female prevalence is more than three or four times higher than the male in both periods but, again, this difference is not significant. Indeed the prevalence in the females, especially in the later period, seems unrealistically high; if one restricts the diagnosis to the single

Table 58: Crude prevalence of osteoporosis (% and 95% CI) at Barton, by sex and period

Period	*Male*	*Female*
Early	3.2 1.1–9.1	9.0* 4.6–16.8
Late	5.6 1.9–15.1	20.5 11.2–34.5
Total	3.4 1.6–7.2	10.7 6.8–16.5

* Includes a single case with an osteoporotic vertebral fracture

female skeleton showing the presence of typical osteoporotic fractures, which is a much more desirable way to proceed, then the prevalence falls to 2.3% (95% CI 0.4–11.8%) which is probably closer to the 'true' prevalence than the figures shown in Table 58.

Bone mineral density

As part of this study, the BMD was measured in a total of 149 femora, 85 from males and 64 from females. Unfortunately, there is no surviving record of the criteria used to select these particular bones but they have come from all phases and a wide range of ages. The results for males and females are shown in Figs. 85 and 86, respectively. The expected decrease in BMD with age was found, although the apparent rate of loss is actually greater in the males than the females; the rate of loss in the males is 0.01 g/cm² per year and in the females, 0.006 g/cm² per year. These rates are at best very approximate and may well not reflect the true state of affairs in the living population. For all the individuals aged 45 or more at the time of death, t-scores were calculated using the BMD values of the young males or females as the reference population. None of

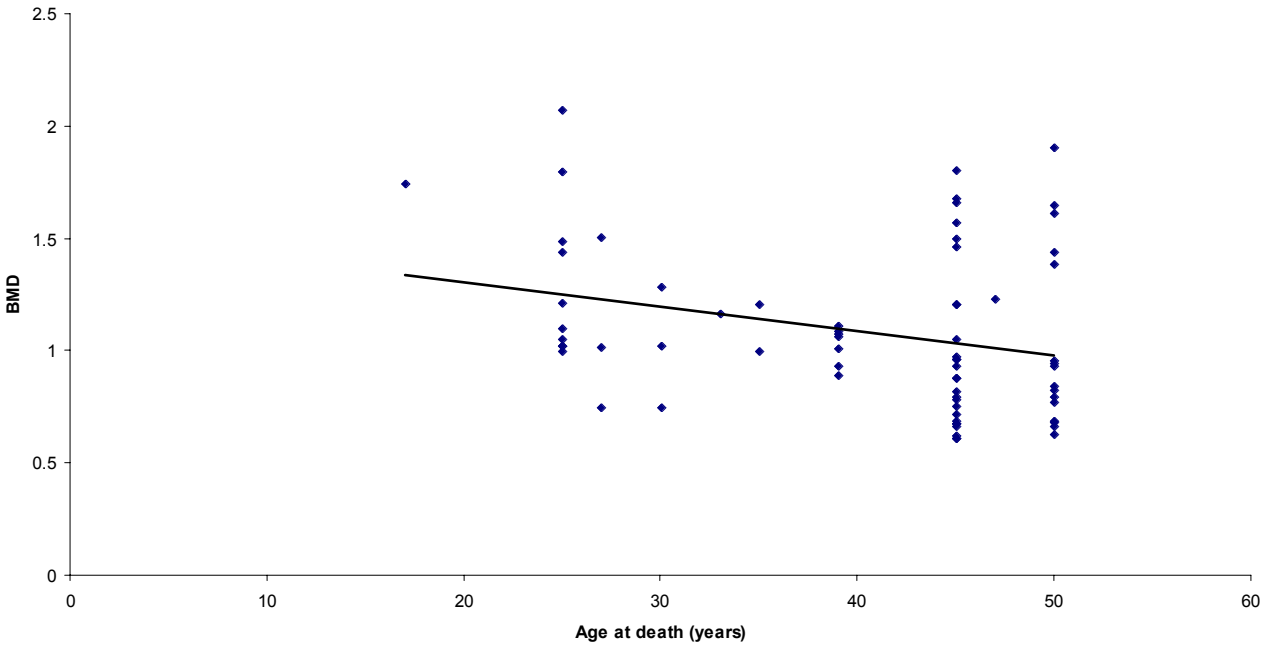

Fig. 85: Bone mineral density (g/cm²) as a function of age in males from Barton

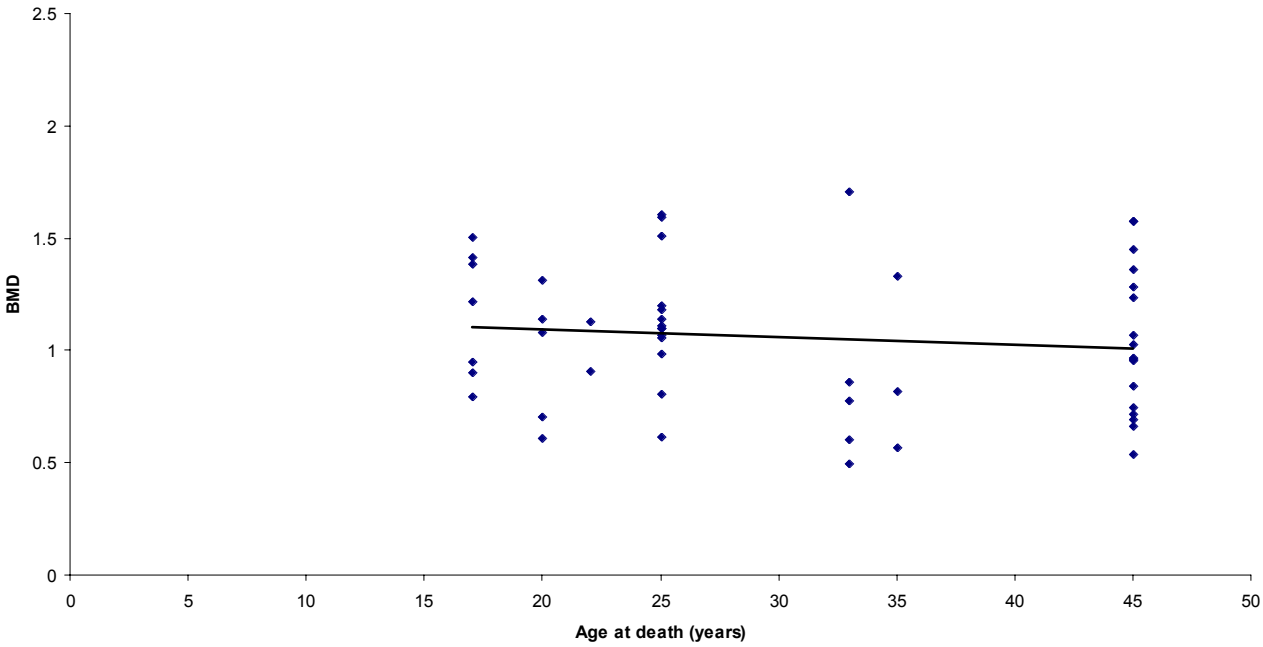

Fig. 86: Bone mineral density (g/cm²) as a function of age in females from Barton

the t-scores was less than −2.5 and so none of the individuals was osteoporotic on this definition. When the scores were aggregated into normal (> −1.0) or osteopaenic (−1.0 to −2.5), 61.5% of the male scores were normal and 38.5% osteopaenic compared with 74.1% and 14.9%, respectively, of the females. It is scarcely credible that a greater proportion of males than females in this population would have a lower BMD and this strongly suggests that measuring BMD in the skeleton is not an accurate reflection of the value during life. These data also indicate how urgently an acceptable and valid way to diagnose osteoporosis in the skeleton is needed.

Rickets

Rickets is caused by a lack of vitamin D and results in defective mineralization of growing bone. Under normal circumstances vitamin D is obtained predominantly by the action of ultra-violet light on precursors in the skin. The compound formed is inactive and is converted to the active form first in the liver and then in the kidney.[3] The active form enhances the uptake of calcium from the gut and may stimulate the production of osteoclasts from stem cells. There are very few sources of vitamin D in the diet, the most important being oily fish and fish oils. During the winter season in northern Europe it is likely that rickets would have been a common occurrence in the past; it was endemic in some of the cities of Great Britain during the latter part of the nineteenth century, when smoke from coal-burning fires darkened the skies and blotted out what little winter sunlight there was. Rickets would also have been likely to develop in children who were breast-fed for long periods (Wharton and Bishop 2003).

The signs of rickets are evident at the growing ends of the long bones, which are splayed and may appear frayed, especially on X-ray. The bones of the skull may become soft and the frontal bone bossed, changes which are referred to as cranio-tabes. If the affected child is walking, the femur and tibia become bowed under the weight of the body; the femur usually bows from front to back and the tibia from side to side (Feldman *et al*. 1990). The pelvis may also become distorted by the femoral heads pushing upwards into the softened pelvic bones. If deformity of the pelvis occurs in girls it may present difficulties with child-birth in later life.[4] When affected children are able to get out into the sun again, healing takes place relatively quickly but the distortions in the legs or the pelvis do not correct themselves. Healed rickets can be detected in adult skeletons by the typical changes in the long bones and, in addition, the femur is usually buttressed by a longitudinal bar of bone on the posterior surface.

There were only ten skeletons at Barton with evidence of active or healed rickets, seven in adults and three in children aged 1, 3 and 5 years, respectively, at the time of death. Rickets is not a killing disease and so these three children must have died from some other inter-current disease of which there was no sign on their skeletons, as is so often the case in children.

The number of cases of rickets noted at Barton can in no way be taken as an accurate indication of the prevalence of the disease in the living population because, as noted above, once the child is exposed to the sun again, the condition is rapidly reversible. Only if there is considerable malformation of the leg bones will the stigmata of the condition persist into adulthood. There is little point in calculating the prevalence of this disease as the results would serve more to confuse than inform.

Osteomalacia

Rickets is a childhood disease; adults who become vitamin D deficient develop a condition known as osteomalacia. This is also characterized by defective mineralization of bone: the bones may become soft and bend and radiologically there may be localized areas of demineralization known as Looser's zones which are pathognomonic of the condition (Steinback and Noetzl 1964). It is scarcely possible to recognize osteomalacia in the skeleton if the bones are not X-rayed and it is likely that most cases go undiagnosed. A single case was diagnosed radiologically at Barton, an adult male (sk. 898, F7869) from the intermediate Phase B/C.

Paget's Disease

Sir James Paget (1877) described a chronic inflammation of the bone which he called osteitis deformans on account of the swelling and deformation in the affected parts of the skeleton. Paget's disease, as the condition is now known, is the second most common bone disorder in elderly people, only osteoporosis occurring more often (Ankrom and Shapiro 1998). The disease presents as a localized disruption of normal bone remodelling with an increase in osteoclast mediated resorption and a compensatory increase in new bone formation.[5] This results in a disorganized structure of woven and lamellar bone, often with enlargement of the affected bone. The nature of the architecture of Pagetic bone predisposes it to fragility and the bones may bend if under gravitational stress, or fracture. The blood supply to the affected bones is increased because of the increased rate of metabolism and in the living a Pagetic bone may feel warm to the touch. If the disease is widespread, the patient may actually suffer from heart failure. The main clinical features of the disease are bone pain, brittleness and deformity; there may be associated neurological signs or symptoms if the affected bones cause pressure on nerves, and in a small proportion of cases, osteosarcoma may develop (Kaplan and Singer 1995). The diagnosis of the disease can be confirmed radiologically by features such as cortical thickening, irregularity of the trabecular outline and alternation of sclerotic and resorptive areas (Whitehouse 2002).

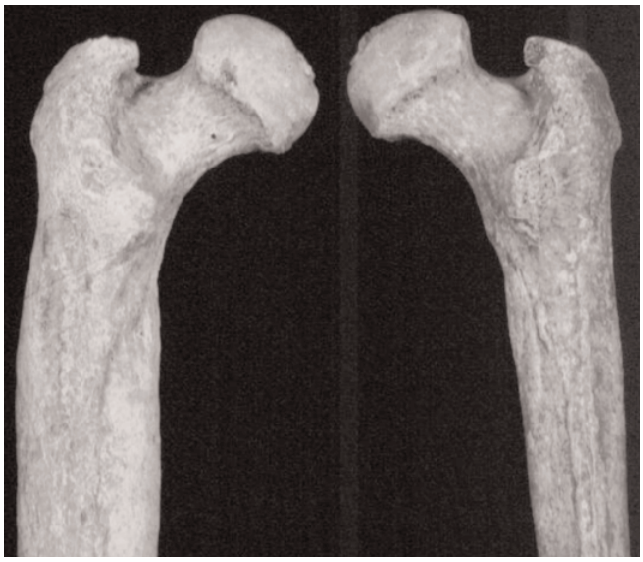

Fig. 87: Paget's disease of the proximal left femur in an elderly male (sk. 1794). Note the greatly widened shaft compared with the normal femur on the right. The diagnosis was confirmed radiologically. Photo: Juliet Rogers

The aetiology of the disease is unclear although it has been suggested that it is the late result of a viral infection, both measles and distemper viruses having been implicated (Khan *et al.* 1996), but this hypothesis is very controversial (Helfrich *et al.* 2000); an association with animals has also been demonstrated and this has been taken to indicate that animals may carry some infectious agent that is aetiologically important (Lopez-Abente *et al.* 1997). Paget's disease is known to run in families and recent research suggests that there may be a genetic fault which causes increased osteoclast activity.[6]

The prevalence of Paget's disease shows remarkable geographical variation. In the United Kingdom the prevalence in those over 40 years of age is 3.5%; in France it is 1.8% and it is even lower in some other European countries, notably in Sweden where the prevalence is only 0.4% (Detheridge *et al.* 1982). Within the UK the disease is more common in women and the prevalence increases with age and is highest in the north of the country (Barker *et al.* 1977). There is now evidence that the prevalence of the disease is decreasing in the UK (Cooper *et al.* 1999), in New Zealand (Doyle *et al.* 2002) and in the USA (Tiegs *et al.* 1999), which might indicate that some environmental agent has either become less common or that some form of resistance has been developed against it in the general population.

Fifteen cases of Paget's disease were identified at Barton, eleven males, three females and one adult of unknown sex. Just over a third of the cases were suspected on visual examination, either because they were increased in size, thickness or vascularity (Fig. 87). All suspect bones were X-rayed and so were the rest of the affected skeletons. Further cases came to light when other bones with an abnormal appearance were X-rayed. One other case was discovered during the radiological examination of bones with fractures, and one during a study of the prevalence of hyperostosis frontalis interna (see Chapter 11).

Nine of the cases came from the early period of the cemetery and the remaining six from the late period. All the skeletons with Paget's disease had at least 40% of the skeleton present, and all that could be assigned an age were at least 35 at death. There were 611 skeletons at Barton that had 40% of the skeleton present and, using these as the denominator, the crude prevalence of Paget's disease was calculated as 2.5% (95% CI 1.5–4.0%). The prevalence in both the early and the late periods was also 2.5% (95% CI 1.3–4.6% and 1.1–5.7%, respectively) showing that there has been no change in the prevalence over time.[7] These data again indicate the problems associated with trying to calculate the prevalence of disease in skeletal assemblages, when small numbers are involved; the shift of one or two

Table 59: Cases of Paget's disease at Barton

Skeleton number (context)	*Phase*	*Sex*	*Age*	*Affected bones*
1331 (F4516)	A	Female	Adult	Pelvis, ulna (with fracture), distal femur
1354 (F3617)	A	Male	Adult	Skull base, calcaneum
2016 (F7063)	A	Male	45+	Proximal humerus, tibia
1451 (F3712)	A/B	Male	45+	Humerus, femur
49 (F288)	B	Male	35–45	Distal tibia
415 (F3408)	B/C	Female	45+	Humerus, femur, proximal tibia
132 (F344)	C	Male	35–45	Pelvis, lumbar vertebrae, sacrum
446 (F1290)	C	Male	45+	Radius (with fracture)
656 (F7721)	C/D	Male	45+	Pelvis, lumbar vertebrae, sacrum, femur
2158 (F7134)	C/D	Male	45+	Humerus, proximal phalanx
2248 (F7167)	C/D	Male	35-45	Pelvis, lumbar vertebrae, sacrum
2310 (F7215)	C/D	Unknown	Adult	Distal tibia
309 (F1223)	D	Female	45+	Pelvis, thoracic and lumbar vertebrae, sacrum
763 (F7926)	D/E	Male	45+	Proximal humerus, proximal phalanx, pelvis
1794 (F4838)	E	Male	45+	Proximal femur, tibia

skeletons from one phase to another can completely change the apparent prevalence of a disease and nullify earlier conclusions. There is another problem that should also be mentioned here, and that is that three bones with Paget's disease were identified among the disarticulated material, two sacra and one femur. They were sufficiently abnormal on visual inspection that they were selected for radiography, when the diagnosis was confirmed. Very few of the other disarticulated bones were X-rayed and so further examples of the disease may easily have been overlooked. One of the sacra was examined histologically and this confirmed the radiological diagnosis (Aaron *et al.* 1992). The disarticulated bones came from contexts sufficiently distant from each other, and from the discrete inhumations with the disease, that it is unlikely that they were associated.

The distribution of the affected bones does not differ from that in contemporary patients. The axial skeleton was commonly involved, as were the tibia, femur and humerus (Table 59).

About three-quarters of all patients with Paget's disease are asymptomatic and in the rest, pain is the most common complaint. It is impossible to say which, if any, of the individuals with the disease at Barton would have had symptoms. Patients with widespread involvement may appear lethargic and somnolent and it is common for those with the condition to have an increased risk of fracture because the abnormal bone is not as strong as normal. Two of the cases at Barton had fractures; in one (sk. 446, F1290) there was a fracture of the radius through the middle of an area of Pagetic bone; and in the other (sk. 1331, F4516) the fracture of the ulna was also through the pathological bone. There was no evidence of malignancy but five individuals had osteoarthritis of the hip (33%) compared with an overall prevalence of 4% in the population as a whole. An increased risk of OA in patients with Paget's disease has been noted, but by no means as great as the one seen at Barton (van Staa *et al.* 2002).

11. PALAEOPATHOLOGY VI: DISH AND BONE-FORMING

There are a number of conditions in which the production of new bone is a prominent feature. Some have been noted in the chapters dealing with joint diseases; those to be considered here are diffuse idiopathic skeletal hyperostosis (DISH) and hyperostosis frontalis interna (HFI) and, in addition, mention will be made of a general tendency to bone-forming, manifested *inter alia* by ossification of entheses.

DISH

DISH is characterized by the exuberant production of new bone in the anterior longitudinal ligament of the spine, with calcification or ossification of extra-spinal entheses or ligaments and, sometimes, other soft tissues as well. Forestier and Rotés-Quérol (1950) were the first to describe the changes in the spine: they noticed that it was generally confined to those beyond middle age and that the prevalence increased markedly with age. Later, Resnick and his colleagues noted that those with what had come to be known as Forestier's disease had extra-spinal manifestations and they coined the term DISH to take these into account (Resnick *et al.* 1975).

The changes in the spine are the result of ossification into the anterior longitudinal ligament and other spinal ligaments. Although these changes can occur throughout the spine they are usually most prominent in the thoracic region. In time, the ossification leads to ankylosis of variable numbers of vertebrae, but the inter-vertebral disc spaces and the facet joints are normal in the absence of other pathology. One of the features of DISH is that the changes are found only on the right-hand side of the thoracic vertebrae because of the presence of the descending aorta on the left hand side.[1] The ossification of extra-spinal entheses produces spurs and spicules of bone at sites of muscle insertion and extra-spinal ligaments may be ossified, especially those around the pelvis. Entheses that are particularly prone to ossification are those around the calcaneum, the patella, and the insertion of the triceps into the olecranon process.

DISH is more common in men than in women and rarely occurs under the age of 40; the crude prevalence over the age of 40 is approximately 4% in men, and 2.5% in women (Mata *et al.* 1997). It has been found in association with a number of other conditions, most notably obesity and late onset (type II) diabetes (Julkunen *et al.* 1971); there is also an association with abnormalities in vitamin A metabolism (Abiteboul and Arlet 1985) and the most recent thinking is that DISH is the end result of a multisystem hormonal disorder (Denko *et al.* 1994).

One interesting feature of DISH is that the prevalence seems to be substantially increased in those who followed the monastic way of life in the medieval period or who were otherwise apparently of high status. This was first suggested in 1985 when I rather facetiously referred to it as a 'new' occupational disease (Waldron 1985). Since then, however, other studies have confirmed the association and it seems to be well established (Rogers and Waldron 2001).

DISH is very easy to recognize in the skeleton; the right-sided ossification in the thoracic region does not occur in any other condition, but to the inexperienced there may be confusion with the osteophytosis of the spine that may accompany disc disease and some of the sero-negative arthropathies. At Barton, the criteria for diagnosing DISH were that three or more vertebrae were fused on the right-hand side in the thoracic region and the disc spaces and the facet joints were normal in the absence of other pathology. Those skeletons in which only two vertebrae were fused, or where there was right-side ossification but no fusion, were considered to be early DISH.

There were 41 cases of DISH at Barton, 32 males, 7 females and 2 adults of unknown sex (Fig. 88). One of

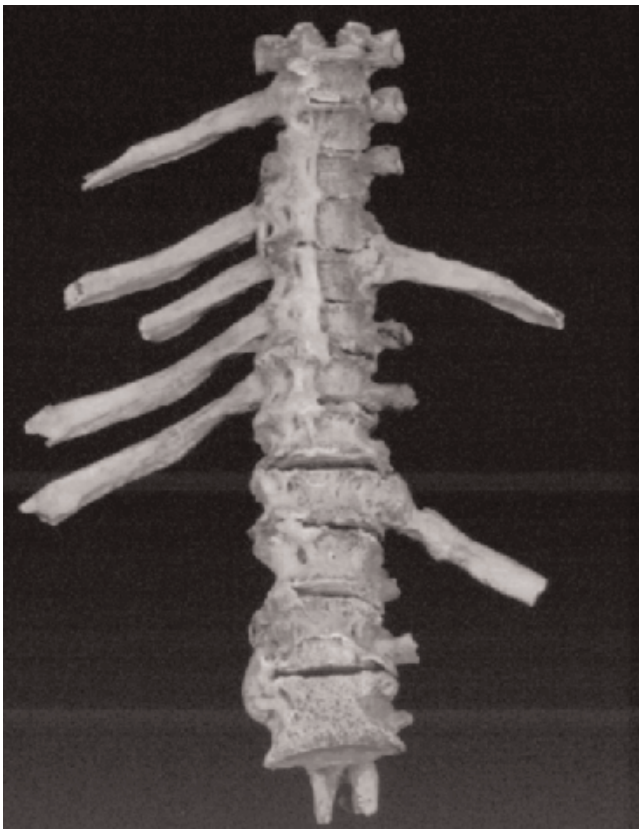

Fig. 88: DISH in a male of unknown age (sk. 772). Ossification into the anterior longitudinal ligament is evident on the right side of all the thoracic vertebrae and the costo-transverse and costo-vertebral ligaments have also been involved resulting in fusion of the ribs to the vertebrae. Photo: Juliet Rogers

Fig. 89: DISH in the spine of Mary Thorley (sk. 740), resulting in the fusion of eleven vertebrae. Photo: Warwick Rodwell

the females with DISH was Mary Thorley (sk. 740, F940), in whom the condition was extensive and florid (Fig. 89). At some time she had fractured several ribs, and the haematoma had ossified and fused three of the fractured ribs together.

The crude prevalence of DISH was 3.7% (95% CI 2.7–5.0%); in the males the prevalence was 7.7% (95% CI 5.5–10.6%) and in the females, 2.9% (95% CI 1.4–5.9%). There was a considerable increase in the prevalence with age for the whole sample, and in the males[2] a marked increase in prevalence in the older individuals from the later, but not the earlier, period

Table 60: Crude prevalence of DISH (% and 95% CI) at Barton, by age and period

Period	*<45 years of age*	*>= 45 years of age*
Early (males only)	3.8 (1.7–8.0)	4.0 (1.7–9.0)
Late (males only)	2.0 (0.4–10.7)	16.4 (8.9–28.3)
Total (both sexes)	1.8 (0.9–3.6)	7.0 (4.6–10.5)

Table 61: Percentage of cases of DISH and early DISH, buried within and without St Peter's church

	Buried inside church	*Buried outside church*
DISH	24.4	75.6
Early DISH	17.4	82.6

(Table 60). This is unlikely to mean that the increased prevalence with age is a recent phenomenon, but rather an artefact of small numbers.

Early DISH: There were only 21 skeletons with what was considered to be early DISH (eDISH), 12 males, 8 females and a single unsexed adult. The crude prevalence was similar to that of DISH, 2.7% (95% CI 1.8–4.7%). There was no difference in the prevalence between the sexes, however, and no increase with age: male prevalence, 2.9% (95% CI 1.7–5.0%); female prevalence, 3.4% (95% CI 1.7–6.5%); prevalence in those <45, 2.3% (95% CI 1.3–4.2), in those >=45, 1.3% (95% CI 0.5–3.4%).

Distribution of DISH and early DISH in the cemetery

The opportunity was taken to plot the distribution of cases of DISH and early DISH within and without the church for each phase separately. At some monastic and ecclesiastical sites, the cases were buried predominantly within the church or chapels attached to them, indicating either that those buried there were monks or of high status. At Barton, there was no clustering of cases of either condition within the church, over three-quarters of all cases being buried in the general cemetery (Table 61).

Bone-forming

Bone-forming is a phenomenon to which Waldron and Rogers (1990) alluded originally some years ago after we had noticed that within any skeletal assemblage there was a proportion of individuals who seemed to have a tendency to ossify soft tissues, especially the entheses, that is, the point at which tendons are

Table 62: Crude prevalence of bone-formers (% and 95% CI) at Barton, by age and period

Period	*<45 years of age*	*>45 years of age*
Early	8.0 (5.4–11.8)	11.4 (7.8–16.6)
Late	3.5 (1.2–9.9)	31.8 21.6–44.0)
Total	6.9 (4.9–9.7)	17.7 (13.8–22.5)

Table 63: Mean bone-former scores for DISH, early DISH and bone-formers at Barton

	Mean score	*Standard deviation*	*n*
DISH	0.80	0.42	25
Early DISH	0.58	0.15	9
Bone-former	0.53	0.20	84

F = 10.57, p = <0.001

attached to bone, probably in response to repeated minimal trauma. The end result is the production of spurs of bone similar to those found in DISH but without the spinal manifestations. Our original characterization of a bone-former was later modified by Rogers and her colleagues and it was used at Barton to determine whether or not an individual skeleton was that of a bone-former or not (Rogers *et al*. 1997). Briefly, the method consists of examining all the sites of entheses that are present in the skeleton and noting at which there is an ossified insertion, or enthesophyte.[3] The total score was divided by the number of insertions inspected and a score equal to or greater than 0.3 was considered to be evidence of bone-forming.

Using this criterion, 118 individuals were considered to be bone-formers or 15.3% (95% CI 13.0–18.1%) of the population on whom the necessary observations could be made. There was a considerable excess of males among the bone-formers and a much greater prevalence in those aged 45 or more at the time of death (Table 62). The prevalence in the males was 20.9% (95% CI 17.2–25.0%) compared with that in the females of 10.5% (95% CI 7.2–15.0), a significant difference. The increase in bone-forming in the older individuals in the late period is remarkable and reflects the increase in DISH in the males of that period, although of greater magnitude.

Among the bone-formers there were individuals with DISH and eDISH, and the bone-former scores were compared between the three groups, DISH, eDISH, and bone-formers with neither.[4] The mean bone-former score for the group with DISH was 0.80, for the eDISH group, 0.58 and for the remainder, 0.53 (Table 63). The difference between the DISH mean and the others was significant ($F = 10.57$, $p < 0.001$).

There are some interesting comparisons to be made between DISH, eDISH and bone-forming. DISH and bone-forming show most similarities, in that the prevalence of both increases with age. Bone-former scores are higher on average in DISH and it may really be seen as the most extreme expression of bone-forming. By contrast, the prevalence of eDISH is similar in males and females, there is no increase with age, and the mean bone-former scores are the same as in bone-forming. There are a number of explanations for this: either eDISH is really a different entity from DISH; eDISH may be a forme fruste of DISH (that is, only partially expressed) which is arrested before it develops into the full-blown entity; or, finally, the changes in eDISH proceed very much more slowly and may not develop until the individual is very elderly indeed. Which – if any – of these explanations is correct cannot be determined from the information which we have at present.

Hyperostosis Frontalis Interna (HFI)

Hyperostosis frontalis interna (HFI) is a condition in which there is thickening of the internal table of the frontal bone.[5] The thickening may take a number of forms and several types were described by Perou (1964) in his classic work. The condition was first described by Morgagni in 1719 in association with obesity and hursutism in an elderly female, and this triad of signs came to be considered as a distinct syndrome which was much later described – with the addition of some psychiatric symptoms – by Stewart (1928) and by Morel (1929); it is still sometimes referred to as Morgagni–Stewart–Morel disease, although its status as a discrete entity is rather dubious.

HFI occurs predominantly in post-menopausal women and is usually discovered fortuitously when the skull is X-rayed; it may sometimes be confused with Paget's disease or acromegaly, but it is almost asymptomatic unless the new bone is so extensive that it causes compression of the underlying cerebral cortex, in which case the patient may present with some cognitive impairment (Zubicaray *et al*. 1997). The aetiology of the condition is obscure but there is some thought that it may be related to hormonal stimulation (She and Szakacs 2004); leptin has been proposed as one such stimulant (Ruhli and Henneberg 2002), but the evidence to support this is sketchy.

HFI is easy to recognize in the skull, but only if the skull is broken, or if it is possible to examine the interior of the intact skull with an endoscope; radiography will detect gross lesions but smaller ones will escape notice. At Barton, the diagnosis was made only on those skulls that were broken; no attempt was made to include further cases with radiography or endoscopy. Under these constraints a total of 36 cases was detected; the majority were in females (20, compared with 13 males and 3 of unknown sex), and most cases were aged over 45 at the time of death (17 of the 24 that

were assigned an age). This is certainly an underestimate of the total number of cases in the assemblage because the interiors of intact skulls were not examined, and so the crude prevalence, 3.9% (95% CI 2.8–5.3%) should be considered as the minimum estimate.

Further analyses showed that whatever it is, HFI is not part of a general bone-forming tendency. Of the 36 cases, 20 were given a bone-forming score and only two of these were classified as bone-formers; this is a rather lower proportion of the assemblage as a whole, but the difference is not significant (95% CI 2.8–30.1%). Only one of the cases of HFI also had DISH and none had eDISH; the prevalence of DISH is similar to the overall prevalence (2.8%; 95% CI 0.5–14.2%).[6]

12. PALAEOPATHOLOGY VII: TUMOURS

The word tumour has an unpleasant connotation in the minds of most people, but in fact, it simply means a swelling and is used to refer to any kind of new growth,[1] wherever it arises. Tumours may be classified in a number of ways but, simply, they may be thought of as either benign or malignant, and either primary or secondary. A benign tumour is one that does not spread beyond the tissue in which it arises, whereas a malignant one spreads to distant tissues. A primary tumour is one which originates in the tissue in which it is found, whereas a secondary tumour (also referred to as metastases) is one found in some other tissue; a secondary tumour is by definition malignant but a primary tumour may be benign or malignant.

Primary bone tumours may arise in any of the tissues normally found in bone – bone, cartilage or blood-vessels, for example – and they may be benign or malignant. They account for only a small proportion of all tumours found in bone; the majority are secondary and spread predominantly from three primary sites, the lung, the breast or the prostate (Coleman 1997).

Primary bone tumours are relatively easy to recognize in bone; some of the malignant tumours produce florid growths of new bone while the benign tumours tend to be recognized from swellings of or on the affected bone. Secondary tumours arising from the lung and the breast form lytic lesions and their presence may be deduced by finding holes in the skull, the ribs or the long bones, for example. Secondary prostatic tumours are bone-forming and they are less easy to detect. They tend to spread to the pelvis and the vertebrae and their presence may be suspected if these bones seem to be unusually heavy; radiography may then show up sclerotic new bone within. There is a relatively rare form of prostatic cancer in which masses of periosteal new bone are formed, sometimes resembling the 'sunburst' seen with an osteosarcoma (Bloom *et al.* 1987; Nguyen 2003); this makes detection very easy (Waldron 1997).

Tumours are not reported very often in skeletal assemblages and this has led to the belief that malignant disease in particular was less common in the past than it is now. By no means all secondary tumours will be visible on visual inspection, however, and there is no doubt that were all adult skeletons routinely X-rayed considerably more cases would come to light, although even with radiography, tumours may be difficult to diagnose. Resnick, for example, says that 'It is with regard to tumors and tumor-like lesions that the interpreter of skeletal radiographs faces the greatest diagnostic challenges. It is essential to know the age of the patient and to have a good knowledge of the sites likely to be affected by the potential lesions' (Resnick and Niwayama 1988, 3617).[2]

Since malignant disease becomes common only in the elderly, it is more prevalent in ageing populations and, until quite recently, many fewer people survived into their 70s and 80s so that the total number of cases would have been less than in contemporary developed countries. Using a model to estimate the expected number of tumours in the past, however, suggests that the age-specific prevalence of malignant disease may not have been greatly different from what it is today (Waldron 1996).

Thirty-three skeletons at Barton were identified as potentially having tumours, although several of them were initially given other diagnoses. All were X-rayed and the radiographs read twice, on separate occasions. Furthermore, another discussion took place to make a firm decision as to the most probable diagnosis and, finally, only seventeen skeletons were considered to have tumours, ten with benign lesions and seven with malignant. For the reasons given above, however, these should be considered as minimum numbers; more would have undoubtedly have been found if all the skeletons had been X-rayed or if the skull had been examined endoscopically, for example.

The skeletons with benign and malignant tumours will be described separately and, finally, an account will be given of a special study conducted to measure the volume of the pituitary fossa with the aim of determining whether evidence could be found for the presence of pituitary tumours.

Benign Tumours

The ten cases of benign tumours found at Barton comprised eight different kinds (Table 64).[3] The sexes were equally distributed, but the age-range was very considerable, as may be seen from the table.

Bone cysts

Bone cysts are fluid-filled spaces within the medullary cavity. They occur most often in the proximal humerus and proximal femur and, although they are frequently asymptomatic, the affected bone may fracture if the cyst wall is unduly thin, and growth of the bone may be impaired (Violas *et al.* 2004).

Two cases, both adult females, were found at Barton and in both the cysts were present in the ilium. The first (sk. 1348, F4540) had a large (20 × 20 mm) lesion, approximately 10 mm deep, situated just above the left sacro-iliac joint. It was remodelled with related smooth edges but the wall and bottom surface of the lesion were 'fluted'. On X-ray the lesion appeared well defined, multi-loculated with a sclerotic margin, and there seemed little doubt that this was a simple bone cyst.

Table 64: Cases of benign tumours at Barton

Skeleton number (context)	*Phase*	*Sex*	*Age*	*Tumour type*
144 (F55)	A	Female	Adult	Haemangioma, skull
1348 (F4540)	A	Female	Adult	Bone cyst, left sacro-iliac joint
1507 (F4596)	A	Unknown	6–18 m	Fibrous cortical defect, fibula
2025 (F5183)	A	Unknown	<6 m	Button sequestrum, skull
1344 (F4530)	A/B	Female	Adult	Bone cyst, right pelvis
95 (F934)	B	Male	45+	Chondroblastoma, right femoral head
415 (F3408)	B/C	Female	Adult	Button sequestrum skull, Paget's disease
427 (F1280)	D	Male	35–45	Osteoma, skull
2242 (F7161)	D	Male	25–35	Osteochondroma, right humerus
1911 (F5032)	E	Male	45+	Chondroma, proximal phalanx of great toe

Fig. 90: Lesion in right ischium (arrowed) of an adult female (sk. 1344). The lesion penetrated through to the acetabulum and the radiological appearance suggested that it was a bone cyst. Photo: English Heritage

The second lesion (sk. 1344, F4530) involved the right ischium (Fig. 90). A hole was situated above the acetabulum measuring about 10 × 5 mm with remodelled edges. The lesion penetrated to the roof of the acetabulum and the X-ray appearances were entirely consistent with those of a bone cyst.

Button sequestrum

This is a radiological finding consisting of a radiodense focus surrounded by a circumscribed radiolucent area within a pathological area of a bone, frequently the skull. There are a number of conditions in which button sequestra are found, including Paget's disease and tuberculosis, and the sign has no specific diagnostic significance (Satin *et al.* 1976; Hoffel *et al.* 1992). Of the two cases found at Barton, one was in the skull of an adult female with Paget's disease (sk. 415, F3408) while the other (sk. 2025, F5183) was in the skull of an infant, less than six months of age; the associated pathology in this case could not be determined but may possibly have been tuberculosis.

Chondroblastoma

This is a rare cartilaginous tumour which originates in the epiphysis of the long bones, most frequently the femur, humerus or tibia (Springfield *et al.* 1985). It is most frequent in men in the second or third decade of life (Turcotte *et al.* 1993). The tumour may cause local pain, swelling and tenderness, and restriction of movement; on occasion it may also pursue a more aggressive course and produce widespread metastases (Ramappa *et al.* 2000).

In the head of the right femur of a male of at least 45 years of age (sk. 95, F934) there was a hole approximately 10 mm in diameter and 10 mm deep. The radiographic appearances of a chondroblastoma are of an osteolytic lesion with well-defined margins, spherical or oval in shape and usually less than 50 mm in diameter; the lesion in this case fitted these criteria and chondroblastoma was considered the most probable diagnosis.

Chondroma

This is a tumour that forms cartilage and develops within the substance of a bone (when it is known as an enchondroma), or on the surface of a bone (when it is called a periosteal chondroma). The tumours may be single or multiple.[4] Solitary chondromas occur most frequently in hands or feet (Stess and Tang 1995) and are usually asymptomatic, being noted only as a painless swelling. During the X-ray examination of a 35–44 year old male (sk. 1911, F5032) a small radiolucency was noted at the proximal end of a first proximal phalanx, which seemed most certainly to be a chondroma.

Fibrous cortical defect

These are tumours composed of whorls of connective tissue cells which are present most frequently in the tibiae and femora of children.[5] They are asymptomatic and are usually considered to be developmental defects. They are seen regularly as an incidental finding in up to 30–40% of X-rays of healthy children

(Caffey 1955). A small (20 × 10 mm) hole with smooth margins in the mid-shaft of one fibula in a one-year-old child (sk. 1507, F4596), with sclerotic margins on X-ray, had radiographic appearances typical of a fibrous cortical defect and the diagnosis did not seem to be in doubt.

Haemangioma

A haemangioma is one of several tumours composed of blood vessels (Wenger and Wold 2000), and occurs most frequently in the spine, skull or facial bones. These tumours are usually asymptomatic but may rarely cause compression of the spinal cord, or pain and facial deformity; they occur most often in middle-aged individuals and are more common in females than in males (Mirra 1989). A round lytic lesion with scalloped margins, and an irregular base with openings through to the endocranial surface, was found in the left frontal bone of an adult female (sk. 144, F55). It measured 15 × 20 mm and was located 30 mm anterior to the coronal suture and 20 mm lateral to the centre of the frontal; it was identified on X-ray as a haemangioma.

Osteochondroma

These tumours are cartilage-covered bony excrescences arising from the surface of bone. They are the most common benign bone tumour occurring most frequently in the proximal humerus, tibia and distal femur. Osteochondromas are probably developmental anomalies rather than true neoplasms, although on rare occasions they undergo malignant change and then exhibit the features of a true neoplasm (Fuselier *et al.* 1984). They may be single or multiple[6] and will be recognized during life as painless swellings on the affected bone, and very rarely they spontaneously regress (Revilla *et al.* 1999).

A single individual, a 15–24 year old male (sk. 2242, F7161) had a large swelling on the posterior surface of the proximal right humerus (the other humerus did not survive) and the radiographic appearances confirmed this as being a solitary osteochondroma.

Osteoma

Osteomas are tumours composed of normal bone. They are usually small, common and easy to recognize and have no clinical significance.[7] They may be found at all ages, but are most common over the age of 40 and a few are likely to be found in any skeletal assemblage. Unusually, there was only one such occurrence at Barton, in a male aged 35–44 (sk. 427, F1280) who had an osteoma near the lambda on the right parietal.[8]

Malignant Tumours

As expected, only a small number of the skeletons at Barton had morphological evidence of malignant change (Table 65). The great majority of cases were male and aged over 45 years at the time of death; it is likely in all these cases that the malignancy was the proximate cause of death.

Leukaemia

Leukaemia is a malignant transformation of white blood cells and there are several different forms, depending on which type of white cell is involved and on the age of the patient. The disease may cause a number of changes in the skeleton (Gallagher *et al.* 1996; Rogalsky *et al.* 1986) and the replacement of normal bone marrow by malignant cells may, in some cases, lead to areas of the periosteum being colonized by cells which escape from the medullary canal through vascular foramina. This results in the widening of the foramina, and exaggerated grooving and porosity of the cortical surface of the metaphysis.

There was a single young male (sk. 1448, F3723) at Barton who was found with an area of periosteal new bone on the shafts of both femora. It was mainly restricted to the medial side of the distal shaft and took the form of a swelling of the vascular markings and some linearity on the surface. There was also increased vascularity on the vertebral arches (Fig. 91). The radiographic changes suggested a possible diagnosis of leukaemia, or perhaps hypertrophic osteoarthropathy which, of course, may be a complication of leukaemia.

Table 65: Cases of malignant tumours at Barton

Skeleton number (context)	*Phase*	*Sex*	*Age*	*Tumour type*
4 (F84)	A	Female	Adult	Lymphoma, both femurs and tibiae
209 (F3055)	A	Unknown	*c.* 14	Round cell tumour, both tibiae
256 (F3336)	A	Male	45+	Multiple myeloma, spine
1448 (F3723)	A	Male	15–25	Leukaemia, spine, both femora
1630 (F4787)	A/B	Male	45+	Widespread metastases, prostate cancer
585 (F1631)	E	Male	45+	Multiple myeloma, skull
976 (F4021)	E	Male	45+	Widespread metastases, prostate cancer

Fig. 91: Increased vascularity of laminae of thoracic vertebrae in a young male (sk. 1448), probably caused by leukaemia. Photo: Juliet Rogers

Lymphoma

Lymphomas are malignancies that arise in lymphoid tissues. Primary lymphomas of bone may occur at all ages, although the majority of affected individuals are middle-aged and males are affected more often than females; the long bones are involved much more frequently than flat bones (Mulligan *et al.* 1999).

An adult female at Barton (sk. 4, F84) had pathological changes that affected the left femur and both tibiae. There was a fusiform swelling on the lateral side of the distal shaft of the femur and both tibiae had fusiform swellings in the mid-shaft. On X-ray, plaques of consolidated periosteal new bone were visible and there was also involvement of the endosteum; these are characteristic signs of a malignant marrow tumour, most likely a lymphoma.

Multiple myeloma

Multiple myeloma accounts for approximately 10% of all haematological malignancies and is primarily a disease of the elderly (Ruiz-Arguelles *et al.* 2004). It is caused by a malignant clone of plasma cells which remain localized to the bone marrow and which secrete a factor that activates osteoclasts, resulting in multiple lytic lesions in the skeleton (Barille-Nion and Bataille 2003). The abnormal cells secrete large amounts of monoclonal immunoglobulins that are present in the serum and excreted in the urine (Angtuaco *et al.* 2004). The lytic lesions are difficult to differentiate from other lytic malignancies of bone unless the abnormal immunoglobulin can be extracted from the bone, in which case the diagnosis is certain (Cattaneo *et al.* 1994).

Two individuals at Barton were identified with multiple myeloma, both elderly males. One (sk. 256, F3336) had lytic lesions in the lumbar spine with L5 showing evidence of vertebral collapse. This individual also had osteoarthritis of the left hip, right knee and feet, and fusion of the lower six thoracic vertebrae as the result of DISH. In the other case (sk. 585, F1631) only the skull was affected with two lytic lesions in the posterior part of the right parietal, one extending over the lambdoid suture on that side. They had sharp margins and were more extensive on the endocranial surface. The larger lesions measured 17 × 17 mm on the external surface and 20 × 20 mm internally. The smaller lesion was 6 × 4 mm on the outside of the skull and 10 × 10 mm on the inside.

Metastatic disease

In the contemporary population, bony metastases are most commonly due to the spread of carcinoma of the breast; the second most common cause is carcinoma of the lung (Katzer *et al.* 2002). Both cause lytic lesions in the skeleton, in the case of breast secondaries because the tumour cells secrete osteoclast-stimulating factors, in particular parathyroid hormone-related protein. Prostate tumours, by contrast, stimulate osteoblasts to make new bone (Moody 1997; Guise and Chirgwin 2003).

Fig. 92: Anterior surface of the sacrum in an elderly male showing roughening and new bone formation (sk. 976). Radiology confirmed this as a case of secondary spread from a primary prostatic carcinoma. Photo: English Heritage

There were two examples of skeletal lesions at Barton that were most probably caused by metastatic disease, secondary to carcinoma of the prostate. Both cases were elderly males with widespread proliferative changes throughout the skeleton. In the first case (sk. 1630, F4787) there was extensive, spongy new bone on the right scapula (the left was missing), the pelvis, ribs, sternum, the distal right clavicle, proximal right humerus and proximal right femur. The changes in the second case (sk. 976, F4021) included roughening of the bone surface and increased vascularity affecting the spine, ribs, sternum, pelvis, sacrum, clavicles and femoral heads (Fig. 92). Small areas of new bone with a hair-on-end appearance were present on the pelvis, left scapula and some ribs. The skull, tibiae and fibulae appeared to be normal. In both cases, radiographs showed the presence of sclerosis within the affected bones which was consistent with the appearance of secondary prostatic cancer.

Round cell tumour

A round cell tumour is one of a group of tumours that share a common histological appearance following staining, and differentiation of the various types, which includes Ewing's sarcoma and rhabdomyosarcoma, may be difficult (Pisick *et al.* 2003). They tend to be aggressive tumours and survival is poor, even with modern treatments (Eralp *et al.* 2002).

Fig. 93: Left tibial shaft from a young child (sk. 209) showing a lytic lesion surrounded by periosteal new bone. The lesion does not penetrate through the cortex, as shown in the accompanying radiograph. Photos: Juliet Rogers

The skeleton of a young child, 13–15 years of age (sk. 209, F3055), was probably affected with such a tumour. The left tibia had a thick layer of periosteal new bone of a greyish, woven appearance over the subcutaneous surface. This area of bone was interrupted by a lytic lesion passing through the full thickness of the new bone and the original cortex; the margins were not remodelled (Fig. 93). The left fibula also had a thin layer of new bone, as did the right tibia. The morphology of the left tibia was somewhat similar to that seen in osteomyelitis, the X-ray appearances showed the presence of the new bone and a radiolucency, and a malignant round cell tumour was considered to be the most probable diagnosis.

Prevalence of Neoplasms

The prevalence of neoplasms at Barton, both benign and malignant, for the early and late periods is shown in Table 66. As expected, the prevalence is very low – although as mentioned earlier, for the malignant tumours the figures must be considered as the minimum in the population – and there has been no apparent change in the frequency of either type of tumour over time.

A Study of Pituitary Volume

A special study was carried out to measure the pituitary volume in a randomly selected sample of skulls in order to determine the likelihood of any of them having had a benign pituitary tumour (Crossley *et al.* 1996). Initially, 88 skulls were randomly selected, and of these 77 met the entry criteria for the study, that is, the skulls were from adults and were intact.[9] The age structure of the study population was as follows:

15–	13
25–	39
45+	25

Seventy-four could be assigned to a phase and 26 of these were from the early period and 48 from the late.

Each skull was X-rayed in the antero-posterior and lateral positions and the pituitary volume was calculated from the resulting films; the upper limit for the normal volume was taken as 1092 mm^3.[10] It was found that 17 of the 77 skulls had a pituitary volume that was greater than the upper limit of normal, 5 from the early period and 12 from the late period. There was no trend for an increase in abnormal volume with increasing age, as may be seen in Table 67. The X-rays of the 17 skulls with an abnormal volume were re-examined for signs of asymmetrical erosion of the pituitary fossa and four were considered to be positive, a prevalence in the total sample of 5.2% (95% CI 2.0–12.6).

Table 66: Crude prevalence of benign and malignant tumours (% and 95% CI) at Barton, by sex and period

	Male	*Female*	*Children*	*Total*
Benign tumours				
Early	0.7 0.2–2.0	0 0–1.0	0 0–0.9	0.2 0.1–0.7
Late	0.5 0.1–2.8	1.6 0.5–4.5	1.0 0.3–3.4	1.0 0.5–2.2
Malignant tumours				
Early	0.5 0.1–1.7	0 0–1.0	0 0–0.9	0.2 0–0.6
Late	1.5 0.5–4.4	0.5 0.1–2.9	0.5 0.1–2.6	0.8 0.4–2.0

Table 67: Age-specific prevalence of abnormal pituitary volume at Barton

Age	*Prevalence*	*95% Ci*
15–	15.4	4.3–42.2
25–	28.2	16.5–43.8
45+	16.0	6.4–84.7

The underlying assumption of this study was that expansion and erosion of the pituitary fossa would be caused by the presence of a pituitary adenoma, although there are other causes, including a Rathke's cleft cyst (Voelker *et al.* 1991).[11] The prevalence of pituitary adenomas as reported in the literature is very variable, depending on the means used to diagnose them. The tumours are often divided in micro-adenomas (<10 mm in size) and macro-adenomas (>10 mm). The prevalence of all adenomas has been estimated to be 16.7% in a review of several published studies, but the prevalence of macro-adenomas is only about 0.2% (Ezzat *et al.* 2004). Many micro-adenomas would be so small that they would not give rise to changes in the pituitary fossa and there is actually little correlation between minor radiological signs such as erosion of the fossa and the presence of a tumour (Hall *et al.* 1994). It is clear that the majority of pituitary adenomas are silent but they may be accompanied by a variety of clinical signs, depending on which of the secreting cells of the pituitary is involved (Ironside 2003); they may also be accompanied by headaches and a disturbance of the visual fields if the tumour impinges upon the optic chiasm.

It is not clear whether the four individuals found with abnormal radiological signs actually had a pituitary adenoma and, if so, whether they had symptoms, but it seems reasonable to assume that one or more may have done so.

13. PALAEOPATHOLOGY VIII: OTHER DISEASES AND CONDITIONS

This chapter will consider a number of congenital and developmental conditions, and some skeletal anomalies that are asymptomatic and do not really constitute disease as it is usually understood, although they are invariably described under the heading of palaeopathology for want of a better rubric.

Fibrous Dysplasia

During the examination of the skeleton it is common to find bones with abnormal shapes or surfaces. The morphology is often sufficiently distinctive for a provisional diagnosis to be made – osteomyelitis or Paget's disease, for example – but in other cases, there are no distinguishing features and diagnosis depends upon X-ray findings. In this way, additional cases of chronic infection, Paget's disease or malignancy may be found. One condition that was diagnosed solely on the basis of the radiographic appearances was fibrous dysplasia.

Fibrous dysplasia is caused by a defect in the formation and maturation of bone and cartilage cells, which leads to well-defined intra-medullary lesions of fibrous tissue and calcified cartilage with cyst-like areas. The condition may affect a single bone (monostotic) or several bones (polyostotic). It may cause pain in and deformation of the affected bones and, because the area of affected bone is relatively weak, pathological fractures are common. The molecular basis of the disease is now well understood (Marie 2001) and this has led to improvements in treatment (Chapurlat and Meunier 2000). Polyostotic fibrous dysplasia may be associated with the presence of café au lait spots on the skin and various endocrine abnormalities, when it is referred to as McCune–Albright syndrome (Hannon *et al.* 2003); it would not be possible to differentiate this condition from polyostotic fibrous dysplasia in the skeleton.[1]

Three individuals were found with fibrous dysplasia at Barton, one monostotic and two polyostotic (Table 68). In one 25–34 year old female (sk. 2785, F7624), the entire shaft of the left femur was swollen, being slightly wider distally. The surface was roughened and irregular and although no cloacae were present, osteomyelitis was initially considered to be the most likely diagnosis. In the case of a 35–44 year old male (sk. 2799, F7632), both the right radius and tibia were swollen. The swelling of the radius was particularly noticeable at the insertion of the biceps muscle, and there were some spicules of bone on the surface of the shaft. The whole of the tibia was grossly swollen with an irregular surface, and again – despite the presence of cloacae – osteomyelitis was considered to be the most likely explanation for these changes. In the final case (sk. 1678, F4655), swellings were found on the ribs of a very poorly preserved skeleton; because of the state of preservation, it was not possible to say whether other bones had also been affected. On X-ray, the changes in every case were typical of fibrous dysplasia and this was the agreed final diagnosis.

Skeletal Dysplasia

There are a great number of conditions that fall within this classification, the best known non-lethal condition being achondroplasia, caused by a defect in the fibroblast growth receptor gene (FGFR3), leading to a defect in the formation of cartilage (Papadatos and Bartsocas 1982). The genetic defect in many of these conditions is now well understood (Ozono 1997), and a skeletal gene database has been established to provide information about bone-related genetic disorders (Jia *et al.* 2001).

The skeletal dysplasias are all uncommon and most are rare so that skeletons with dysplasia are seldom found within an archaeological assemblage. There was one skeleton of an adult female at Barton (sk. 1667, F4807), however, that was very abnormal. The skeleton was poorly preserved with only the left glenoid, most of the bones of the hands, the bones of the forearms, the pelvis, the lower thoracic and lumbar vertebrae, some ribs and the sternum present. The vertebrae were increased in height but the bones of the pelvis were all very gracile and light. The area occupied by the sacroiliac joints was much smaller than normal, both acetabula were shallow with a defect in their superior margins, and the sciatic notches were very wide. By contrast, the radii, ulnae and the carpal bones were large and robust enough to belong to a male. The vertebrae appeared normal on X-ray but the pelvis seemed dysplastic.

Table 68: Cases of fibrous dysplasia at Barton

Skeleton number (context)	*Phase*	*Sex*	*Age*	*Bones affected*
1678 (F4655)	A/B	Male	25–34	Ribs (very little of the remainder of the skeleton had survived)
2785 (F7624)	D/E	Female	25–34	Left femur
2799 (F7632)	E	Male	35–44	Right radius and right tibia

It was unfortunate that the lower limbs were missing although the changes in the pelvis were not typical of those seen subsequent to lower limb paralysis, for example. The skeleton was presented to the Bristol Bone Dysplasia Group in the hope that they might be able to suggest a diagnosis, but none was forthcoming and the cause of these changes remains unknown.

Developmental Dysplasia of the Hips

This term has superseded the old term, congenital dislocation of the hips, because the condition is not always present at birth and does not always result in dislocation. It occurs in approximately 1% of all neonates and several risk factors are known including breech presentation, family history and post-natal swaddling (Backe *et al.* 2002); girls are much more likely to be affected than boys and the left hip is affected about four times as commonly as the right (Rosendahl *et al.* 1996).

Two skeletons at Barton were found with the condition, both female; one was aged 15–24, and the other an adult of uncertain age. In the first case (sk. 2427, F5389), the right hip was affected. The acetabulum was obliterated and there was a false acetabulum on the blade of the ilium, and the femoral head was also dysplastic. Although it was first considered that this might be a case of tuberculosis, developmental dysplasia was finally agreed as the most likely diagnosis.

The second skeleton (sk. 1710, F4822) had lesions of both hips. Again, both acetabula had been obliterated and remodelled and large pseudo-arthroses were present on the ilium, posterior to the normal position. Both acetabula were wide and shallow and both were eburnated, indicating that the individual had survived a long time with the condition. It is very likely that she would have walked with a waddling gait as the condition had clearly not been corrected during her lifetime.

Idiopathic Scoliosis

Scoliosis refers to a lateral curvature of the spine. There are a number of causes for scoliosis (Burwell 2003), including an association with the Klippel–Feil syndrome (Thomsen *et al.* 1997) (see below), but the commonest form is the so-called idiopathic form. Three types of idiopathic scoliosis are recognized: infantile which occurs before the age of 3 and which resolves spontaneously in most cases; juvenile, occurring between the ages of 3 and puberty and usually progressive; and adolescent. Adolescent scoliosis is the most common, occurring more often in girls than in boys, and the prevalence increases with age, from 6 to 12 (Stirling *et al.* 1996).

Scoliosis is a complicated deformity for, in addition to the lateral curvature, there is also rotation of the vertebrae. As the disease progresses, the vertebrae and spinous processes rotate towards the concavity of the curve, the posterior parts of the ribs on the convex side are pushed backwards, causing the characteristic hump in the chest, while the anterior parts of the ribs are pushed anteriorly. The ribs are often thinner than normal, the vertebrae are wedged towards the concave side, osteophytes may be present, and osteoarthritis of the costo-vertebral joints.

The severity of the condition varies, from children with only a slight non-progressing curvature to those in whom the curve measures more than 40° by the so-called Cobb method, and who would nowadays generally be considered for surgery.[2] In those with a substantial deformity, severe cardiac and respiratory complications may ensue in later years (Branthwaite 1986); back pain is a common feature of the condition but most people with it function well and experience no shortening of life (Weinstein *et al.* 2003).

There were six skeletons at Barton with scoliosis in two of which (sk. 565, F1615 and sk. 1425, F3683) the condition seemed to be secondary to poliomyelitis; in another (sk. 190, F3308) the cause seemed to be traumatic,[3] but the remaining three were considered to be idiopathic, although the age of onset could not, of course, be determined so they may have been of either the juvenile or the adolescent form (Table 69).

Two of these skeletons were poorly preserved. In one (sk. 2389, F4929), only the lower four thoracic vertebrae had survived although all the lumbar vertebrae were present. There was a curve to the left of T10 and T11, a curve to the right of L1 and L2, and another curve to the left of L4 and L5 (Fig. 94). The vertebral arches were mostly damaged, but the corkscrewing of the spine was obvious when the vertebral bodies were

Table 69: Cases of idiopathic scoliosis at Barton

Skeleton number (context)	*Phase*	*Sex*	*Age*	*Region affected*
1939 (F7003)	A	Female	Adult	Thoracic scoliosis to right; associated fracture of two ribs
1951 (F7021)	A	Female	Adult	Upper thoracic curve to left and with kyphosis, T3–6 fused; lower thoracic curved to right with T8–10 fused posteriorly; T11 and T12 fused posteriorly. Ribs very slender and lacking normal curve
2389 (F4929)	E	Male	Adult	Curve to left of T10 and T11; only lower four thoracic vertebrae present. L1 and L2 curved to right; L4 and L5 curved to left. Transitional vertebra with vestigial right sacral facet joint

assembled. In the second of this group (sk. 1939, F7003) all the vertebrae had survived although their condition was poor, but nevertheless a thoracic scoliosis to the right could easily be made out. This individual had been subject to trauma at some time and two of the rib fragments that were present each had two healed fractures.

Fig. 94: Spinal column from the rear from a male of unknown age with scoliosis (sk. 2389). There is a curve to the left of T10 and T11, a curve to the right of L1 and L2, and another curve to the left of L4 and L5. Photo: Juliet Rogers

The best preserved skeleton of the three (sk. 1951, F7021), an adult female, showed the most extensive changes of the spine and the ribs. There was minimal change in the five remaining cervical vertebrae apart from a slight displacement of the arches to the right. All the thoracic vertebrae had survived: T3, T4 and T5 were fused around the bodies and the facet joints with a left scoliosis and some kyphosis. The eighth, ninth and tenth vertebrae were also fused posteriorly with right-sided scoliosis; T11 and T12 were also fused around the facet joint. The lumbar vertebrae were normal although there was bilateral osteoarthritis of the facet joints of T12 and L1. The ribs were abnormal in shape, as expected, being gracile and twisted. The marked curvature of the spine was readily apparent when the spine was re-assembled.

Spina Bifida Occulta

Spina bifida occulta is the term used to describe a sacrum in which all the posterior laminae are unfused. It is clinically insignificant, unknown to the individual, and not to be confused with the condition of spina bifida in children, in which the defect occurs in the lumbar spine and which may be accompanied by a herniation of the contents of the spinal cord; this most serious condition would invariably have been quickly fatal in the past. The most mild form of the condition in children is also called spina bifida occulta and may be silent, but in some children there is a dimple or a tuft of hair over the defect in the underlying vertebra. To avoid confusion, it would be preferable to use some other term to describe the condition in the sacrum such as bifid sacrum, but long usage of the usual phrase probably militates against any change in nomenclature.

Bifid sacrum is one of the easiest conditions to recognize in the skeleton and, rather surprisingly, only four cases were found at Barton (Table 70), two of which were associated with other minor abnormalities of the spine. The prevalence of the condition was only 1.4% (95% CI 0.6–3.6%), which is considerably lower than a prevalence of 8% that is found at some other sites (Saluja 1986), or 11% found in a large series of unselected radiographs (Brailsford 1929). This is not a condition that could easily be overlooked and so there is very little possibility of misdiagnosis and, for reasons which are obscure, it seems that its prevalence really is lower at Barton than would have been expected.

Table 70: Cases of bifid sacrum at Barton

Skeleton number (context)	*Phase*	*Sex*	*Age*	*Associated anomalies*
1963 (F5112)	A	Male	25–34	Spinous process of T12 and T12 unfused
1973 (F5129)	A	Male	25–34	
2344 (F7230)	C/D	Unknown	13–15	L5 partially fused to S1
1312 (F3272)	E	Male	15–24	

Transitional Vertebrae

The L5/S1 junction tends to be unstable and the lower lumbar vertebra may on occasion have an expanded transverse process which may articulate or fuse with the wing of the sacrum. At Barton, 15 instances of transitional vertebra were noted, a prevalence of 1.6% (95% CI 1.0–2.6%). This again is lower than the prevalence reported in the early literature in either unselected skeletons (Moore 1924) or in the general population (6–8.1%) (Lanier 1939; Brailsford 1929), and the reasons for this discrepancy are, again, not obvious.

Six Lumbar Vertebrae

Another aspect of the instability of the lower lumbar spine is the presence of six lumbar vertebrae, which some consider to be an atavistic condition, mimicking the situation in the early hominids.[4] There were 44 skeletons with six lumbar vertebrae, 39 adults and 5 juveniles, giving a crude prevalence of 4.2% (95% CI 3.1–5.7). This is similar to the prevalence of 3% found in a large radiographic study by Ford and Goodman (1966).

Relationship between bifid sacrum, transitional vertebrae, six lumbar vertebrae and spondylolysis

There are a number of instances in the literature in which an increase in the prevalence of bifid sacrum has been found in skeletons with spondylolysis, and a decrease in the prevalence of transitional vertebrae. A case-referent study of British material, however, failed to confirm either relationship (Waldron 1993) but nevertheless it was decided to investigate the situation at Barton. The four cases of bifid sacrum had none of the other conditions, although two were associated with minor vertebral abnormalities (Table 70).

Table 71: Related crude prevalences of some abnormalities of the lumbar spine

	Six lumbar vertebrae	*Transitional vertebrae*	*Spondylolysis*
Six lumbar vertebrae		12.8* 5.6–26.7	12.8* 5.6–26.7
Transitional vertebrae	33.3* 15.2–58.3		6.7 1.2–29.8
Spondylolysis	10.6 4.6–22.6	2.1 0.4–11.1	

The prevalence of the other two conditions in those with the condition shown in the first column is ascertained by reading horizontally across the table.

*Prevalence significantly different from crude prevalence in total assemblage

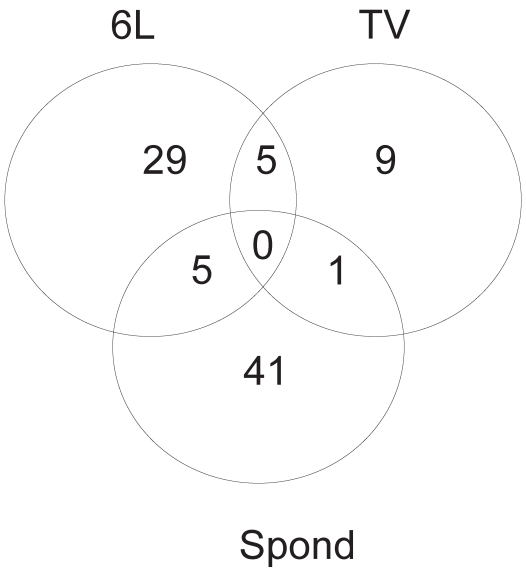

Fig. 95: Distribution of six lumbar vertebrae (6L), transitional vertebrae (TV), and spondylolysis (Spond) at Barton

The relationships between the other three conditions are shown in Fig. 95, and Table 71 shows the prevalence of each with respect to the others. There is a significant difference between the prevalence of transitional vertebrae and spondylolysis in those with six lumbar vertebrae, and between the prevalence of six lumbar vertebrae in those with transitional vertebrae, compared with the crude prevalence in the whole assemblage. These data suggest that there is an association between having six lumbar vertebrae and transitional vertebrae, and that those with six lumbar vertebrae are more susceptible to spondylolysis.

Other Skeletal Anomalies

There were fourteen other skeletons at Barton with a variety of other skeletal anomalies which are detailed in Table 72. Some of the anomalies are trivial: those involving minor changes in the vertebrae or the fusion of tarsal bones or ribs, for example. The two examples of dysplastic hips presented with shallow acetabula and a degree of coxa valga, but there was no evidence of any associated pathology.

Hydrocephalus

One skeleton, that of a baby (sk. 1931, F7012), had extensive deposits of periosteal new bone on both the internal and external tables on the skull, in excess of what could be considered to be physiological. There were no other changes, but when the skull was X-rayed, the radiologist considered that hydrocephalus was a possible cause; in the absence of confirmatory changes in the skull, this must, however, be taken as a very tentative diagnosis.

Table 72: Cases with skeletal anomalies at Barton

Skeleton number (context)	*Phase*	*Sex*	*Age*	*Anomaly*
252 (F3332)	A	Male	25–34	Small gap in right posterior arch of atlas; right atlanto-occipital joint double; fusion of T2/3
426 (F1565)	A	Unknown	45+	Fusion of C4–6; Klippel–Feil syndrome
1081 (F3156)	A	Unknown	Adult	Right tibia and talus fused; distal tibia slightly expanded with increased vascular markings
1931 (F7012)	A	Unknown	Baby	?hydrocephalus
1965 (F5109)	A	Female	Adult	Unfused posterior arch in atlas; 7 mm gap
1992 (F7036)	A	Male	Adult	Dysplastic hips
453 (F3422)	A/B	Female	Adult	Fusion of C3/4; Klippel–Feil syndrome
1454 (F3727)	A/B	Male	25–34	Right external auditory meatus absent; microtia
1460 (F4737)	A/B	Male	15–25	Dysplastic hips
109 (F392)	B	Male	45+	Fused left ribs
405 (F3064)	B/C	Female	Adult	Fusion of C3/4; Klippel–Feil syndrome
1558 (F4628)	B/C	Female	25–34	Bifid arch L5
1083 (F3159)	C/D	Unknown	Adult	Left talus and calcaneus fused
2374 (F7244)	C/D	Unknown	1½–2	Right external auditory meatus absent; microtia

Microtia

Two skeletons (sk. 1454, F3727 and sk. 2374, F7244) were found in which the right external auditory meatus was missing. This defect is commonly associated with microtia, that is, absence of the majority of the pinna. It is not common, occurring in approximately one in 10,000 births; it is more frequent in males, and may be either unilateral or bilateral. When unilateral, the right ear is more often affected than the left, as here. In a substantial number of children with this condition, there are associated malformations of the face or heart (Harris *et al.* 1996), or malformations of the inner ear (Calzolari *et al.* 1999). Typically, there is a conductive hearing deficit on the affected side.

There is a tendency for microtia to run in families (Llano-Rivas *et al.* 1999), but the large temporal separation between the two cases at Barton almost certainly precludes them from being closely related.

Klippel–Feil syndrome

This is a condition in which there is congenital fusion of the cervical vertebrae, with or without fusion in other regions of the spine. The true incidence of the condition is not known with certainty but the prevalence has been found to be less than 1% in radiographs of a modern population (Gjorup and Gjorup 1964) and in the skeletons in the Terry collection (Brown *et al.* 1964). (Although incidence is mentioned in the title of this paper, it is prevalence that the authors were investigating.) A genetic locus for the condition has been identified although it is not clear whether it is a discrete entity or part of a continuum of congenital spinal disorders (Tracy *et al.* 2004). Three types of Klippel–Feil syndrome (KFS) are described and all those at Barton were type II, that is, the fusion was confined to the cervical spine. Types I and III are associated with a high risk of scoliosis (Thomson *et al.* 1997); in type II the risk is low and it was interesting that none of the Barton cases had scoliosis.

The prevalence of KFS at Barton was 0.3% (95% CI 0.1–0.9%), which is in line with modern observations. The individuals with the disorder would probably not have been greatly inconvenienced by it, although they may have noted some limitation of movement in the neck. They may also have been noted to have a short, slightly webbed neck which is one of the concomitants of the syndrome.

14. PALAEOPATHOLOGY IX: DENTAL DISEASES

Teeth generally survive well and dental disease always accounts for a substantial proportion of the total amount of pathology seen in a skeletal assemblage. At Barton the presence or absence of individual teeth was noted, whether a tooth was lost *post mortem* or *ante mortem*, whether it was unerupted and whether it had caries or an apical abscess associated with it. The degree of calculus was also noted and the degree of alveolar disease; these were both scored on a four-point scale. No attempt was made to categorize caries beyond a simple present/absent dichotomy, nor was any attempt made to detail any dental anomalies. What follows is a simple account of dental disease which could be supplemented by reference to more specialist investigations.[1]

Number of Teeth Present

A total of 24,354 adult teeth could be accounted for, 15,003 of which were *in situ* or loose, and 9,351 of which were absent, either through *ante mortem* or *post mortem* loss, or because they were unerupted (Table 73). This represents 39.3% of the total of 62,080 expected, a rather low proportion.[2] A greater proportion of the number of expected lower teeth was present than the upper, 44.7% compared with 33.7%. The data in Table 73 show that in percentage terms the pre-molars were better represented than the anterior teeth (incisors and canines) and molars; this may be explained by the fact that the anterior teeth are more susceptible to trauma and the molars to dental disease. The anterior part of the maxilla and the mandible are also more likely to be damaged *post mortem*, with consequent loss of the teeth.

Ante Mortem Tooth Loss

Teeth may be lost for a variety of reasons – accidental trauma, fighting, scurvy or deliberate extraction – but the majority are lost as the result of periodontal disease (Chapple 2004); see further below. The socket of a tooth that has been lost during life will quickly begin to remodel and eventually it may be completely filled in. By contrast, the socket of a tooth that has been lost after death will be pristine with no trace of remodelling, so that there is no difficulty in distinguishing between the two states, the only exception being if the teeth were lost at or around the time of death. There is also no way to tell exactly the mechanism by which the tooth was lost, although the presence of a fractured jaw with tooth loss would be suggestive, as would the presence of a considerable degree of periodontal disease.

Table 73: Number of teeth accounted for at Barton

	Anterior	*Pre-molars*	*Molars*	*Total*
Teeth present				
Upper	2437	1932	2164	6533
Lower	3050	2460	2960	8470
Total	5487	4392	5124	15003
% of total expected	23.6	28.3	22	24.2
Teeth absent				
Upper	1775	752	1411	1411
Lower	2172	1042	2199	5413
Total	3947	1794	3610	9351
% of total expected	17	11.6	15.5	15.1
Grand total	9434	6186	8734	24354

Table 74: Distribution of *ante mortem* tooth loss at Barton

	Anterior	*Pre-molars*	*Molars*	*Total*
Upper jaw				
n	433	240	1024	1697
% of total	9.4	5.2	22.2	
Lower jaw				
n	654	529	1724	2907
% of total	14.2	11.5	37.4	
Grand total	1087	769	2748	4604

Table 75: Proportion of individuals with *ante mortem* tooth loss at Barton, by age and sex

Age (years)	*Male*	*Female*
15–	40.0	39.3
25–	49.3	45.0
35–	91.0	66.7
45+	85.0	83.1

A total of 650 individuals had lost teeth during their lifetime at Barton, 326 males and 278 females, and between them they had lost 4,604 teeth, an average of 7.1 per individual. Substantially more teeth were lost from the lower jaw than the upper, but in both jaws, molar teeth were more frequently lost than any other type (Table 74). The proportion of individuals who lost teeth at different ages is shown in Table 75; in both sexes there is a very obvious trend for an increased tooth loss with increasing age, as would be expected.

Dental Caries

Dental caries is the destruction of the structures of the teeth by the action of acid produced by bacteria in dental plaque.[3] The first sign of caries on the enamel surface is a white or brown spot, the brown spot indicating that the lesion has arrested. The white spot continues to develop, destroying the enamel and the dentine, to form a cavity. The lesions vary in extent and may be found on any of the tooth surfaces or on the roots if they are exposed, but the location of the lesions has varied in time according to the diet (Hillson 1996). There are a number of elaborate systems for recording the site of caries on the different tooth surfaces,[4] but none was used here.

In total, 839 teeth were found with caries (Fig. 96), occurring in a total of 399 adults (182 males and 190 females), an average rate of 2.1 caried teeth per individual. There were, in addition, 27 children with caried milk teeth. As may be seen from Table 76, the distribution of caries throughout the mouth was again very uneven, by far the greater proportion of caried teeth being molars, and most of those were in the lower jaw (Fig. 97).

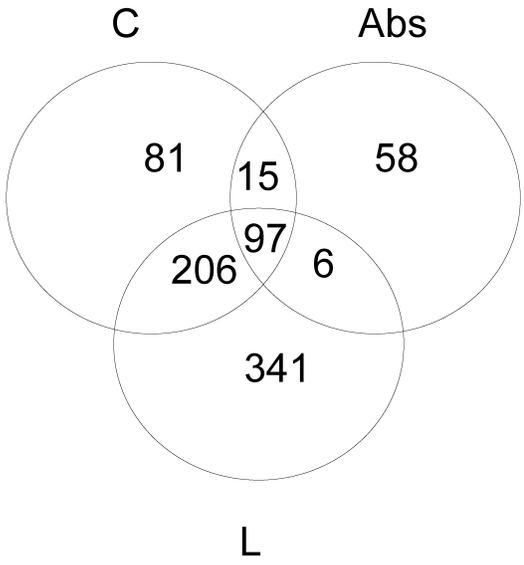

Fig. 96: Distribution of dental caries (C), ante mortem *tooth loss (L), and dental abscess (Abs) at Barton*

Table 77: Proportion of individuals with dental caries at Barton, by age and sex

Age (years)	*Male*	*Female*
15–	30.0	47.6
25–	40.6	40.7
35–	34.3	50.0
45+	46.8	53.9

The proportion of individuals affected by caries by sex and age is shown in Table 77. The trend for increasing caries with increasing age is much stronger than for *ante mortem* tooth loss, and the fact that almost half the youngest females had at least one caried tooth is remarkable.

Dental Abscess

An abscess in the jaw is easily recognized as a defect in the bone around the root of the affected tooth if the tooth remains *in situ*. Abscesses in children are usually secondary to caries which invades the pulp cavity

Table 76: Distribution of dental caries at Barton

	Anterior	*Pre-molars*	*Molars*	*Total*
Upper jaw				
n	97	129	198	424
% of total	11.6	15.4	23.6	
Lower jaw				
n	44	82	289	415
% of total	5.2	9.8	34.4	
Grand total	141	211	487	839

Table 78: Distribution of dental abscesses at Barton

	Anterior	*Pre-molars*	*Molars*	*Total*
Upper jaw				
n	30	41	75	146
% of total	10.7	14.6	26.8	
Lower jaw				
n	25	24	85	134
% of total	8.9	8.6	30.4	
Grand total	55	65	160	280

Table 79: Proportion of individuals with dental abscesses at Barton, by age and sex

Age (years)	*Male*	*Female*
15–	8.3	15.5
25–	13	15.7
35–	37.1	16.7
45+	25.4	25.8

Table 80: Crude prevalence of dental disease (% and 95% CI) at Barton

Period	*Caries*	*Ante mortem tooth loss*	*Abscess formation*
Early	34.6	56.5	15.9
	30.9–38.4	52.6–60.4	13.2–19.0
Late	49.6	84.3	19.7
	47.8–55.5	79.5-88.1	15.4–24.8
Total	39.7	64.7	17.5
	36.7–42.8	61.7–67.6	15.3–20.0

Fig. 97: Left mandible from a female of c. *20 years of age with caries of the second molar (sk. 2222). There is also a moderate degree of alveolar disease. Photo: English Heritage*

producing inflammatory change in the pulp. This can progress to necrosis, with bacterial invasion of the alveolar bone. In adults, the most common form of abscess is a periodontal abscess in which the supporting structures of the teeth are involved. Many species of bacteria are involved in the infection, which is painful but usually otherwise uncomplicated, although osteomyelitis of the jaw may follow; the mandible is affected more often than the maxilla, because the maxilla has a better blood supply (Herrera *et al.* 2000). Maxillary sinusitis may also follow if the infection breaks through the floor of the sinus (see Chapter 8).

A total of 176 individuals at Barton had dental abscesses, 89 males and 77 females, with a total of 280 lesions; this is an average of 1.6 per individual affected. As before, the molars bore the brunt of the damage (Table 78), and there was some evidence for a trend of increasing disease with increasing age (Table 79).

Relationship Between Different Dental Diseases

The relationship between caries, abscess formation and *ante mortem* tooth loss is shown in Fig. 96. Of the 804 individuals with dental disease a quarter had both caries and tooth loss and almost one in eight had all three types of lesion. The most common single event was *ante mortem* tooth loss which, in most cases, was probably secondary to periodontal disease.

The Prevalence of Dental Disease

The prevalence of each of the three types of lesion studied here is shown in Table 80. The dental health of the population seems to have been very poor for most of the period, with almost 40% of the adults having caries and almost two-thirds having lost teeth during life. There is a striking and significant increase in the prevalence of caries and of *ante mortem* tooth loss in the late period; the rise in the prevalence of abscesses is not significant.

Periodontal Disease

Periodontitis – inflammation of the periodontal tissues – is caused by bacteria present in dental plaque and

Table 81: Proportion of individuals with dental disease at Barton, by grade of periodontal disease

Dental disease	*Grade of periodontal disease*			
	1	*2*	*3*	*4*
Caries	40.8	56.0	68.8	88.5
Ante mortem tooth loss	35.7	39.9	49.0	61.5
Abscess formation	12.2	18.6	20.2	26.0

Table 82: Proportion of individuals with dental disease at Barton, by grade of dental calculus

Dental disease	*Grade of dental calculus*			
	1	*2*	*3*	*4*
Caries	67.6	61.3	64.6	68.3
Ante mortem tooth loss	40.1	45.3	47.0	65.0
Abscess formation	18.6	20.3	21.6	15.0

follows on from gingivitis (Kinane 2000). There is a release of cytokines which initiate inflammation and the production of factors that result in destruction of the alveolar bone (Page 1998). There is a strong link between periodontal disease and other systemic diseases, some of which are potentially fatal (Page 2002).[5] In the skeleton, periodontal disease is recognized by recession of the alveolar margin; two types of bone loss have been recognized on dental X-rays, horizontal and vertical (Goaz and White 1994), but this distinction is not always made when reporting it in the skeleton, and this was the case here. Instead, a simple four-point score was recorded for each individual, from 1 = no alveolar recession, to 4 = considerable recession. The scoring is entirely subjective, but was done by the same observer and has clinical significance so that it is a worthwhile exercise.

The proportion of individuals with the various forms of dental disease by grade of periodontal disease is shown in Table 81. There is a marked trend for the proportion of those with all forms of disease to increase as the grade of periodontal disease increases, and it is clear that the mouths of those with the most severe form of periodontal disease would have been in a fearful state: most having caries, two-thirds having lost teeth, and a quarter having dental abscesses, not to mention the unpleasant effects of all having swollen, infected gums and, almost certainly, halitosis.

Dental Calculus

Dental calculus is mineralized plaque composed primarily of calcium phosphate. It occurs in two forms, supra- and subgingival – that is, calculus on the crown of the tooth or the exposed roots – and both forms occur in the majority of adults (White 1997). There is considerable site specificity for supragingival calculus which tends to occur most often on the lingual surfaces of the lower anterior teeth, presumably because this is the most alkaline area of the mouth, since it is constantly bathed in saliva, and calculus develops in an alkaline environment (Dawes1998). When recording the presence of calculus in the skeleton the distinction between supra- and subgingival calculus is frequently not made and although some elaborate scoring methods have been developed (Bodney and Brothwell 1986), the same simple four-point scale was used here as for periodontal disease.

As may be seen from Table 82, the presence of calculus does not seem to affect the prevalence of dental disease, with the exception of *ante mortem* tooth loss where the number of individuals who have lost teeth increases moderately in the first two positive grades (2 and 3), but increases markedly in the final grade. The reasons for this are not immediately obvious.

Enamel Hypoplasia

Enamel hypoplasia in children has been discussed in Chapter 8 but, in addition to the children with this condition, six young adults were noted to have it, three males (sk. 604, F1674; sk. 1414, F4555 and sk. 2226, F7499) and three females (sk. 1059, F4062; sk. 1346, F4548 and sk. 1446, F3720). Two of the males were aged 15–24, while all the other individuals were aged 25–34 at the time of their death.

Defects in the enamel can result from a wide variety of causes, including birth trauma (Seow 1991), low birth weight (Fearne *et al.* 1990), infections, and a variety of systemic illnesses (Pindborg 1982). The aetiology in any particular case in a skeletal assemblage is, therefore, impossible to determine and may very well be multifactorial. The clinical significance in these six adults is therefore open to suggestion.

15. THE PARISH RECORDS

In 1538, Thomas Cromwell ordered parish records to be kept. Every parish was to have a register into which the curate, in the presence of one or more of the church wardens, was to enter every Sunday the marriages, baptisms and burials that had taken place during the previous week. Failure to comply would result in a fine of 3s 4d which was to go towards the upkeep of the church. The injunction was not well received, and many parishes failed to comply with it. New injunctions were issued in succeeding reigns but it was not until the ecclesiastical mandates of 1597 and 1603 that more detailed regulations were laid down as to how the registers should be kept. Following these mandates, each parish was required to purchase a parchment book into which the old paper records were to be transcribed, and this book was to be kept in a coffer with three locks. Furthermore, a copy was to be sent every year to the bishop of the diocese within a month after Easter, and this was to be preserved in the Episcopal records.

During the Civil War and the Interregnum, the registers were poorly kept but were continued again after the restoration of the monarchy, although it was clear that in very many parishes the registers were deficient. To regulate matters more effectively, control of the registers was taken out of the hands of the church and put into those of the legislators. George Rose's Act of 1812[1] promulgated regulations about keeping registers, the most important consequence of which was that, from 1813, burial and baptism registers were in the form of pre-printed books which enabled more information to be recorded, including the age of death, although there were frequently inaccuracies; the date of birth was not commonly added to the register until after about 1860 (for further details see Waters 1883).

The parish registers, of course, recorded only the baptisms and burials of those who were in the communion of the Church of England and although dissenters had their own means of registration, these were often not recognized in law. It was clear that as the proportion of dissenters in the population was growing, the use of the parish registers to provide an accurate reflection of the demography of the country as a whole was becoming less and less reliable. This state of affairs was rectified by the passage of the Registration Acts of 1836, under which all births, deaths and marriages were to be registered in a public office, irrespective of the religion of those concerned.[2] The Acts came into force on 1 July 1837, and quickly superseded the parish registers as a source of reliable national statistics.

Long runs of the parish records of both St Peter's and St Mary's have survived, and it was decided to examine them in order to try to compare the demography so obtained with that derived from the examination of the skeletons.[3] The records for St Peter's start in October 1566 and finish in December 1857; those for St Mary's start in October 1570 and finish in October 1855, although there is a single baptismal entry in March 1857. There are in excess of 28,000 entries in all, with rather more at St Peter's than St Mary's, and more baptisms than burials for the period as a whole (Table 83). One hundred of the baptismal records and twenty-five of the burial records were incomplete and thus the sex of the individual could not be determined, an error rate of only 0.4%.

Until the early part of the eighteenth century, the number of burials generally exceeded the number of baptisms, but thereafter the converse was the case, the discrepancy becoming particularly noticeable during the first half of the nineteenth century (Fig. 98). There are a number of reasons for this: an increase in family size and a fall in the infant and child mortality rates are perhaps among the most important.

When the records are examined separately (Figs. 99 and 100), it can be seen that baptisms at St Mary's were almost always fewer in number than at St Peter's, but that burials at St Mary's consistently exceeded those at St Peter's until the eighteenth century, and thereafter were almost always fewer.

Table 83: Total number of entries in the Barton parish records

	Baptisms			*Burials*		
	St Peter's	*St Mary's*	*Total*	*St Peter's*	*St Mary's*	*Total*
Male	4,081	3,359	7,440	3,636	3,196	6,832
Female	3,822	3,229	7,051	3,576	3,208	6,784
Total	7,918	6,598	14,516	7,272	6,444	13,716

Note that for 60 of the baptisms at St Peter's and 40 at St Mary's there is no indication of the sex of the infant; likewise, 15 of the burial entries at St Peter's and 10 at St Mary's give no indication of the sex of the individual

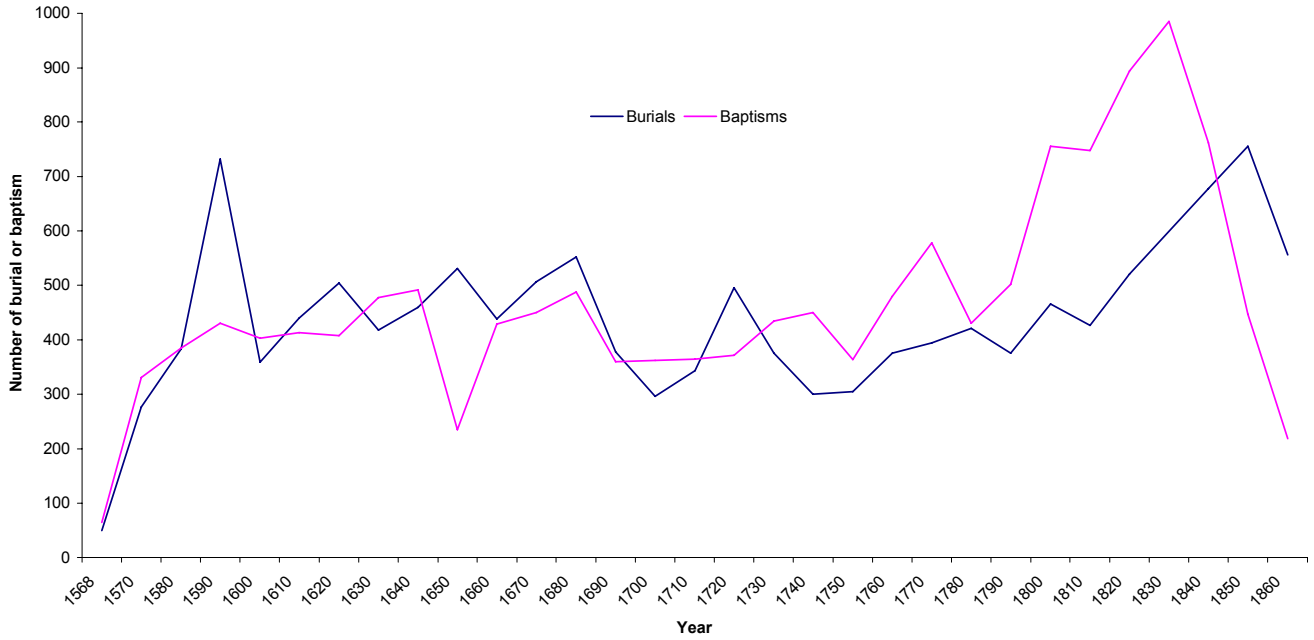

Fig. 98: Number of burials and baptisms recorded in the Barton parish registers (St Peter's and St Mary's combined)

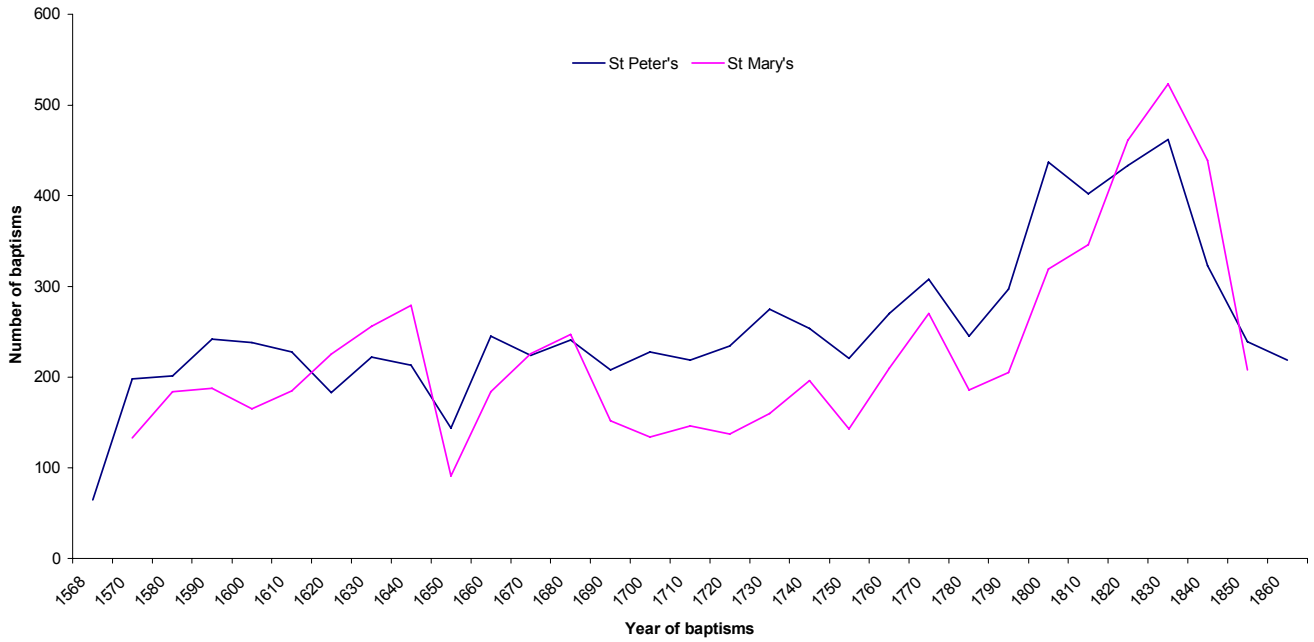

Fig. 99: Number of baptisms recorded in the Barton parish records for each church. Note the drop in numbers in the early part of the seventeenth century during the period of the Protectorate

The Plague in Barton in 1593

The most striking feature of the burials shown in Fig. 98 is the great increase in the 1590s. This is entirely due to deaths during the crisis year of 1593 during which 274 individuals were buried, compared with an average of 52 in the previous years. In the latter part of the sixteenth century the plague was in the north of England, affecting the counties of Northumberland, Westmorland, Cumberland, Durham and Yorkshire, and in 1593 it came to Barton.

When the year began, there was little indication of what was to follow, with sixteen burials in January and seventeen in February, a slight increase on the previous years, but March heralded the ensuing onslaught. In that month there were 39 burials in the town, twice as many in St Mary's as in St Peter's for some reason. The records for April and May show that the epidemic was then at its peak with 77 burials taking place in April and 69 in May, in both months more at St Peter's than at St Mary's, the reverse of the earlier trend. By June, however, the epidemic seems to have been on the wane

Fig. 100: Number of burials recorded in Barton for each church

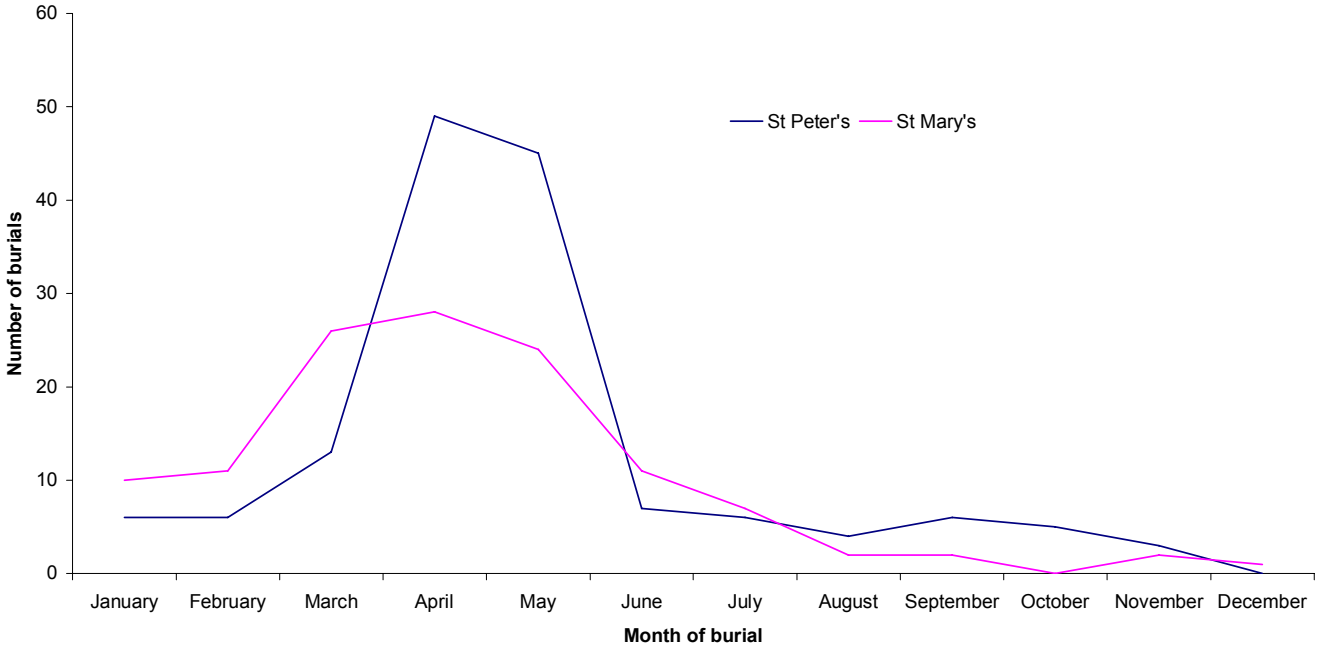

Fig. 101: Number of burials recorded in the Barton for each month during the plague epidemic in 1593

and the number of burials was returning to pre-plague levels (Fig. 101). For the rest of the year the number of burials was small – no more than eight in any month – which suggests that a proportion of those who died in the plague were the old and infirm, whose death had been brought about prematurely by the plague.

There is no indication of how the plague was brought to Barton during this crisis year. Barton was a significant port in those days and the plague may have been brought in by a ship carrying the infection on board, ports being always vulnerable to the spread of an infection from such a source. In November 1590 there is a marginal note in the St Mary's register to the effect that:

> *A stranger being master of a shippe chanced to dy hear and was buried.*

The fact that the death of this master mariner was of sufficient interest to warrant a note in the parish records, however, suggests that it was a rare event, and there is little doubt that had a ship come to port with

the plague on board, some of the crew would have died and their deaths too would almost certainly have been noted in the parish records. That this did not happen may be taken as evidence against the infection coming from the sea and in all probability it spread from one of the other infective foci in the north, either in Lincolnshire – Barton's own county – or, perhaps, from further afield.

Sex-ratio of those affected

Nowadays, although all ages and both sexes are susceptible to the plague (Butler 1989), most cases occur in males and in young people (Boisier *et al.* 2002). The historical data relating to English epidemics, however, do not show such a clear distinction. In their classic study of the 1603 plague in the London parish of St Botolph's without Bishopsgate, the Hollingsworths found that the number of deaths in males outnumbered that in females (the ratio was 1.29:1) and that the death rate was increased in all age-groups under 25 (Hollingsworth and Hollingsworth 1971). The Hollingsworths explained the sex difference in terms of social habits. Women, they suggested, were less liable to be bitten by infected fleas because of their greater cleanliness of person and dress, and because they did not go near the places where rats were to be found as frequently as men.

St Botolph's was a poor parish and at the well-to-do central London parish of St Olave, Old Jewry, more females than males died in the Great Plague of 1665: 35 compared with 18, respectively (Herlan 1980). At Barton, more females than males were affected – in the ratio of 1.2:1 (Fig. 102) – but other historical data show that the picture is far from consistent. Thus at Penrith in Cumberland, which was struck by the plague between 1597 and 1598, there was also an excess of females among the dead (Scott *et al.* 1996). On the other hand, at Colyton in Devon, where the plague was present in 1645–46 (Schofield 1977), and at Eyam in Derbyshire, in 1665–66 (Bradley 1977), the sexes were affected in approximately equal numbers.

Such historical evidence as there is from parish records in England, therefore, does not support the view that the epidemiology of the plague – certainly so far as the sex-ratio of those affected is concerned – has remained constant over the course of several centuries. This is not surprising, and indeed it would be more remarkable if there *were* a consistent pattern, given the many factors that must influence the outcome: nutritional status, personal and general hygiene, the ability to move away from infected areas, and the existence of concomitant diseases must all to some extent determine those who did or did not become infected. One modern explanation for the susceptibility of young males is that females are more likely to be iron-deficient than males. The causative organism of the plague, *Yersinia pestis*, requires a plentiful supply of iron in order to replicate and while tissues that are relatively depleted in iron may not prevent colonisation, they may not permit the bacterium to multiply sufficiently rapidly to produce an overwhelming infection (Ell 1984).

Iron deficiency may have been important in determining who developed the plague at Barton, and this would have been more likely to be common among the poor. Poverty, which is likely to be a marker for both poor nutritional status and poor hygiene, was a significant factor in Bristol during the plague epidemics that

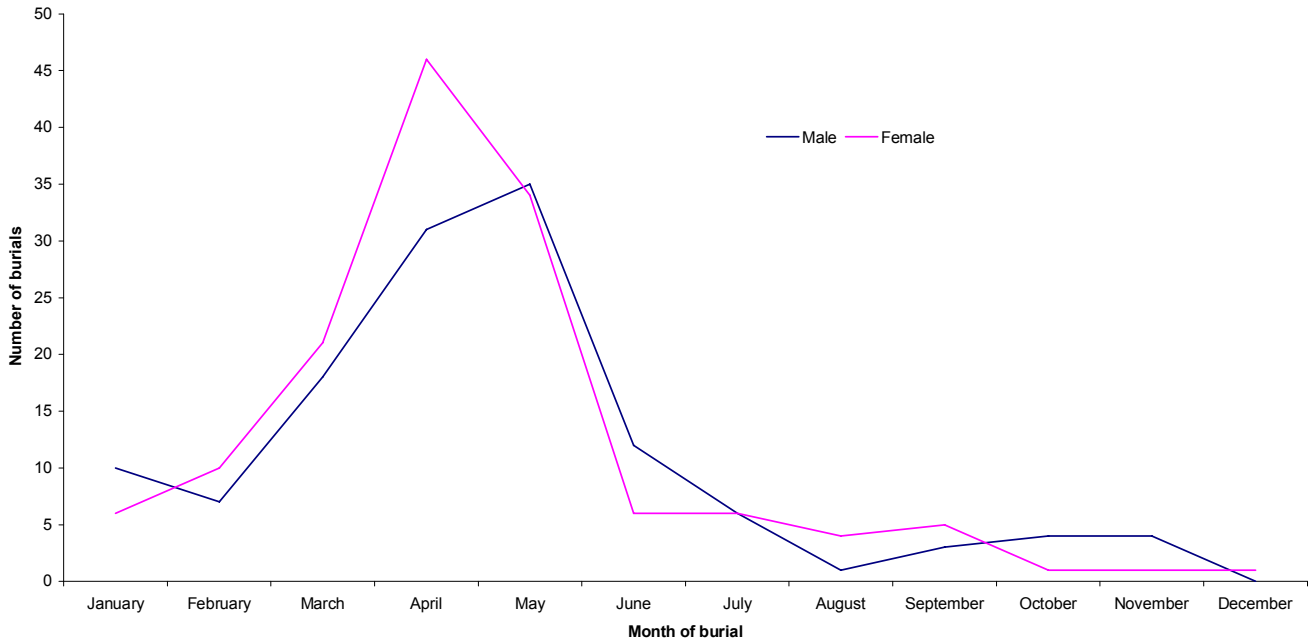

Fig. 102: Number of male and female burials recorded in the Barton parish records for each month during the plague epidemic of 1593

occurred there during 1540–1650, for example. The poor parishes tended to be more badly affected than the rich parishes in the city, the trend increasing during the latter part of the period studied (Slack 1977).

In addition to any environmental or personal factors that might have determined who died at Barton, there are likely to be other factors peculiar to the bacterium itself that affect its virulence, while the dynamic interaction between the rat, flea and human populations will contribute to the variable expression of the disease. Even the modern epidemiologist finds it difficult to control – much less to know – all the variables that might determine the outcome of an exposure to an infectious agent, and the historical epidemiologist is not likely to be better placed than his modern counterpart. To paraphrase Oscar Wilde, we may perhaps say that biology is never pure and rarely simple, and we should not expect simple relationships to be the norm.

The effect of the plague on the population of Barton

In the four months that the plague was at its most virulent, 203 people were buried in Barton but given that the parish records do not provide a record of the number of deaths in the town but the number of burials within the parish, this is bound to be a slightly conservative estimate of the total number of deaths. As mentioned in Chapter 3, the population of Barton in 1563 was estimated to be 1,040, and it seems reasonable to assume that, 30 years later, it would be little more. During 1593, 274 individuals were buried, approximately 26% of the population. Of these, 185 died during March to May when the epidemic was at its most severe. Given that the mean number of burials in the years 1566–92, inclusive, was 52, the plague accounted for 222 excess deaths in this small town, about a fifth of all those inhabiting it.

At the end of the plague year, 1593, the clerk of St Peter's added a marginal note to the parish records:

> *Hoc anno et circa hoc anni temps fuit mag. Pestis et pestilensis gravis et contagio hor ingrassans oppid.*
> During this year, a major and contagious plague and pestilence appeared among us.

There can be no doubt that the effect on the town was devastating and there can have been few families that were not involved. Of the 148 families that lost relatives in the plague (81 at St Peter's and 67 at St Mary's), 105 lost at least one member and a few lost up to ten. Reading the list of the dead belonging to those families who lost most members (Table 84) reminds one strongly of the First World War memorials in the small towns and villages in England, where the dismal roll shows that the male generations of some families were almost completely wiped out. From Table 84 we can see how the plague struck at the four families who lost most members during March and May. The Johnsons lost four people early in March and another in early April. They may then have thought the worst was past, only to have another John and Isabell succumb in the middle of May. The Kings, on the other hand, all died at the end of March or the beginning of April, while the Woods lost no fewer than seven of their number in April and Agnes was the last of all to die in May. For the Crowthers (or Crowders), the effects of the epidemic were signalled by the death of Elizabeth in early April; no more deaths occurred during the rest of the month but then six further members of the family all perished during the early days of May. Jane Pyborn died at the end of April and then followed a brief respite before there was a ghastly loss of life towards the end of May, when no fewer than eight of the family died. The fear that was engendered by the disease, the awful waiting to see whether or not more of your kinsmen were to die, the alternating relief at

Table 84: The death roll of some families during the plague months of 1593 at Barton

March	Elizabeth Johnson
	John Johnson
	Raulph Johnson
	William Johnson
	An King
	Alice King
	Jennett King
	Robert King
	John King
April	Elizabeth Crowder
	Mary King
	Elizabeth King
	John Johnson
	Agnes Wood
	Isabell Wood
	Joane Wood
	John Wood
	Elizabeth Wood
	Elizabeth Wood
	William Wood
	Jane Pyborn
May	Mathew Crowder
	Edward Crowder
	William Crowther
	Nicolas Crowther
	Margret Crowther
	John Crowther
	John Johnson
	Isabell Johnson
	William Pyborn
	George Pyborn
	Jennet Pyborn
	William Pyborn
	John Pyborn
	Cecily Pyborn
	Robert Pyborn
	Margret Pyborn
	Agnes Woode

apparently being spared, and the terror when one after another died, can only be imagined.

Ten years after the plague, the population of Barton was still 10% lower than before the plague struck, and it would be many years before it recovered completely from that awful visitation.

Comparison with Skeletal Data

The main aim of studying the parish records was to see to what extent they validated the demographic data obtained from the skeletal examination. It was hoped to obtain age at death from the parish records, but it was not possible to do this in sufficient numbers for analysis until the age of death was routinely recorded in the new register books introduced in 1813. The sex of those buried throughout the period, however, were determined for virtually all the records, as noted above.

Sex-ratios

The sex-ratio (male-to-female) for the whole period covered by the parish records at St Peter's was 1.02, at St Mary's 0.99, and for the two 'parishes' combined, 1.01; that is to say, as near to unity as could reasonably be expected. For comparison with the skeletal data, the sex-ratio from the parish records was determined for the three archaeological phases that overlapped the parish records, Phases A, A/B and C: the results are shown in Table 85. The skeletal analysis differs from the parish records most notably in Phase B, where there is apparently a considerable male excess. The reason for this is almost certainly due to the fact that very few skeletons from this phase were sexed, only 24 males and 19 females, and the resulting ratio is, therefore, not very reliable. The difference between the results from Phase A/B may also be due to the relatively small number of skeletons assigned a sex (42 males and 45 females); where the numbers are more substantial – in Phase A – there is extremely good agreement between the two sets of data.

Table 85: Male-to-female ratios (M:F) compared between parish records and skeletal analysis

Phase	*Parish records*	*Skeletal analysis*
A (1700–1850)	1.03	1.02
A/B (1500–1850)	1.04	0.93
B (1500–1699)	1.07	1.26

Age at death

The age of death obtained from the parish records after 1813 could only properly be compared with the skeletal data from Phase A and the results of this comparison are shown in Fig. 103. There are considerable differences between the two sets of data, most notably the excess of infants and the deficits of the elderly, in the skeletal data. There is also a considerable excess of deaths in the 15–24 year age group in the skeletons and a lack of individuals in the 35–44 year age group.

It is difficult to explain why there are so many more infants in the skeletal record than in the parish records, since skeletons from young children are among the easiest to recognize and age accurately, and the likelihood of substantial error would not seem to be great. Many of the difficulties experienced by those who examine skeletal remains are related to problems of preservation

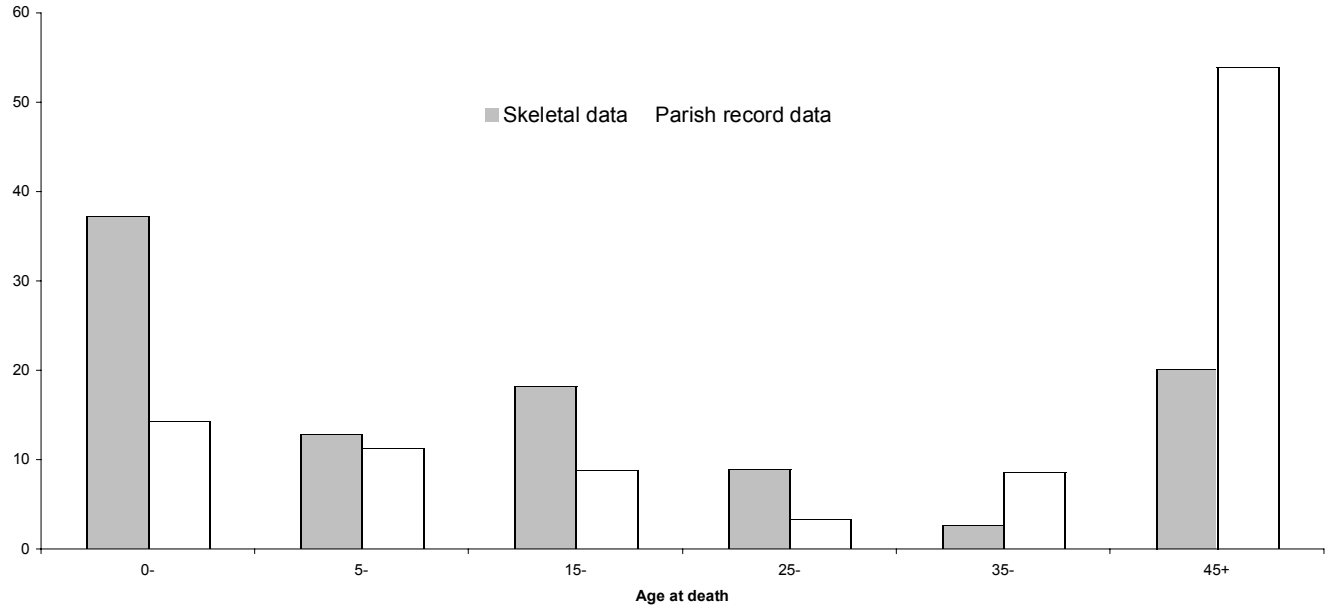

Fig. 103: Comparison of distribution of ages at death at Barton derived from parish records (both churches) and skeletal data

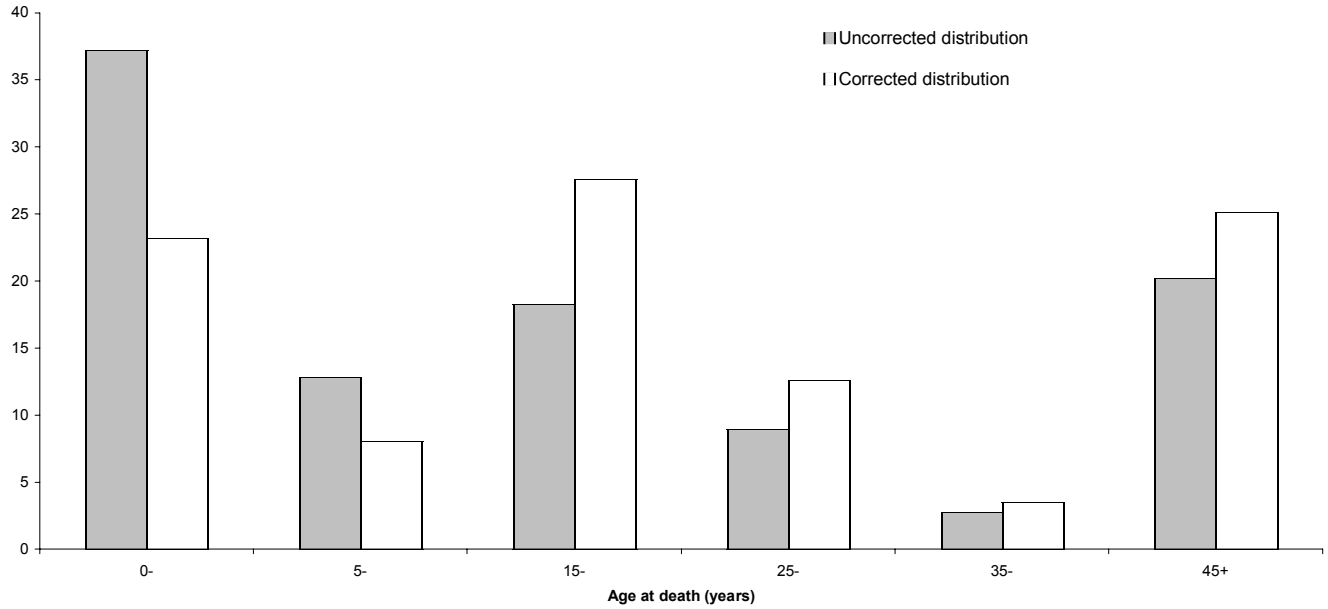

Fig. 104: Comparison of distribution of ages at death at Barton derived from uncorrected and corrected skeletal data. See text for description of correction procedure

and the received wisdom is that the skeletons of young children survive less well than those of adults; these data suggest that perhaps at Barton the opposite was true and that the skeletons of the young survived *better* than those of older individuals. One also has to remember that a substantial number of the adult skeletons could be neither aged nor sexed, and so do not appear in the demographic analysis. Virtually all children's skeletons *will* be counted, however, since although they cannot be given a sex, it is almost always possible to determine the age-group (0–4, or 5–14) to which they belong; there is thus an inbuilt bias toward exaggerating the proportion of infants and children in an assemblage, unless the preservation of the adult skeletons is particularly good.

From the comparison it seems probable that many of the adult (15+) skeletons were under-aged and that many properly belong in older age-groups; this seems particularly the case in the 15–24, and the 25–34 year age-groups. The parish records show, as one would expect, that many people survived into old age; over half those who were buried were aged over 45 and there were 22 nonagenarians, 10 males and 12 females. The oldest, Teresa Stead, was 99 when she died. Even allowing for the possibility that the relatives of very elderly individuals may have exaggerated their age at death, there seems to be little doubt that reliance on a skeletal assemblage will substantially under-estimate the age at death of many of the adults. This is by no means a novel observation, but simply confirms what others have found and shows how necessary it is to find more accurate methods of determining age at death from the skeleton.[4]

A slightly improved age distribution can be obtained if the skeletons that are neither aged nor sexed are redistributed to the various age and sex classes to get a 'corrected' distribution using a technique that is described elsewhere (Waldron 2001). When this is done it can be seen from Fig. 104 that the corrected distribution now has a smaller proportion of infants and juveniles, and a higher proportion of individuals in the oldest age group. The proportions in the younger age-groups are also increased, however, and this means that errors in ageing will tend to be exaggerated.

Expectation of life

The data from the parish records were used to construct life tables for both 'parishes' combined, and the results are shown in Table 86. There was little difference between either the males or the females; females had to wait until much later before they enjoyed a longer expectation of life than their male contemporaries. The men living in the 'parish' of St Peter's apparently enjoyed a slightly longer life than those in St

Table 86: Expectation of life (years) at Barton, based on parish records

		St Peter's	*St Mary's*	*Total*
Male	e_0	44.6	39.8	43.0
	e_5	50.6	48.1	49.8
Female	e_0	43.3	43.4	43.3
	e_5	46.5	46.3	46.4

Table 87: Comparison of expectation of life (years) at Barton, derived from skeletal and parish record data

	Phase A skeletons	*Parish records*
e_0	23.3	43.9
e_5	29.7	48.6

Mary's, but this seems to have been almost entirely the result of a lower infant mortality rate.

The comparison between life expectancy based on the skeletons from phase A and the parish records is shown in Table 87. At birth (e_0), life expectancy estimated from the skeletons is twenty years less than that estimated from the parish records, and this difference is maintained at the age of five. These differences are, of course, entirely explained by the data in Fig. 103, where the greatest proportion of skeletons seems to be in the 0–4 year age group, whereas in the parish records, the greatest proportion is in the 45+ year age group. What is clear from this comparison is the construction of life tables from ages derived from skeletal assemblages is likely to give very dubious results and we side with those who would like the method quietly put to rest (Bocquet-Appel and Masset 1982; for a rebuttal see van Gervan and Armelagos 1983).

16. IN CONCLUSION

The strongest impression to be gained from the study of the human remains from St Peter's church is one of stability. Whether one considers the physical characteristics, or the pattern of disease, there seems to have been almost no change worthy of note over the 900-year period. The mean height of the population has not varied; the expectation of life seems to have improved (as estimated at age 20) only in the last – post-1700 – phase; and the prevalence or character of very few diseases has altered. The most notable changes affected osteoarthritis; OA of the hands tends to affect more joints in the post-medieval period, and there is a three-fold increase in OA of the elbow and hip, which might indicate a change in behaviour or activity in the later period. Behavioural changes might also account for the considerable increase in trauma suffered by males in the later period.

Judging from the skeletal evidence, however, it is apparent that for a long period Barton provided a stable environment in which to live; the population were seemingly well – or at least adequately – nourished, and their toll of disease was unremarkable.

When making these judgements, some important caveats have to be entered, however, apart from those that are inherent in any examination of the skeleton and which are the bane of any epidemiological study of past populations; in these I include indifferent preservation, difficulties in ageing and sexing which hinder the accurate estimation of age and sex-specific prevalence rates, and – most particularly – estimates of life expectancy. And how wonderful it would be, to be able to get to grips with still-birth and infant mortality rates! And even with what is by any reckoning a very substantial assemblage – larger than any so far reported on in this country – the winnowing processes of poor preservation, phasing, and sorting into age and sex classes, inevitably results in having to deal with small numbers and large confidence intervals, which tend to obscure any real differences that might have been present in the population during life.

Important as these limitations on the study are, one has also to remember two other very significant factors. First – and if this point seems obvious, it is sometimes conveniently forgotten – an examination of the skeleton will provide evidence only of those diseases that affect it. The great bulk of human morbidity and mortality is due to diseases of the soft tissues. Failure to bring this fact to a conscious level has sometimes led to the statement that a population represented by its skeletons was 'healthy', despite all being dead – some at very young ages – and with nothing known of the conditions that may have affected heart, lungs, liver and kidneys. Unfortunately, the study of a skeletal assemblage will permit only a very meagre description of general morbidity; how disease of the skeleton is manifested during life is – in most cases – beyond our comprehension, and no conclusions can be drawn about the level of morbidity in the population as a whole except to say that many were likely to have been impeded by and in pain from osteoarthritis, and that substantially more would have been liable to intermittent toothache.

The second matter for consideration is that events are averaged out over the long time bases that are imposed by the phasing. Typically, the period that an assemblage relates to is measured in centuries; exceptions do occur, of course – for example, plague pits and battle cemeteries – and a very lucky investigator may even know the date and time of death of those whose remains he or she is examining.[1] The long time base means that short-term fluctuations are obscured. Who can doubt that the population of Barton was affected by the twin catastrophes of the fourteenth century, the great famine and the Black Death? Yet their occurrence is undetectable, as is the most calamitous event for which we have objective historical evidence, the outbreak of plague in 1593, when about a fifth of the town died. This event has left no shadow on the human remains and would have gone unnoticed but for the parish records. That should give all bone specialists pause for thought before they claim more than their evidence permits.

The study of the remains of our long-dead ancestors is a privilege that, despite the caveats above, permits the closest link with those who lived, aspired, loved and died so long ago, and offers the most immediate insight into their society. The fullest possible account, however, will always depend upon the integration and interpretation of all available evidence, skeletal, archaeological, historical, biochemical and artistic. The present report provides the evidence that may allow other scholars to venture further and understand more.

APPENDIX 1

Publications Incorporating Information Derived from Barton

The human bones from Barton are recognised as a research resource of international importance and have been studied by a number of scholars associated with the project. Papers which make significant use of information derived from their study are listed below. For full publication details see the bibliography at the end of this volume.

Barber 1994
Barber, Shepstone and Rogers 1995
Barber, Shepstone and Rogers 1997
Crossley, Levy and Rogers 1996
Dieppe and Rogers 1989
Dieppe and Rogers 1992
Goode, Waldron and Rogers 1993
Hacking, Allen and Rogers 1994
Lim, Rogers, Shepstone and Dieppe 1995
Norman, Rogers and Dieppe 1994
Parish, McNally and Rogers 1996
Rogers 1981
Rogers 1982
Rogers 1988
Rogers 1996a
Rogers 1997
Rogers and Dieppe 1992
Rogers and Dieppe 1993a
Rogers and Dieppe 1993b
Rogers and Dieppe 1994
Rogers, Lim and Shepstone 1995
Rogers, Lim, Shepstone and Turnquist 1996
Rogers, Shepstone and Dieppe 1997
Rogers and Waldron 1986
Rogers and Waldron 1988b
Rogers and Waldron 1995
Rogers, Waldron, Dieppe and Watt 1987
Rogers, Waldron and Watt 1991
Rogers, Watt and Dieppe 1995
Rogers and Young 1994
Rogers, Young and Dieppe 1993
Sharma, Rogers and Buckland-Wright 1996
Shepstone, Rogers, Kirwan and Silverman 1997a
Shepstone, Rogers, Kirwan and Silverman 1997b
Waldron, Rogers and Watt 1994
Wiggins 1996
Wiggins and Rogers 1995

Appendix 2: Excavated Burials from St Peter's Church

Key to abbreviations used

Archaeology

BH xx	radiocarbon dating sample reference
coffin	substantive evidence for a coffin
?coffin	circumstantial evidence for a coffin
roves	presence of diamond roves (also known as clench bolts)
(T)	preserved timber
dendro	timber which has a tree-ring date
(dendro)	timber submitted for tree-ring dating but not (yet) dated
sk.	skeleton

Phase

0 = a burial that could not be phased.

Pathology

btum	benign tumour
caries	dental caries
co	cribra orbitalia
dentabs	dental abscess
dish	diffuse idiopathic skeletal hyperostosis
disloc	dislocation
ea	erosive arthropathy
eh	enamel hypoplasia
eoa	erosive osteoarthritis
fibdys	fibrous dysplasia
hallvag	hallux valgus
head wnd	head wound
hfi	hyperostosis frontalis interna
maligt	malignant tumour
oa	osteoarthritis
om	osteomyelitis
op	osteoporosis
osdiss	osteochondritis dissecans
pdb	Paget's disease of bone
pnb	periosteal new bone (not specified)
pnb rib	periosteal new bone on rib
psa	psoriatic arthropathy
ra	rheumatoid arthritis
sepa	septic arthropathy
sn	Schmorl's nodes
spond	spondylolysis
stt	soft tissue trauma
tb	tuberculosis

Age ranges used (years)

Infant	0–4
Juvenile	5–15

For adults, the best that can usually be done is to provide a range into which the age at death probably falls (see p. 35). The exception is for the named sample when the precise age is given.

NB: In the list that follows, some skeleton numbers refer to burials from which no bone was suitable for analysis (*i.e.* phase totals derived from this table will not match those in the text).
This list does not contain the empty/exhumed graves.

Sk. no.	*Context*	*Phase*	*Sex*	*Age*	*Pathology notes*	*Archaeology notes*	*Fig. no.*
1	66	A	Female	Adult	dish; oa	coffin	36
2	67	A	Male	17		coffin	36
3	82	A	Male	45+	caries; oa	coffin	36
4	84	A	Female	Adult	maligt; om	?coffin	36
5	85	A	Unknown	Adult	osdiss	coffin	36
6	86	A	Juvenile	12		?coffin	36
7	90	C	Female	45+	eoa; op	med. floor tiles in fill	33, 53
8	95	A	Female	Adult	oa; osdiss	coffin	36
9	97	A	Unknown	25–34	caries		36
10	98	A	Female	Adult	caries; hfi; oa	coffin	36, 44
11	87	A	?Female	Adult	caries; dentabs	?coffin	36
12	99	A	Unknown	Adult		?coffin	36
13	96	A	?Male	Adult	trauma	coffin	36
14	640	B	Male	25–34	caries; osdiss; trauma	?coffin	34
15	88	A	Unknown	Adult	caries; pnb	?coffin	36

Sk. no.	*Context*	*Phase*	*Sex*	*Age*	*Pathology notes*	*Archaeology notes*	*Fig. no.*
16	104	D/E	Male	22	sn	coffin	32
17	108	D/E				No skeleton was lifted	32
18	206	C	Female	45+	dentabs; hallvag; oa	coffin	33
19	210	C	Male	Adult	oa		33
20	901	A/B	Unknown	Adult		coffin	36
21	906	A	Unknown	Adult			36
22	912	A	Male	25–34		coffin	36
23	248	A	Infant	0			36
24	244	A				coffin, body dissolved before lifting	36
25	921	A	Unknown	Adult			36
26	922	A	Male	17		coffin	36
27	908	A	Juvenile	8	caries	coffin	36
28	229	A	Male	45+	dish; oa; pnb; sn; trauma	coffin	36
29	966	B	Female	Adult	spond		34
30	716	E	Juvenile	6		BH 22	30
31	941	A	Male	Adult	oa	coffin	36
32	272	A	Infant	0		coffin	36
33	273	A	Infant	0		coffin	36
34	391	A	Infant	1	co	coffin	36
35	900	A	Male	20	caries	coffin	36
36	286	B	Infant	0		coffin	34
37	907	A	Male	20	sn	coffin	36
38	287	B	Male	Adult		?coffin	34
39	288	A/B	Unknown	17	caries	?coffin, pin, textile	36
40	979	A/B	?Female	Adult			36
41	3576	B	Female	15		?coffin	not on plan
42	982	A	Male	Adult	oa; trauma	?coffin	36
43	985	B	Juvenile	6		?coffin	34
44	910	A/B	?Male	Adult	oa	?coffin	36
45	984	A	Male	Adult		coffin	36
46	983	A	Unknown	Adult	oa	coffin	36
47	973	A	Female	Adult		Margaret Roberts; coffin, wood & cloth, coin	36
48	3577	B	Male	Adult			not on plan
49	3578	B	Male	35–44	pdb; pnb		not on plan
50	988	A	Male	Adult			36
51	299	B	Unknown	Adult	oa		34
52	986	B	Female	25–34	caries		34
53	322	B	Male	Adult	trauma		34
54	992	A	Female	Adult		coffin	36
55	994	A	Male	45+	caries		36
56	997	B	Female	Adult			34
57	997	B	?Male	Adult	om	?coffin	34
58	998	A	Infant	1			36
59	990	B	Unknown	Adult			34
60	990	B	Unknown	Adult			34
61	990	B	Unknown	Adult			34
62	990	B	Juvenile	Adult			34
63	999	B/C	Infant	2			34
64	338	B	Unknown	Adult	eoa	coffin, coin	34
65	339	B	Female	Adult			34
66	345	A	Female	25–34	oa; op; trauma	coffin	36, 74
67	339	B	Male	35–44			34
68	346	B	Unknown	Adult			34
69	1001	B	Female	Adult	caries; oa; op; spond; trauma		34
70	347	A	Female	45+	oa; op; osdiss; pnb	coffin	36, 51
71	348	A	Male	Adult			36
72	349	A	Male	45+	caries; oa; trauma		36
73	3579	A/B	?Male	Adult	oa		not on plan
74	1005	B	Male	Adult			34
75	1002	B	Unknown	Adult			34
76	1003	C	Infant	1			33
77	350	B	Unknown	Adult			34
78	1006	B	Unknown	Adult			34
79	351	A	Male	25–34	caries		36
80	3580	A/B	Unknown	Adult			not on plan
81	1008	B	Unknown	Adult	trauma		34, 75
82	1009	B	Unknown	Adult			34
84	7953	C	Male	Adult	pnb		33
85	7954	C	Male	25–34	rickets; pnb		33
86	7955	C	Male	25–34	caries		33
87	1011	B	?Male	Adult	pnb		34
89	352	A	Female	Adult	hfi; oa	coffin	36

90	360	B	?Female	Adult			34
91	237	A/B	Female	25–34	caries; trauma	coffin	36
92	1022	B	Unknown	Adult	trauma		34
93	1021	B	Male	25–34	caries; oa		34
94	1020	B	Juvenile	15			34
95	934	B	Male	45+	bent		34
96	1019	B	Male	Adult	oa; osdiss		34
97	?279		Male	45+		coffin – mahogany	not on plan
98	361	B	Female	17	caries	coffin	34
99	1023	C	Male	Adult			33
100	1024	C	Unknown	Adult			33
101	1025	C	Male	Adult			33
102	1026	C	Unknown	Adult	pnb		33
103	1015	B	Female	25–34	pnb		34
104	363	A	Male	45+			36
105	364	A	Female	Adult	co; dentabs		36
106	7971	A	Juvenile	13		coffin	36
107	365	A	Male	45+	caries; dentabs; spond	coffin, dinner plate, coins (2)	25, 36
108	274	A	Infant	0		coffin	36
109	392	B	Male	45+	oa; trauma	coffin	34
110	393	B	Female	Adult			34
111	394	B	Unknown	Adult			34
112	395	C	Female	Adult			33
113	395	C	Female	Adult			33
114	368	A/B	Male	45+	caries; dentabs; hfi; sn; trauma	coffin, incised floor tile	36
115	7957	A	Infant	0			36
116	366	B	?Male	25–34	caries	?coffin, 9" tile beneath knees	34
117	405	B	Female	Adult	gout; oa		34
118	293	A	Infant	0		coffin	36
119	366	B	Female	25–34	dentabs		34
120	409	C	Female	Adult	caries	coin	33
121	409	C	Unknown	Adult			33
122	410	C	Female	25–34	caries; dentabs; gout; oa		33
123	419	A	Infant	1	pnb	coffin	36
124	417	A	Infant	1		coffin	36
125	367	B	Infant	0			34
126	1104	D	Male	45+			32
127	416	C	?Female	45+	caries; dentabs; oa; op; pnb; trauma		33
128	425	C	Male	45+	trauma	shroud, textile	33
129	367	B	Female	Adult	pnb		34
130	357	B	Male	Adult		coffin, tiles laid over fill	34
131	356	B	Female	17		coffin	34
132	344	C	Male	35–44	dish; pdb		33
133	1103	A	Female	Adult	oa; op	coffin	36
134	430	C	Female	Adult		coffin	33
135	431	A	Female	Adult		coffin	36
136	354	B	Male	Adult	caries; dentabs; trauma	coffin, tiles laid over fill	34
137	415	C	?Female	Adult		coffin	33
138	432	A	Female	Adult	caries	coffin	36
139	434	B/C	Female	Adult			34
140	433	B	Male	25–34			34
141	433	B	Unknown	Adult			34
142	437	A	Female	20		same as sk. 628	36
143	23	A	Male	45+	dish; oa; trauma	coffin	36
144	55	A	Female	Adult	bent; rickets; oa; trauma	coffin	36
145	59	A	Male	45+	trauma	?coffin	36
146	1093	C	Male	25–34	caries; dentabs; oa; trauma	coffin	33
147	1128	B	Unknown	16	rickets		34
148	1131	B	Male	Adult	caries; trauma		34
149	1133	B	Juvenile	5		coffin	34
150	1135	A	Female	25–34		coffin	36
151	1137	A				coffin with textiles, bone too decayed to lift	36
152	3294	A	Female	20		coffin	36
153	3023	A	Male	45+	oa; osdiss; trauma	coffin	36
154	3295	A	Female	Adult			36
155	3296	A	?Male	Adult	co; oa	coffin	36
156	3022	A	Female	45+	hfi; om	coffin, coin	36
157	3297	A	Male	17	caries		36
158	3298	A/B	Female	Adult			36
159	3025	A	Female	Adult	caries	coffin	10, 36
160	1141	B	Male	17			34
161	1140	B	Female	17		coffin	34
162	1142	B	Unknown	15		?coffin	34
163	1140	B	Infant	0			34

Sk. no.	*Context*	*Phase*	*Sex*	*Age*	*Pathology notes*	*Archaeology notes*	*Fig. no.*
164	448	C	Female	23–34	oa		33
165	1147	B	Male	35–44	caries; dentabs; trauma	coffin	34
166	3024	A	Unknown	Adult		coffin	10, 36
167	3299	A/B	Juvenile	5		?coffin	36
168	3300	A	Female	25–34		?coffin	36
169	3301	A	Male	Adult		coffin	36
169	7958	0	Male	Adult			not on plan
170	3026	A	Male	45+	oa; sepa	coffin	36
171	3302	A	?Male	Adult		coffin	36
172	3303	A	Male	45+	caries	?coffin	36
173	3304	A/B	Female	Adult			36
174	3027	A	Female	Adult	oa; trauma	coffin, bowl below base board	36
175	1149	A	Female	20	caries; dentabs	coffin	36
176	1150	A	Female	Adult	caries; oa; pnb	coffin	36
177	446	C	Male	25–34	caries	coffin	33
178	1152	B	Female	Adult	dish; oa	coffin	34
179	305	C	Male	45+	caries; dentabs; head wnd; sn		33, 76
180	1153	A	Juvenile	11		coffin	36
181	3305	A/B	Female	Adult			36
182	3031	A	?Male	Adult	eh; pnb	coffin	36
183	3306	A/B	Unknown	Adult			36
184	3030	A	Male	25–34	oa	coffin	36
185	3307	A/B	Male	35–44	sn	?coffin	36
186	1158	D	Male	Adult	co; dentabs; oa; sn		32
187	1154	D	Female	45+		?coffin, bone die	32
188	452	C	Juvenile	5			33
189	452	C	Infant	0	pnb		33
190	3308	A/B	?Female	Adult	kyphosis; trauma	?coffin	36
191	3309	A	Male	45+	trauma	coffin	36
192	3038	A	Female	Adult		coffin, AE ring	36
193	3310	A	Male	Adult		?coffin	36
194	1157	A	Male	25–34		coffin	36
195	1159	C	Juvenile	9		coffin	not on plan
196	456	B	Male	45+			34
197	1162	A	Male	Adult	trauma		36
198	325	C	Female	20		coffin, 'cloth of gold'	33
199	1163	A	Female	Adult	oa	coffin	36
200	457	C	Infant	0			33
201	458	C	Male	25–34	caries		33
202	1164	D	Infant	0			32
203	1164	D	Infant	0			32
204	1164	D	Female	45+	caries; oa; trauma	coffin, lime over skull & thorax	23, 32
205	449	C	Female	17	caries	AE crucifix & finger-ring	33
206	460	C	Unknown	Adult			33
207	3311	B/C	Unknown	Adult		?coffin	34
208	3312	B/C	Male	35–44		?coffin	34
209	3055	A	Juvenile	13–15	co; eh; maligt		36, 93
210	3313	A	Male	Adult	sn	coffin, ?med fretwork	36
211	3314	B/C	Infant	4			34
212	3315	A/B	Male	Adult	pnb	?coffin	36
213	463	A	Male	17	trauma	coffin & textile, coin	36
214	464	A	Male	Adult		coffin & textile	36
215	465	A	Male	45+	oa; osdiss		36
216	3581	B	Male	22	caries; sn		not on plan
217	3316	A/B	Female	Adult	oa; trauma	?coffin	36
218	3317	A/B	Male	20	sn		36
219	3033	A	Male	45+	caries; oa; trauma	coffin, wooden stake, ?autopsy	29, 36
220	3318	B/C	Male	30		?coffin	34
221	3319	B/C	Unknown	25–34	caries	?coffin	34
222	3039	A	Female	Adult		coffin	36
223	3320	A/B	?Female	Adult		?coffin	36
224	3321	A	Female	Adult		?coffin	36
225	3322	A/B	Unknown	16		?coffin	36
226	467	A	Juvenile	Adult			36
227	468	A	Juvenile	14			36
228	1186	D	Male	25–34	dentabs; trauma	chalice & paten, ?fe obj.	13, 32
229	1178	B	Unknown	Adult	pnb	coffin	34
230	1177	D	Juvenile	10	sepa; trauma		32
231	7959	C	Infant	0		?coffin	not on plan
232	466	A	Male	Adult		coffin	36
233	1184	D	Male	25–34			32
234	470	C	Female	45+	oa; osdiss	glazed tile	33
235	1190	D	Female	25–34	co	tile & pot	32

236	1191	D	Male	Adult	trauma		32
237	1189	D	Infant	1			32
238	1188	D	Unknown	Adult			32
239	1187	D	Infant	1			32
240	476	D	Juvenile	14			32
241	487	C	Male	45+	dentabs	AE stud	33
242	489	C	Female	25–34	caries; oa	coffin	33
243	3323	A/B	Male	Adult		coffin	36
244	3324	B/C	Female	25–34	caries	?coffin	34
245	3325	A/B	Unknown	Adult		?coffin	36
246	3326	A/B	Male	Adult		?coffin	36
247	3327	A/B	Male	Adult		?coffin	36
248	3328	A/B	Male	Adult		?coffin	36
249	3329	A/B	Female	25–34	caries; eh; oa	?coffin	36
250	3330	A/B	Juvenile	Adult		?coffin	36
251	3331	B/C	Juvenile	13			34
252	3332	A	Male	25–34	oa		36
253	3333	B/C	Male	Adult	trauma	?coffin	34
254	3334	A/B	Juvenile	13		?coffin	36
255	3335	A/B	?Female	Adult	sn	?coffin	36
256	3336	A	Male	45+	maligt; oa; trauma	coffin	36
257	3337	A	Male	25–34		?coffin, truss	36
258	3041	A	Male	Adult	caries; dentabs	coffin, truss	26–27, 36
259	3043	A	Male	Adult		coffin, buttons, textile	36
260	3044	A	Juvenile	5	caries; co	coffin	36
261	3338	B/C	Male	25–34		?coffin	34
262	3339	A/B	Infant	4			36
263	3040	A/B	Juvenile	5	co	?coffin	36
264	3340	A/B	Unknown	Adult			36
265	3045	A	Female	35–44	caries	coffin, finger-ring	36
266	490	C	Male	45+	oa; pnb		33
267	1203	D				bones did not survive washing	32
268	1206	D	Female	19	caries	coffin	32
269	1205	D	Male	45+	caries; dentabs; oa		32
270	497	C	Female	Adult			33
271	3341	B/C	Male	25–34	caries; eh; sn		34
272	3342	C	Male	35–44	caries; dentabs; dish; eh	BH 44	33
274	8001	A–D	Unknown	Adult		?coffin	not on plan
275	3343	A/B	Male	Adult		?coffin	36
276	1207	D	Female	Adult			32
277	1207	D	Male	Adult	pnb		32
278	1501	A	Female	Adult	oa; trauma	coffin, leather coffin lining	36
279	1291	C	Unknown	Adult			33
280	1210	D	Unknown	Adult	caries; dentabs; gout; trauma	grave end shaped for head	32
281	3344	B/C	Female	15			34
282	3345	B/C	Infant	3		?coffin	34
283	3346	B/C	Unknown	16			34
284	1214	D	Infant	0			32
285	1150	A	Female	20			36
286	3347	B/C	Unknown	Adult		?coffin	34
287	3348	B/C	Unknown	Adult		?coffin	34
288	3349	B/C	Juvenile	15		?coffin	34
289	3350	A/B	Unknown	Adult		?coffin	36
290	3351	A/B	Unknown	Adult		?coffin	36
291	3352	B/C	Unknown	Adult		?coffin	34
292	3353	A/B	Female	Adult	kyphosis; oa	?coffin	36
293	3354	B/C	Unknown	Adult		?coffin	34
294	3355	B/C	Unknown	Adult		?coffin	34
295	3356	A/B	Unknown	Adult		?coffin	36
296	1217	C	?Male	Adult			33
297	1218	D	Unknown	Adult			32
298	3357	B/C	Juvenile	5		?coffin	34
299	3358	B/C	Male	39	caries	?coffin	34
300	3359	B/C	Juvenile	5		?coffin	34
301	3049	B/C	Unknown	Adult	oa	coffin	34
302	3360	B/C	Infant	1			34
303	3361	A	Female	17		coffin	36
304	3056	A	Male	45+	caries; dish; oa	coffin	36
305	7960	A	Male	Adult			36
306	7961	A	Male	Adult			36
307	7962	A	Male	Adult	om		36
308	1221	C	Male	45+	caries		33
309	1223	D	Female	45+	caries; oa; osdiss; pdb		32
310	3362	C/D	Juvenile	14	trauma	?coffin	33

Sk. no.	*Context*	*Phase*	*Sex*	*Age*	*Pathology notes*	*Archaeology notes*	*Fig. no.*
311	3367	B/C	Male	35–44	oa	?coffin	34
312	3363	B/C	Juvenile	7		?coffin	34
313	3364	A	Unknown	Adult	caries	coffin	36
314	3365	A/B	Female	Adult	sn	?coffin	36
315	3366	B/C	Unknown	Adult		?coffin	34
316	3368	B/C	Juvenile	12		?coffin	34
317	1227	D	Male	25–34	caries		32
318	1506	D	Juvenile	11	caries; co		32
319	1236	D	Female	Adult	caries		32
320	1235	D	Female	Adult			32
321	1237	D	Juvenile	Adult			32
322	1238	D/E	Unknown	Adult			32
323	1239	A/B	Male	25–34	caries; oa; sn		not on plan
324	1234	D	Infant	Neonate			32
325	1241	E	Unknown	Adult			30
326	1240	D	Female	45+	caries; dentabs; pnb		32
327	3050	B/C	Male	35–44	caries; dentabs; trauma		34
328	3369	B/C	Male	Adult	caries; dentabs; spond; trauma	?coffin	34
329	3370	B/C	Male	Adult	op; sepa; stt	?coffin	34, 81
330	3371	A/B	Unknown	Adult			36
331	3372	B/C	Juvenile	Adult			34
332	3373	C/D	Juvenile	15			33
333	3374	B/C	Female	25–34	caries; co; dentabs; oa	?shroud, BH 45	34
334	3375	C/D	Infant	3			33
335	3376	A/B	Male	Adult		?coffin	36
336	3377	A/B	?Female	17	caries; dentabs; eh	?coffin	36
337	3378	A/B	Female	17	caries; eh	?coffin	36
338	1536	D	Infant	1			32
339	1524	C	Juvenile	15	caries; dentabs	interred with 340	12, 33
340	1524	C	Juvenile	12	caries	interred with 339	12, 33
341	1242	D	Unknown	Adult			32
342	1244	C	Female	Adult	oa; ra	coffin	33, 57
343	1538	A	Unknown	Adult			36
344	1527	B	Infant	0			34
345	1243	D	Male	Adult	psa		32, 59–61
346	1245	D	Juvenile	Adult			32
347	3054	A	Female	25–34	dentabs; rickets	coffin	36
348	3053	A	Female	25–34	caries	coffin, lace end	36
349	3057	A	Male	50+	pnb; oa; trauma	coffin	36
350	3379	A	Female	Adult		?coffin	36
351	3380	B/C	Unknown	Adult		?coffin	34
352	3381	B/C	Unknown	Adult		?coffin	34
353	3382	B/C	Unknown	Adult		?coffin	34
354	3383	B/C	?Male	Adult		?coffin	34
355	3384	B/C				?coffin, not lifted	34
356	3385	B/C	Juvenile	9		?coffin	34
357	3386	B/C	Juvenile	6		?coffin	34
358	1542	C	Male	Adult			33
359	1527	B	Unknown	Adult			34
360	1507	D	Juvenile	6			32
361	1527	B	Juvenile	5			not on plan
362	1250	D	Male	25–34	head wnd; pnb	coffin	32, 77
363	1252	D	Male	45+	co; oa	coffin	32
364	1251	D	Unknown	Adult			32
365	1253	D	Infant	1			32
366	1544	D	Juvenile	10			32
367	1545	A	Female	Adult	oa	coffin, textile	36
368	1546	B/C	Unknown	Adult	oa; sn		34
369	1547	A	Male	Adult	sn	coffin, traces of leather lining	36
370	1548	A	Male	Adult		coffin	36
371	1556	D	Infant	Neonate			32
372	1278	A	Male	45+	oa	coffin	36
373	1526	C	Female	Adult			33
374	1554	D				Arm only, not saved	32
375	1555	C	Unknown	Adult			33
376	1266	D	?Male	25–34			32
377	1267	D	?Female	Adult	trauma		32
378	1558	D	Juvenile	13	disloc; sn		32
379	1269	D	Female	25–34	caries		32
380	1551	D	Unknown	45+	caries; co; oa; trauma		32
381	1281	A	Infant	3			36
382	3387	B/C	Juvenile	8			34
383	3388	B/C	Infant	4	co	?coffin	34

384	3060	A/B	?Male	Adult		AE lace end	36
385	3389	B/C	Infant	1		?coffin	34
386	3390	B/C	Juvenile	10		?coffin	34
387	3391	B/C	Male	Adult		?coffin	34
388	3392	B/C	Female	Adult		?coffin	34
389	3393	B/C	Juvenile	6		?coffin	34
390	3394	B/C	Infant	3		?coffin	34
391	3395	B/C	Juvenile	5		?coffin	34
392	3059	B/C	Infant	0			34
393	3396	B/C	Juvenile	6		?coffin	34
394	3066	A/B	Female	Adult			36
395	3397	B/C	Infant	1		?coffin	34
396	3398	B/C	Infant	3		?coffin	34
397	3399	B/C	Infant	0		?coffin	34
398	3400	C/D	Juvenile	8		?coffin	33
399	3401	B/C	Male	Adult	oa; trauma	?coffin	34
400	3402	C/D	Unknown	45+	disloc	?coffin	33
401	1559	B	Juvenile	8	caries	coffin	34
402	3061	A/B	Unknown	16			36
403	3062	A	Female	Adult	oa	coffin	36
404	3063	A	Male	45+	oa	coffin, ring	36
405	3064	B/C	Female	Adult	oa	with sk. 411	34
406	3058	A	Female	Adult	dentabs	coffin	36
407	3065	A	Male	45+	caries; dentabs; eh	coffin	36
408	3403	B/C	Infant	0			34
409	3078	B/C	Juvenile	9	pnb	buckle	34
410	3404	A/B	Unknown	Adult		?coffin	36
411	3064	B/C	Fetus	7 mths		with sk. 405	34
412	3405	A/B	Unknown	Adult			36
413	3406	B/C	Unknown	No age			34
414	3407	B/C	Infant	0			34
415	3408	B/C	Female	45+	bent; pdb		34
416	3409	B/C	Male	Adult	oa	?coffin	34
417	3410	C/D	Juvenile	8		?coffin	33
418	3411	C/D	Juvenile	Adult		?coffin	33
419	3070	C/D	Female	Adult			33
420	3412	B/C	Female	Adult		?coffin	34
421	1566	D	Infant	1		textile	32
422	1563	D	Infant	2			32
423	1276	D/E	?Male	25–34	caries; dentabs; oa		32
424	1277	D	Female	Adult	caries; co	?charred board over body, ?coffin	32
425	1564	D/E	Male	45+	caries; eh; oa	coffin	32
426	1565	A	Unknown	45+	co; oa; trauma	coffin	36
427	1280	D	Male	35–44	bent; caries; dentabs; sn		32
428	1283	D/E	?Female	45+	caries		32
429	1285	D	Juvenile	10			32
430	1286	D	Juvenile	5		?charred board over body	32
431	1575	C	?Female	45+	oa		33
432	1575	C	Infant	1			33
433	1576	D/E	Infant	3			32
434	1289	E	Female	25–34	caries; dentabs		30
435	3413	B/C	Juvenile	9		?coffin	34
436	3069	A/B	Male	50+	dish; oa; sn; spond		36
437	3414	C/D	Juvenile	6		?coffin	33
438	3415	B/C	Female	25–34	caries; dentabs	?coffin	34
439	3416	B/C	Male	Adult	sepa	?coffin	34
440	3417	A	?Female	Adult	oa	coffin	36
441	3074	A	Unknown	17	caries		36
442	3418	B/C	?Female	Adult		?coffin	34
443	3419	B/C	?Female	Adult		coffin	34
444	3075	A	Male	Adult	oa	coffin	36
445	3420	A	Male	17	om	?coffin	36
446	1290	C	Male	45+	pdb; oa; trauma	coffin	33
447	1294	D	Infant	3	co		32
448	1295	D	Infant	1		?coffin	32
449	1296	D	Infant	4			32
450	1296	D	Infant	0			32
451	1299	A	Male	50+		coffin	36
452	3421	C/D	Infant	4			33
453	3422	A/B	Female	Adult			36
454	3423	B/C	Unknown	20		?coffin	34
455	3424	C/D	Juvenile	10		?coffin	33
456	3425	C/D	Male	45+	co; oa	?coffin	33
457	3426	C/D	Infant	3		?coffin	33

Sk. no.	*Context*	*Phase*	*Sex*	*Age*	*Pathology notes*	*Archaeology notes*	*Fig. no.*
458	3427	C/D	Female	Adult	op; oa; trauma	?coffin	33
459	3428	B/C	Unknown	Adult		?coffin	34
460	3429	C/D	Unknown	Adult		?coffin	33
461	1301	D	Infant	1			32
462	3430	B/C	Unknown	Adult		?coffin	34
463	3431	B/C	Male	Adult		?coffin	34
464	3432	C/D	Infant	4		?coffin	33
465	3433	C/D	Juvenile	8		?coffin	33
466	3434	B/C	Unknown	Adult		?coffin	34
467	3435	B/C	Female	25–34	caries	?coffin	34
468	3436	B/C	Male	Adult		?coffin	34
469	3437	A/B	Female	20		coffin	36
470	3438	A/B	Juvenile	Adult		?coffin	36
471	3439	A/B	Female	Adult		?coffin	36
472	3440	A/B	Male	Adult		?coffin	36
473	3441	A/B	Infant	0			36
474	3442	A/B	Female	Adult		coffin	36
475	3443	B/C	Juvenile	7		?coffin	34
476	3444	A/B	Juvenile	Adult		coffin	36
477	3445	A/B	Unknown	Adult		?coffin	36
478	3446	A/B	Unknown	Adult		?coffin	36
479	3447	B/C	Male	Adult		?coffin	34
480	3448	B/C	Female	17	caries	?coffin	34
481	3449	B/C	Female	Adult	oa	coffin	34
482	3450	B/C	?Female	Adult		?coffin	34
483	1303	D	Juvenile	5		coffin	32
484	1582	C	Male	Adult	sn		33
485	3451	B/C	Unknown	15	pnb	?coffin	34
486	3452	A/B	Infant	0		?coffin	36
487	3076	A	Female	Adult	caries; dentabs; oa	coffin	36
488	3453	B/C	?Male	50+		?coffin	34
489	3454	B/C	Infant	1			34
490	3455	C/D	Juvenile	13		?coffin	33
491	3456	C/D	?Female	15		?coffin	33
492	3457	C/D	Female	Adult		?coffin	33
493	8000	A–D	Infant	No age			not on plan
494	3458	B/C	Infant	4	co		34
495	1604	E	Infant	1	co		30
496	3459	B/C	Female	15			34
497	3460	B/C	Fetus	6 mths		?coffin	34
498	3461	B/C	Female	Adult	oa	?coffin	34
499	3462	C/D	Female	45+	gout; oa; op	?coffin	33
500	3463	B/C	Juvenile	14			34
501	3464	B/C				did not survive lifting	34
502	3465	B/C	Unknown	Adult			34
503	3466	A/B	Female	Adult		?coffin	36
504	3467	B/C	Male	Adult		?coffin	34
505	3468	B/C	Unknown	Adult			34
506	3469	A/B	Unknown	Adult			36
508	1312	D	Infant	0		coffin	32
509	1600	E	Infant	Neonate		coffin	30
510	1605	E	Juvenile	5			30
511	1314	D	Infant	0			32
512	3470	C/D	Female	25–34		?coffin	33
513	3471	C/D	Unknown	Adult	oa; op; trauma		33
514	3472	C/D	Juvenile	10	co	?coffin	33
515	3473	C/D	Infant	2	co		33
516	3474	C/D	Unknown	Adult			33
517	3475	B/C	Infant	1			34
518	3476	C/D	Infant	3			33
519	3477	B/C	Unknown	Adult			34
520	3478	C/D	Unknown	Adult			33
521	3479	C/D	Male	Adult		?coffin	33
522	1606	D/E	Infant	1	co	coffin	30
523	1607	D	Juvenile	8			32
524	1608	C	Male	25–34	sn		33
525	1609	E	Fetus	6 mths			30
526	1610	E	Fetus	8 mths			30
527	3480	C/D	Infant	0			33
528	3481	B/C	Male	Adult	oa; osdiss; trauma		34
529	3482	C/D	Unknown	Adult	pnb; stt; trauma		33
530	3483	C/D	Infant	3			33
531	3484	B/C				not lifted	34

532	3485	C/D	Unknown	Adult			33
533	3486	C/D	Male	Adult			33
534	3487	C/D	Juvenile	9		?coffin	33
535	3583	0	?Male	Adult		coffin	not on plan
536	3488	C/D	Male	25–34	caries	?coffin	33
537	4096	E	Male	Adult	oa	coffin, BH 23	30
538	3489	D/E	?Female	25–34	caries	?coffin	32
539	3490	D	?Female	45+	oa	BH 85	32
3491	540	C/D	Juvenile	7	co	?coffin	33
541	4097	E	Male	25–34	caries; dentabs; oa	coffin	30
542	3528	D/E	Infant	1			30
543	4098	A	Female	17	caries	coffin	36
544	4099	D/E	Unknown	25–34			32
545	3527	E	Juvenile	14		coffin, roves	30
546	4102	E	Female	17		coffin, ear muffs (2)	30
547	25	A	Female	35–44	caries	coffin	36
548	1320	D	Female	Adult			32
549	1323	C	Female	18	caries; co; sn	coffin	33
550	1326	D	Female	45+	caries	?coffin	32
551	1620	E	Infant	No age			30
552	1589	D/E	Female	45+	caries; dentabs; oa; sn		32
553	1332	D	Female	25–34	caries; dentabs; oa	coffin	32
554	1621	E	Male	25–34	caries	coffin, ear muffs (2), waterlogged	18, 30
555	1335	B	Unknown	Adult	oa	part of sk. 564	34
556	1336	B/C	Juvenile	15			34
557	1337	B	Unknown	Adult			34
558	1617	E	Female	45+			E
559	1340	A	?Male	45+	caries; oa	coffin	36
560	1627	E	Infant	Neonate			30
561	1627	E	Infant	0			30
562	1343	E	Male	45+	caries	charred lid, pillow stones (2)	30
563	1344	C	Unknown	Adult			33
564	1335	B				part of sk. 555	34
565	1615	A	Female	17	caries; co; dentabs; eh; kyphosis		36
566	1326	D	Infant	Neonate		?coffin	32
567	1320	D	Infant	0			32
568	1347	D/E	Female	25–34			32
569	1349	E	Male	Adult	oa	coffin	30
570	1632	E	Male	Adult		coffin	30
571	1635	D/E	Male	Adult	caries	?coffin	32
572	1636	D/E	?Male	25–34		?coffin	32
573	1637	D/E	Unknown	Adult		?coffin	32
574	1354	D	Infant	0			32
575	1638	D/E	Male	21			32
576	1357	E	Male	Adult			30
577	1359	D/E	Infant	3			30
578	1639	D/E	Female	45+	trauma		32
579	1646	E	Female	Adult	osdiss	coffin, ear muff (1)	30
580	1647	E	Female	20	co; pnb	coffin, pillow (1)	30
582	1648	D	Male	45+	caries; dentabs; sn		32
583	1360	E	Unknown	Adult			30
584	1627	E	Female	25–34	caries; dentabs; sn		30
585	1631	E	Male	45+	caries; maligt; oa	nails/roves, ?coffin	30
586	7963	0	Infant	1			not on plan
587	1361	D	Infant	0			32
588	7964	0	Male	45+			not on plan
589	1650	E	Male	Adult		coffin	30
590	1353	A	Unknown	Adult		?coffin	36
591	1363	E	Male	Adult	dish; oa		30
592	1364	E	Male	Adult		BH 24, grave cut by A–S chancel	30
593	1662	D/E	Female	Adult			32
594	1663	D/E	Male	45+	caries; sn		32
595	1367	A	Male	45+		coffin	36
596	1367	A	Female	Adult	oa; osdiss	coffin	36
597	1665	D	Male	35–44	caries; oa; sn		32
598	1644	E	Male	50+	oa; sn	coffin	30
599	1645	E	Infant	0			30
600	1642	E	?Female	Adult		coffin	30
601	3574	B	Infant	0		?coffin	34
602	3575	B	Female	16			34
603	1666	D/E	Female	Adult		coffin	32
604	1674	E	Male	20	eh	coffin	30
605	1368	D/E	Male	50+		coffin	30
606	1675	E	Female	Adult	co; sn	coffin	30

Sk. no.	*Context*	*Phase*	*Sex*	*Age*	*Pathology notes*	*Archaeology notes*	*Fig. no.*
607	1673	D	Female	45+	caries		32
608	1678	D	Female	20		?coffin	32
609	1370	D/E	Unknown	Adult			30
610	1681	D/E	Infant	3	co	coffin	32
611	1682	D/E	Unknown	Adult	caries		32
612	1379	E	Male	Adult		?coffin	30
613	1683	E	Female	45+	sn	coffin, ear muffs	30
614	1375	E	Fetus	7 mths			30
615	1684	D	Female	Adult	caries	coffin, ?head at east end	32
616	1376	E	Infant	Neonate	co		30
617	1685	B/C	Male	25–34	caries; dentabs; eh		34
618	1686	C	Juvenile	8	caries		33
619	1687	B/C	Male	45+	caries; pnb; oa; op; trauma	coffin	34
620	1688	D	Infant	0	pnb		32
621	1377	E	Infant	0			30
622	1380	E	Male	Adult		coffin	30
623	1378	E	Fetus	6 mths			30
624	1689	E	Male	45+	oa; trauma	coffin, ear muffs	30
625	1690	D	Infant	0			32
626	1696	D	Unknown	Adult		?coffin	32
627	1704	A/B	Unknown	Adult		coffin, textile	36
628	1705	A/B	Female	20			36
629	1705	A/B	Infant	0			36
630	1706	A/B	Female	Adult	oa; pnb		36
631	1702	E	Female	25–34		coffin, pillow (1)	30
632	1703	D/E	Unknown	Adult			32
633	1710	D/E	Male	45+		coffin, ear muffs (2)	32
634	1394	E	Fetus	7 mths			30
635	1395	E	Juvenile	8	co	coffin	30
636	1397	E	Infant	1		?coffin	30
637	1386	E	Unknown	Adult		coffin	15, 30
638	1400	E	Male	25–34	caries; oa	coffin	30
639	4103	E	Male	Adult	trauma	coffin	30
640	1341	A	Female	77	oa; op	Ann Latham (d.1831), lead coffin in vault, not disturbed	36
641	3492	D	Male	18	caries; dentabs	BH 84	32
642	3493	C/D	Juvenile	12			33
643	3494	B/C	Unknown	Adult		?coffin	34
644	3495	C/D	Juvenile	6			33
645	3496	C/D	Unknown	Adult			33
646	3497	B/C	Juvenile	5			34
647	3498	D/E	Juvenile	10			32
648	3499	D/E	Male	Adult	caries; oa		32
649	7714	D	Male	25–34	oa; pnb	BH 62	32
650	7715	C/D	Male	45+	oa		33
651	7716	C/D	Infant	1			33
652	7717	D/E	Female	Adult			32
653	7718	C/D	Male	Adult	oa		33
654	7719	C/D	Infant	4			33
655	7720	C/D	Juvenile	15			33
656	7721	C/D	Male	45+	pdb		33
657	7722	C/D	Male	45+	oa		33
658	7723	B/C	Infant	0			34
659	7724	C/D	Unknown	Adult	oa		33
660	7725	D/E	Male	25–34			32
661	1716	A/B	?Male	25–34	caries; oa	coffin	36
662	1413	D	Infant	0			32
663	1714	D	Male	22		coffin	32
664	1715	A	Infant	0		?coffin	36
665	1527	B	Unknown	Adult	gout		34, 63
666	1721	D	Infant	0			32
667	7726	C/D	Female	Adult			33
668	7727	C/D	Female	Adult	oa		33
669	7728	C/D	Female	20			33
670	7729	C/D	Male	Adult			33
671	3092	D	Male	25–34	caries; pnb; spond	coffin, BH 87	32
672	7730	0	Infant	0			not on plan
673	7731	B/C	Unknown	Adult			34
674	7732	C/D	Juvenile	9			33
675	7733	C/D	Female	45+	caries; dentabs; trauma		33
676	7734	C/D	Unknown	Adult			33
677	1414	D	Infant	0			32
678	1416	D	Infant	3			32

679	1732	E	Female	25–34	oa	coffin	30
680	1417	D	Juvenile	9			32
681	7735	C/D	Infant	0			33
682	7736	C/D	Infant	4			33
683	7737	C/D	Unknown	Adult			33
684	7738	C/D	Unknown	Adult	trauma		33
685	7739	C/D	Female	Adult	trauma		33
686	7740	C/D	Female	Adult			33
687	7741	C/D	Female	Adult			33
688	7742	C/D	Female	Adult	sn		33
689	7743	C/D	Juvenile	5			33
690	7744	B/C	Unknown	Adult			34
691	7745	C/D	Female	Adult			33
692	7746	B/C	Unknown	Adult			34
693	7747	C/D	Infant	0			33
694	7748	C/D	Unknown	Adult			33
695	7749	B/C	Male	15			34
696	7750	C/D	Unknown	Adult	trauma		33
697	7751	B/C	Male	20	sn		34
698	7752	C/D	Unknown	Adult	trauma		33
699	7753	C/D	Infant	1			33
700	7754	C/D	Juvenile	6	co		33
701	7755	C/D	Juvenile	8	discitis; pnb		33
702	7756	C/D	Female	Adult	pnb		33
703	7757	C/D	Unknown	Adult	oa; sn		33
704	7758	C/D	Male	15	rickets		33
705	7759	C/D	Female	20	rickets		33
706	7760	B/C	Male	35–44	trauma		34
707	7761	B/C	Juvenile	7			34
708	7762	C/D	Infant	2			33
709	7763	C/D	Unknown	Adult			33
710	7764	C/D	Male	25–34			33
711	1733	D	Female	25–34	caries; eh		32
712	1419	D/E	Juvenile	6	co		32
713	1420	D/E	Infant	0			32
714	7765	C/D	Unknown	Adult			33
715	7766	C/D	Infant	0	pnb		33
716	7767	C/D	Female	Adult	eoa		33, 55
717	1421	D	Female	45+	dentabs		32
718	7768	C/D	Unknown	Adult			33
719	7769	D/E	Unknown	Adult	om		32
720	7770	C/D	?Female	Adult			33
721	7771	C/D	Unknown	Adult			33
722	7772	C/D	Infant	No age			33
723	7773	B/C	Juvenile	5			34
724	7774	0	Unknown	Adult			not on plan
725	7775	D/E	Juvenile	8			32
726	1737	D	Unknown	Adult			32
727	1422	C	?Female	Adult			33
728	1739	D	Unknown	Adult			32
729	1429	D	Juvenile	6	pnb		32
730	7776	C/D	Female	17			33
731	7777	D/E	Female	20	caries; dentabs; pnb		32
732	7778	C/D	Male	25–34	pnb; sn	coffin	33
733	7779	C/D	Unknown	Adult			33
734	7780	C/D	Infant	1	pnb		33
735	7781	C/D	Female	Adult			33
736	7782	C/D	Infant	0			33
737	7783	C/D	Male	35–44	oa; sn	charred board	33
738	7784	C/D	Female	Adult			33
739	7785	D/E	Juvenile	8			32
740	940	A	Female	79	dish; oa; trauma	Mary Thorley (d. 1833), coffin in brick vault	36, 89
741	1433	D	Infant	4	co	coffin	32
742	1432	D	Male	17			32
743	1430	D	Unknown	Adult	pnb; trauma		32
744	1436	D	Juvenile	8			32
745	3088	B/C	Juvenile	13			34
746	7786	D/E	Unknown	Adult	pnb; trauma		32
747	7787	C/D	Unknown	Adult			33
748	3089	D	Female	45+	caries; oa	coffin, BH 88	32
749	7788	C/D	?Male	45+	caries; oa		33
750	7789	C/D	Female	Adult			33
751	1208	D	?Female	Adult			32

Sk. no.	*Context*	*Phase*	*Sex*	*Age*	*Pathology notes*	*Archaeology notes*	*Fig. no.*
752	3088	B/C	Infant	0			34
753	7790	C/D	Juvenile	6			33
754	7791	C/D	Female	25–34	co		33
755	7792	C/D	Female	25–34			33
756	7793	D/E	Infant	2			32
757	7794	D/E	Infant	2			32
758	7795	D/E	Unknown	Adult	discitis; oa; pnb; trauma		32
759	1438	D	Male	Adult	stt		32
760	1439	D/E	Unknown	Adult			32
761	1440	A	Female	13		coffin, Papal Bulla	36
762	7796	C/D	Unknown	Adult			33
763	7926	D/E	Male	45+	pdb		32
764	7798	C/D	Female	Adult	pnb; oa; trauma		33
765	7799	D/E	Male	45+	co; oa	BH63	32
766	7800	D/E	Female	17	eh		32
767	7801	D	Male	25–34	caries; sn	coffin	32
768	7802	D/E	Female	25–34		charred board, 3 rivets	32
769	7803	C/D	Female	Adult		?coffin	33
770	7804	C/D	Juvenile	6			33
771	7805	C/D	?Female	45+	caries; dentabs; oa; trauma		33
772	7806	C/D	Male	Adult	dish; pnb; oa; trauma		33, 88
773	7807	D/E	Unknown	Adult			32
774	7808	C/D	Juvenile	5	pnb rib; trauma		33
775	7809	C/D	Unknown	Adult	sepa		33
776	1753	E	Female	25–34		coffin(T), BH 09, dendro	19–21, 30
777	1444	D	Male	25–34		?coffin	32
778	1443	E	Male	35–44	dish		30
779	1446	D	Juvenile	5	co; discitis; pnb	?coffin	32
780	1447	D	Infant	0	pnb		32
781	1448	D/E	Infant	2			32
782	1449	D	Infant	2	co	?coffin	32
783	1452	B	Male	50+		coffin, lead disc	34
784	7810	C/D	Juvenile	5			33
785	7811	C/D	?Female	15			33
786	7812	C/D	Female	25–34	oa		33
787	3094	A/B	Female	Adult		?coffin	36
788	7813	D/E	Female	45+	caries; hallvag; oa; op	?coffin	32
789	7814	B/C	Unknown	Adult	rickets		34
790	7815	A	Unknown	Adult			36
791	1450	D	Juvenile	7			32
792	1453	D	Infant	1			32
793	1765	E	Female	Adult		coffin	30
794	1454	E	Male	45+	caries	?coffin	30
796	1455	D/E	Female	Adult		coffin	32
797	1754	E	Female	45+	caries	coffin	30
798	7816	D/E	Infant	2			32
799	7817	C/D	Infant	4	co		33
800	3096	D	Male	45+	caries; trauma; head wnd		33, 73
801	7818	D/E	Unknown	Adult			32
802	7819	D/E	?Female	Adult	trauma		32
803	7820	D/E	?Female	Adult			32
804	7821	B/C	?Female	Adult			34
805	7822	C/D	Unknown	Adult			33
806	7823	B/C	Infant	3			34
807	7824	B/C	Unknown	Adult			34
808	7825	A/B	Juvenile	Adult			36
809	7826	C/D	Female	Adult	oa; spond	BH 66	33
810	7827	C/D	Juvenile	12			33
811	7828	C/D	Female	Adult	trauma		33
812	7829	D/E	Unknown	Adult		?coffin	32
813	7830	C/D	Female	17	trauma		33
814	7831	C/D	Unknown	Adult			33
815	7832	A	Male	Adult		?coffin	36
816	7833	C/D	Unknown	Adult		?coffin	33
817	7834	C/D	Male	Adult	oa; ra	?coffin	33
818	7835	C/D	Juvenile	5			33
819	7836	B/C	Female	Adult	caries; dentabs	coffin	34
820	3099	B/C	Infant	1		coffin	34
821	1459	D/E	Infant	No age			32
822	1462	E	Unknown	Adult		fe object, ?buckle	30
823	1463	D/E	Juvenile	5			32
824	7837	B/C	Male	20	sn		34
825	3102	C/D	Male	17	pnb		33

826	3102	C/D	Juvenile	15	caries; eh		33
827	7838	D/E	Unknown	Adult	oa		32
828	7839	C/D	Juvenile	Adult			33
829	7840	C/D	Female	Adult	trauma		33
830	7841	D/E	Juvenile	7			32
832	1466	D/E	Unknown	Adult			32
833	1769	E	Male	45+	dentabs; eh; oa; pnb	coffin	30
834	1467	D	Male	35–44	sn		32
835	1469	B	Female	15	caries	?coffin	34
836	1468	D	Male	Adult		?coffin	32
837	1470	D	Unknown	Adult	oa		32
838	1770	D/E	Infant	3		coffin	32
839	1473	D/E	Juvenile	6	co	?coffin	32
840	1471	D/E	Female	Adult			32
841	1775	E	Male	45+		coffin	30
842	1774	E	Female	Adult		roves, BH 25	30
843	1474	E	Male	Adult		coffin	30
844	1472	B	Infant	3			34
845	1776	E	Infant	2		coffin, ear muffs (2)	30
846	7842	B/C	?Male	Adult			34
847	7843	C/D	Infant	0			33
848	7844	D/E	Female	25–34			32
849	7845	D/E	Female	45+	caries		32
850	3105	D	Female	45+	caries; oa; stt	?coffin	32
851	3104	D/E	Male	50+	caries; dentabs; oa; trauma		32
852	3103	D/E	Infant	0			32
853	7846	C/D	Female	15	co; rickets		33
854	7847	D/E	Female	45+	caries; eh		32
855	7848	D/E	Juvenile	8			32
856	7849	D/E	Juvenile	10			32
857	7850	C/D	Juvenile	6			33
858	7851	C/D	Female	25–34	caries; eh		33
859	7852	B/C	Male	25–34	caries; trauma	?coffin	34
861	1481	E	Infant	No age			30
862	1477	D/E	Female	45+	caries; hfi	coffin	32
863	1476	A	Infant	No age		coffin	36
864	1478	D/E	Unknown	20	trauma		32
865	1479	E	Male	45+			30
866	1480	D	Infant	1			32
867	1483	D/E	Male	45+	caries; dentabs; discitis; oa; sn; trauma	coffin	32
868	1484	E	Unknown	Adult			30
869	1485	E	Female	Adult		?coffin/charred board	30
870	1464	E	Male	45+	caries	coffin, stones (7 around body)	30
871	1771	D/E	Female	25–34	oa	coffin	32
872	1487	D/E	Unknown	Adult			32
873	7853	C/D	Juvenile	10			33
874	7854	C/D	Unknown	Adult			33
876	3106	C/D/E	Infant	1	pnb		not on plan
877	7855	C/D	Female	17	spond		33
878	7856	C/D	Male	25–34			33
879	7857	C/D	Infant	4			33
880	7858	C/D	?Male	Adult			33
881	7859	C/D	Female	Adult	pnb		33
882	7860	C/D	Juvenile	5	pnb		33
883	7861	B/C	Unknown	Adult			34
884	7862	B/C	?Male	Adult			34
885	7863	C/D	Female	16	co	BH 67	33
886	7864	A/B	Unknown	Adult			36
887	7865	A/B	Unknown	Adult	oa		36
888	7866	0	Infant	3			not on plan
889	7867	B/C/D	Unknown	Adult			
890	7868	D/E	Male	Adult			32
891	1486	E	Juvenile	5			30
892	1490	D/E	Female	20	caries	coffin	32
893	1784	E	Unknown	Adult	ea; pnb		30
894	1492	E	Unknown	45+		coffin	30
895	1494	D/E	Infant	3	co		32
896	1491	E	Female	Adult	om		30
897	1493	E	Unknown	Adult			30
898	7869	B/C	?Male	Adult	rickets; pnb; sn; trauma	BH 86	34
899	7870	B/C	Female	Adult	oa		34, 56
900	7871	A/B	Male	25–34			36
901	7872	B/C	Unknown	Adult			34

Sk. no.	*Context*	*Phase*	*Sex*	*Age*	*Pathology notes*	*Archaeology notes*	*Fig. no.*
902	7873	B/C	?Female	Adult			34
903	7874	A/B	Female	Adult			36
904	3109	A/B	Unknown	Adult			36
905	3110	A/B	Unknown	Adult			36
906	3111	A/B	Infant	4	trauma	coffin	36
907	7928	A/B	Unknown	Adult			36
908	7929	A/B	Male	22	pnb; trauma		36
909	7932	A/B	Male	Adult		coffin	36
910	7930	A/B	Juvenile	5	rickets; pnb		36
911	3112	A	Male	45+	caries; dentabs; oa; trauma	coffin	36
912	7931	B/C	Unknown	Adult			34
913	7933	B/C	?Male	15			34
914	3113	A	Male	Adult		coffin	36
915	3117	D/E	Infant	0		?charred board	32
916	1495	E	Unknown	Adult			30
917	1785	E	Unknown	17		?coffin	30
918	1498	E	Male	45+		coffin	30
919	1497	E	Infant	1			30
920	1786	E	Male	Adult		coffin	30
921	1787	E	Male	25–34		roves	30
922	1791	E	?Female	Adult	oa	coffin(T), ?roves, dendro	30
923	1691	E	Female	Adult		coffin	30
924	4005	D/E	Female	25–34		coffin	32
925	4007	E	Infant	1		coffin	30
926	4006	E	Juvenile	6			30
927	1792	E	Juvenile	8		coffin	30
928	4008	D/E	Unknown	Adult			32
929	1333	E	Unknown	Adult		coffin	30
930	4012	E	Juvenile	Adult		coffin	30
931	7934	A/B	Female	Adult			36
932	7935	A/B	Female	Adult			36
933	3123	B/C	Male	25–34	caries	coffin	34
934	7936	B/C	Juvenile	13			34
935	3121	A	Female	Adult	co	coffin	36
936	3122	A	Unknown	Adult		?coffin	36
937	7941	B/C	Juvenile	15			34
938	7937	B/C	Unknown	Adult			34
939	7938	B/C	Juvenile	6			34
940	7942	B/C	Female	Adult	oa		34
941	1794	E	Male	25–34		coffin(T)	30
942	4014	E	Female	25–34	caries	?coffin	30
943	4015	D/E	?Male	Adult	sn; spond		32
944	3114	E	Juvenile	6		timber	30
945	3114	E	Female	25–34	caries	coffin, rove (1– ?stray)	30
946	3119	D/E	Juvenile	10		coffin	32
947	3118	D/E	Male	45+		coffin	32
948	3120	D/E	Female	45+	caries; oa	coffin	32
949	7875	C/D	Female	25–34			33
950	7876	C/D	Male	35–44	trauma		33
951	7877	B/C	Female	Adult	caries		34
952	7878	B/C	Unknown	Adult			34
953	7879	C/D	Infant	0			33
954	4017	E	Infant	1			30
955	1796	D/E	Infant	3		coffin	32
956	4018	E	Juvenile	5			30
957	4019	E	?Male	25–34		BH 72, coffin & clay filling	22, 30
958	4001	E	Unknown	Adult		BH 26	30
959	4020	E	Infant	1			30
960	7880	D	Unknown	Adult	oa; pnb		32
961	7881	D/E	Juvenile	13			32
962	7882	C/D	Unknown	Adult			33
963	7883	D/E	Juvenile	6			32
964	7884	D/E	Infant	2			32
965	7885	D/E	Female	45+	caries		32
966	7886	C/D	Male	25–34	caries	coffin	33
967	7887	D/E	Male	25–34	eh; trauma		32
968	7888	D/E	Male	17			32
969	7889	D/E	Unknown	Adult			32
970	7890	D/E	Female	Adult			32
971	3124	D/E	Female	45+	caries; oa	large cobble on chest	32
972	7891	D/E	Male	Adult			32
973	7892	D/E	?Female	25–34	oa		32
974	7893	C/D	Female	Adult	oa; trauma		33

975	7894	C/D	Male	45+	caries; dentabs		33
976	4021	E	Male	45+	hfi; maligt; spond		30, 92
977	4022	E	?Female	Adult	oa		30
978	4023	E	Unknown	Adult		?coffin	30
979	7895	C/D	Infant	0			33
980	7896	D/E	Female	Adult			32
981	7897	D/E	Male	45+			32
982	7898	D/E	Female	Adult			32
983	7899	D/E	Juvenile	6			32
984	7900	D/E	Infant	2			32
985	7901	D/E	Infant	4			32
986	1797	E	?Male	Adult	oa	coffin, BH 27	30
987	1798	D/E	Juvenile	5	co		32
988	4030	E	Female	Adult		coffin	30
989	4027	E	Male	45+	dentabs; oa; trauma		30
990	4028	E	Female	45+	caries		30
991	4029	D/E	Unknown	Adult		coffin	32
992	4034	E	Juvenile	13			30
993	4033	E	Male	25–34		?coffin	30
994	1772	D/E	Female	20	pnb	coffin	32
995	4035	E	Female	25–34	co	coffin, ear muffs (2), ?Saxon pot	30
996	4036	E	Female	25–34	caries; oa	coffin	30
997	3126	C/D	Male	25–34	sn	coffin	33
998	7943	B/C	Unknown	Adult			34
999	3130	D/E	Male	Adult			32
1000	7944	A/B	Female	Adult	oa; pnb	BH 65	36
1001	7945	A/B	Female	Adult			36
1002	7946	A/B	Unknown	20	pnb		36
1003	7939	A/B	Unknown	17			36
1004	3127	A	?Female	Adult	pnb	coffin	36
1005	7947	A/B	Female	Adult		coffin	36
1006	3128	C/D	Male	22	sn	buckle	33
1007	7940	B/C	Female	Adult			34
1008	7948	C/D	Unknown	Adult			33
1009	3125	A	Female	Adult	oa	coffin	36
1010	7949	B/C	Juvenile	12	om		34
1011	4038	E	?Female	25–34	caries	?coffin	30
1012	3504	D/E	Unknown	15		?coffin	32
1013	3505	D/E	Unknown	Adult		?coffin	30
1014	4041	A	Female	45+	caries; oa; trauma		36
1015	4040	E	?Male	35–44	caries; sepa; trauma	coffin, clay filled, BH 73	30
1016	4042	E	Juvenile	7	co	?coffin	30
1017	3500	E	Male	Adult		roves	30
1018	3501	E	Female	20		coffin(T)	30
1019	1431	D/E	Male	Adult	oa		32
1020	4046	E	Male	45+	pnb	coffin	30
1021	7902	C/D	Infant	1	co; pnb		33
1022	3129	E	Male	45+	caries; dentabs; oa	coffin	30
1023	3132	D/E	Male	15		?coffin	32
1024	3582	0	Unknown	Adult			not on plan
1025	3131	D/E	Male	Adult	sn; trauma	coffin	32
1026	4047	E	Female	45+	caries; dentabs; hfi; sinusitis		30
1027	4048	E	Unknown	25–34			30
1028	4049	E	Female	45+	caries; oa	pot	30
1029	4050	E	Female	25–34	caries	?coffin	30
1030	4052	D	?Female	Adult			32
1031	3506	E	Male	45+		coffin	30
1032	3507	E	Female	25–34		coffin	30
1033	4053	E	Infant	No age		?coffin	30
1034	4054	E	Unknown	Adult	oa		30
1035	4055	E	Unknown	Adult			30
1036	4056	E	Male	Adult		coffin	30
1037	7950	B/C	Male	Adult	pnb		34
3133	1038	D/E	Male	17		coffin	32
1039	3134	D	Female	Adult		?coffin	32
1040	3147	D/E	Male	45+		?coffin	32
1041	3135	D/E	Female	Adult	oa	BH 43	32
1042	7903	D/E	Female	20			32
1043	7904	D/E	Male	45+	oa		32
1044	7905	D/E	Male	20			32
1045	7906	D/E	Male	18			32
1046	7907	D/E	Female	Adult	sn		32
1047	3154	D/E	Male	45+	dentabs; gout; oa; trauma	coffin, glass	32
1048	7908	D/E	Female	Adult			32

Sk. no.	*Context*	*Phase*	*Sex*	*Age*	*Pathology notes*	*Archaeology notes*	*Fig. no.*
1049	7909	D/E	Female	Adult	oa; pnb		32
1050	7910	D/E	Female	25–34	caries; trauma	BH 64	32
1051	4009	A	Male	Adult	oa; trauma		36
1052	4057	E	Unknown	Adult		coffin(T)	30
1053	1790	E	Female	45+	oa	coffin(T), pillow stone, wands (2), BH 10, dendro	30
1054	4058	E	Male	Adult			30
1055	4061	E	Infant	1		?coffin	30
1056	4060	E	Female	Adult		?coffin	30
1057	4059	E	Infant	2		coffin	30
1058	1773	D/E	Male	Adult	oa; sn; trauma	coffin(T), dendro	30
1059	4062	E	Female	25–34	eh		30
1060	4063	E	Female	Adult			30
1061	4064	E	Infant	1		coffin, clay filled	30
1062	4010	C	Female	35–44	eh	coffin	33
1063	4065	C	Male	35–44	dish; oa; spond	coffin	33
1064	4066	E	Female	45+	dentabs		30
1065	3510	D	Unknown	Adult			32
1066	1793	E	Unknown	Adult		coffin	30
1067	4067	E	Juvenile	6		coffin, clay filled	30
1068	4069	E	Female	45+	dentabs; pnb; oa; spond	?coffin/charred board, pot	30
1069	4070	E	Juvenile	6	co	coffin	30
1070	4071	E	Unknown	Adult		coffin, BH 16	30
1071	4072	E	Female	45+		?coffin	30
1072	3512	D/E	Juvenile	6			30
1073	4073	D/E	Infant	No age			32
1074	4074	D/E	Male	Adult	oa; trauma		32
1075	4075	D/E	Unknown	Adult			32
1076	3513	E	Infant	No age		coffin	30
1077	3145	C/D	Male	25–34	sn; trauma	coffin	33
1078	3151	D	Infant	1		?coffin	32
1079	3152	D/E	Juvenile	5	co		32
1080	3153	D/E	Male	45+		coffin	32
1081	3156	A	Unknown	Adult		coffin	36
1082	3157	E	Unknown	Adult	pnb	coffin, BH 28	30
1083	3159	C/D	Unknown	Adult		coffin	33
1084	7951	D/E	Unknown	Adult			32
1085	7952	C/D	Male	45+			33
1086	3158	C/D	Unknown	Adult	trauma	?coffin	33
1087	3144	D/E				not lifted	32
1088	7911	D/E	Male	20			32
1089	3136	D/E	Juvenile	14		timber, roves (3)	32
1090	3143	D	Infant	1	co		32
1091	3514	E	Male	25–34	sepa	coffin	30
1092	3180	E	Female	25–34	oa; sn	coffin	30
1093	3174	D/E	Infant	0		coffin	32
1094	3515	D	Male	Adult	pnb; trauma		32
1095	3516	D	Male	17	sn		32
1096	4079	E	Female	45+	dentabs	coffin, ear muff (1)	30
1097	3517	D	Infant	2			32
1098	4078	E	Unknown	17		ear muff (1)	30
1099	3522	E	Unknown	45+	dentabs		30
1100	3523	D	Male	45+	caries	coffin	32
1101	4080	E	Female	Adult	pnb	?coffin/charred board	30
1102	4081	E	Juvenile	10		?coffin	30
1103	4082	E	Male	45+	discitis; oa; sn; spond; trauma		30
1104	3518	E	Male	45+	hfi		30
1105	4092	E	Female	25–34		coffin, ear muffs (2)	30
1106	4094	E	Male	45+	dentabs; dish; trauma	coffin	30
1107	4084	E	Male	45+	hfi; oa	coffin	30
1108	4083	D/E	?Male	Adult			32
1109	4085	E	Male	Adult	om; trauma	?coffin	30
1110	4086	E	Infant	1		coffin	30
1111	4087	E	Female	45+	caries; dentabs; oa		30
1112	4088	E	Infant	2			30
1113	3520	D/E	Infant	1			32
1114	1675	E	Unknown	Adult			30
1115	4089	E	Unknown	Adult		coffin	30
1116	4090	E	Unknown	Adult	co		30
1117	4093	E	Female	25–34		coffin	30
1118	4095	E	Unknown	Adult			30
1119	4091	E	Unknown	Adult		?coffin	30
1120	3526	D	Female	45+	caries; oa	coffin, ?pillow stone, bead	32

1121	4100	E	Infant	2		coffin, clay filled	30
1122	4101	E	Male	45+	oa	stones (2) under body	30
1123	3178	E	Infant	0			30
1124	3199	D/E	Infant	1		coffin	32
1125	3179	E	Infant	0			30
1126	3190	D/E	Infant	0			32
1127	3168	D/E	Male	25–34	caries		32
1128	3167	D/E	Female	Adult	caries	coffin	32
1129	3177	D/E	Infant	0			32
1130	3183	D/E	Female	Adult			32
1131	3187	D/E	Male	25–34	pnb	coffin	32
1132	3171	D/E	Infant	2			32
1133	3200	D/E	Female	Adult		coffin, metalwork	32
1134	3191	D/E	Male	45+	stt	?coffin	32
1135	3196	E	Juvenile	7		charred board	30
1136	3532	D/E	Male	25–34			30
1137	1504	C	Female	25–34			33
1138	4104	B	Unknown	Adult	caries; oa; pnb	coffin	34
1139	4106	E	Male	45+	caries; oa; trauma	coffin	30
1141	3533	E	Infant	No age			30
1142	4105	E	Unknown	Adult		coffin	30
1143	4107	E	Female	20		?coffin	30
1144	3535	D/E	Female	45+	sn		30
1145	4108	A/B	Unknown	45+		coffin	36
1146	7912	D/E	?Male	45+	caries; sn		32
1147	3166	E	Infant	0			30
1148	3162	E	Female	45+	caries		30
1149	3164	E	Male	45+	pnb; sn; trauma		30
1150	7972	0	?Female	Adult	dentabs; oa	coffin, burial not on plan	–
1151	4110	E	Female	45+	caries; oa; osdiss	coffin	30
1152	3540	D	Unknown	Adult			32
1153	3541	D	Juvenile	12			32
1154	1475	B	Unknown	20		?coffin	34
1155	3538	D/E	Infant	2			32
1156	3539	C	Female	25–34	caries; sfe		33
1157	4115	D	Male	25–34		chalice & paten, stones, ?shroud	32
1158	3103	D/E	Female	17	caries; pnb; sn	coffin	32
1159	3166	E	Female	25–34	caries; oa; trauma	pot	30
1160	3177	D/E	Male	13			32
1161	3201	D/E	Female	25–34		coffin	32
1162	3202	E	Male	45+	caries	coffin, BH 29	30
1163	3204	E	Unknown	Adult		coffin	30
1164	7913	C/D	Infant	2			33
1165	3206	E	Infant	No age			30
1166	3185	D/E	Female	Adult	caries		32
1167	3207	E	Juvenile	9		coffin	30
1168	3209	D/E	Infant	1		coffin	32
1169	3210	D/E	Male	35–44			32
1170	3211	D/E	Juvenile	10			32
1171	4111	E	Unknown	Adult		coffin	30
1172	3543	D/E	Unknown	45+		coffin	30
1173	4116	E	Male	25–34		coffin	30
1174	3508	E	Female	25–34	oa	coffin(T), dendro	30
1175	3539	C	Male	20			33
1176	4117	E	Male	35–44	dentabs; oa	coffin	30
1177	4118	E	Male	25–34		coffin	30
1178	1489	C	Female	25–34	sepa; sn	?coffin, with sk. 1179	33
1179	1489	C	Fetus	0		with sk. 1178	33
1180	3548	D/E	Juvenile	6	eh	coin	32
1181	4124	E	Female	25		coffin, ear muffs (2)	30
1182	4125	E	Unknown	Adult			30
1183	3547	E	Male	45+		grave lining (T), dendro	30
1184	3539	C	Female	16			33
1185	4128	C	Male	25–34	dentabs	coffin	33
1186	3212	D/E	Infant	1	pnb	ear muff (1)	32
1187	3214	D/E	Unknown	Adult		?coffin	32
1188	3215	D/E	Juvenile	7			32
1189	3216	D/E	Juvenile	5		coffin	32
1190	3171	D/E	Infant	0			32
1191	3181	E	Female	17	sn	coffin, pot	30
1192	3217	E	Infant	0		coffin	30
1193	3205	E	Unknown	Adult		coffin	30
1194	3221	D/E	Infant	1		coffin	32
1195	3219	D/E	Unknown	Adult		coffin	32

Sk. no.	*Context*	*Phase*	*Sex*	*Age*	*Pathology notes*	*Archaeology notes*	*Fig. no.*
1196	3502	E	Juvenile	8	co	coffin(T), BH 30	30
1197	4130	E	Female	45+	caries; sn	coffin	30
1198	4131	E	Female	45+	caries; dentabs	roves, BH 31	30
1199	3556	E	Juvenile	12		coffin	30
1200	3557	E	Infant	2			30
1201	4129	D	Male	25–34	caries		32
1202	1799	E	Female	45+	pnb	coffin, BH 32	30
1203	3220	E	Juvenile	9	co		30
1204	3224	D/E	Male	50+	oa; pnb	coffin	32
1205	3226	D/E	Female	25–34	caries	coffin	32
1206	3225	E	Juvenile	11		coffin	30
1207	3227	D/E	?Female	25–34		?coffin	32
1208	3228	E	Female	25–34	sn	coffin, BH 33	30
1209	3229	E	Male	25–34	sn		30
1210	3230	D/E	Infant	0			32
1211	3222	E	Female	Adult	caries; hfi; oa	coffin	30
1212	3231	E	Female	Adult		?coffin	30
1213	3232	E	Juvenile	8			30
1214	3233	E	Male	45+	oa; trauma	coffin	30
1215	3234	E	Male	25–34	caries	coffin, charcoal	30
1216	4132	D	Infant	1		coffin	32
1217	4133	D	Male	25–34	trauma	flint pebble	32
1218	4136	D	Infant	0			32
1219	4137	D	Unknown	Adult			32
1220	3561	D/E	Female	45+	caries; co	?coffin, ear muff (1)	32
1221	4138	E	Male	25–34	hfi	coffin, pillow stone (1)	30
1222	4139	D	Unknown	Adult		?coffin	32
1223	4140	D	Juvenile	11			32
1224	4141	D	Juvenile	6			32
1225	3503	E	Male	45+	caries; osdiss	coffin (T)	30
1226	4143	E	Female	25–34		coffin, pot	30
1227	3553	E	Male	35–44	pnb; trauma	coffin	30
1228	4152	E	Unknown	Adult		coffin, clay filled	30
1229	3235	D/E	Female	25–34	caries; trauma	coffin	32
1230	3236	E	Juvenile	8			30
1231	3237	E	Juvenile	6	caries	coffin, pot, pillows (3)	30
1232	3243	D/E	Infant	1			32
1233	3242	E	Infant	3			30
1234	3244	E	Male	Adult	oa	coffin, BH 34	30
1235	3246	E	Female	35–44	oa; trauma	coffin	30
1236	4147	D	Female	Adult	sn		32
1237	4145	D	Unknown	Adult			32
1238	4146	D	Unknown	Adult			32
1239	4148	D	Infant	4			32
1240	4149	D	Male	Adult	pnb		32
1241	3564	E	Male	Adult		coffin(T), BH 07 & BH35 (dendro)	30
1242	4150	D	Unknown	Adult	pnb		32
1243	4151	D	Infant	2			32
1244	4153	E	Juvenile	11		?coffin	30
1245	4154	D	Juvenile	Adult			32
1246	4155	D	Juvenile	7			32
1247	4156	D	Juvenile	Adult			32
1248	4157	D	Unknown	Adult			32
1249	4158	D	Male	Adult	oa		32
1250	4159	D	Female	Adult			32
1251	3565	E	Male	35–44	dentabs; sn		30
1252	4160	E	Infant	1		coffin	30
1253	4161	E	Infant	0	co	?coffin	30
1254	3566	E	Female	45+	caries; oa	?coffin	30
1255	3567	E	Female	Adult		coffin	30
1256	4164	D/E	Male	25–34	caries; co; sn		32
1257	4165	D/E	Female	Adult			32
1258	4166	D/E	Infant	2	co		32
1259	4167	D/E	Female	17	co		32
1260	3247	E	Female	25–34	caries	coffin, BH 17	30
1261	7914	E	Female	Adult			30
1262	3253	D/E	Female	25–34	caries; oa		32
1263	3254	E	Male	45+			30
1264	7915	D/E	Male	Adult	osdiss		32
1265	3255	E	Infant	2			30
1266	3249	E	Female	25–34			30
1267	3091	E	Male	25–34	caries	coffin	30
1268	3250	E	Male	45+	pnb	coffin	30

1269	3257	D/E	Male	17	trauma	coffin	32
1270	7916	E	Infant	2		ear muff (1)	30
1271	4171	D/E	Juvenile	9		?coffin	32
1272	4169	D/E	?Male	Adult		?coffin	32
1273	4170	D/E	Infant	2	rickets		32
1274	4168	D/E	Infant	1			32
1275	4172	D/E	Infant	0			32
1276	4173	D/E	Juvenile	5			32
1277	4174	D/E	Female	20	co		32
1278	4175	D/E	Unknown	Adult		?coffin	30
1279	4176	D/E	Female	20		?coffin	32
1280	4181	E	Female	Adult	op	coffin(T), BH 08, dendro	30
1281	4177	D/E	Unknown	Adult		?coffin	32
1282	4182	D/E	Unknown	Adult		?coffin	32
1283	4183	E	Male	Adult	oa	coffin, BH 36	30
1284	4184	E	Male	Adult		?coffin	30
1287	3256	E	Infant	0			30
1288	3194	D/E	Male	Adult	oa; trauma	?coffin	32
1289	7917	D/E					32
1290	3258	E	Male	Adult		?coffin	30
1291	3163	E	Female	45+	caries; dentabs; oa; sn	coffin	30
1292	3259	D/E	Male	Adult		?coffin	32
1293	3260	E	Female	Adult		coffin	30
1294	3263	D/E	?Male	Adult			32
1295	3262	D/E	Male	20	caries; discitis		32
1296	7918	C/D	Infant	0			33
1297	3261	D/E	Male	45+		coffin	32
1298	7919	E	Male	Adult			30
1299	3271	E	Male	45+		coffin	30
1300	3266	D/E	Unknown	Adult			32
1301	4185	D/E	Male	25–34	caries	?coffin	32
1302	3568	E	Male	25–34			30
1303	4186	D/E	Female	25–34		coffin(T), dendro	32
1304	3264	E	Male	Adult	stt	coffin	30
1305	7920	E	Female	17		coffin	30
1306	3265	E	Infant	1		coffin	30
1307	3267	D/E	Male	45+	pnb; trauma	?coffin	32
1308	3268	E	Male	Adult		coffin	30
1309	3269	E	Male	25–34	oa; pnb	coffin	30
1310	3270	E	Male	Adult	oa; trauma	coffin	30
1311	3271	E	Infant	2		ear muffs (2)	30
1312	3272	E	Male	17	co; discitis; tb	coffin	30, 66
1313	7921	D/E	Male	Adult		?coffin	32
1314	3276	E	Unknown	Adult		coffin	30
1315	3277	E	Male	45+	caries; oa; trauma	coffin	30
1316	3278	D/E	Male	50+	caries; dentabs; oa; pnb; sn		32
1317	3275	E	Fetus	8 mths		?timber	30
1318	3279	E	Female	20	sn	?coffin	30
1319	7922	E	Unknown	Adult			30
1320	3188	E	Female	Adult	oa		30
1321	3280	E	Male	25–34	trauma	coffin, BH 89	30
1322	3281	E	Female	20	eh		30
1323	3288	E	Male	33		coffin, ear muffs (2), BH 37	30
1324	3289	E	Male	39	sn	coffin	30
1325	3290	E	Unknown	Adult		coffin	30
1326	3291	E	Male	25–34	dentabs; pnb	?coffin	30
1327	3292	E	Male	Adult	oa; pnb	?coffin	30
1328	7923	E	Unknown	Adult			30
1329	7924	E	Male	Adult			30
1330	4504	A	Male	17	discitis	coffin	36
1331	4516	A	Female	Adult	oa; pdb	coffin	36
1332	7927	B/C	Unknown	Adult			34
1333	3615	A	Infant	1		coffin	36
1334	4515	A	Female	17	caries; dentabs	coffin	36
1335	4507	A	Female	17	caries; co; eh	coffin	36
1336	4517	A	Female	20	caries; eh	coffin	36
1337	4518	A	Female	22	caries	coffin	36
1338	4516	A	Male	Adult	dish; oa; op	coffin	36
1339	4537	A	Male	25–34	co	coffin	36
1340	4527	A	Male	Adult	oa; spond	coffin	36
1341	4543	A	Infant	2	co	coffin	36
1342	4539	8	Female	Adult		coffin	36
1342	4545	A	Female	Adult	caries	coffin	36
1343	4547	A	Male	50+	oa; trauma		36

Sk. no.	*Context*	*Phase*	*Sex*	*Age*	*Pathology notes*	*Archaeology notes*	*Fig. no.*
1344	4530	A/B	Female	Adult	bent; sepa		36, 90
1345	4553	A	Female	Adult	oa	coffin	36
1346	4548	A/B	Female	25–34	eh		36
1347	4536	A	Male	45+	as	coffin	36
1348	4540	A	Female	Adult	bent; oa	coffin	36
1349	4549	B/C	Juvenile	11			34
1350	4552	A	Male	25–34	caries; pnb; oa; trauma	coffin, large ring	36
1351	3619	A	Fetus	8 mths		coffin	36
1352	3620	A	Fetus	8 mths		coffin	36
1353	3621	A	Infant	0		coffin	36
1354	3617	A	Male	Adult	dentabs; oa; pdb; pnb	coffin	36
1355	3639	A	Fetus	8 mths		coffin, with sk. 1356	36
1356	3641	A	Female	36	caries	Mary Goy (d. 1820), coffin, with sk. 1355	36.
1357	3634	A	Infant	0		coffin	36
1358	3618	A	Juvenile	13		coffin	36
1359	3626	A	Juvenile	12	caries; eh	coffin, coin	36
1360	7925	B/C	Juvenile	5			34
1361	3644	A	Male	20	caries; eh		36
1362	3647	A	Female	45+	dish; oa	coffin	36
1363	3613	A	Male	20	caries; dentabs; dish; sn; trauma	coffin	36
1364	3649	A	Infant	0		coffin	36
1365	3650	A	Male	25–34	caries; dish; eh; rickets; trauma	coffin, coins (2)	36
1366	3637	A	Male	45+	caries; oa; op	coffin	36
1367	3651	A	Infant	Neonate		coffin	36
1368	3648	A	Infant	0		coffin	36
1369	3623	A	Female	Adult	oa	coffin	36
1370	3643	A	Male	45+	caries	coffin, coin	36
1371	3635	A	Unknown	Adult		coffin	36
1372	3656	A/B	Unknown	12	caries; dentabs		36
1373	3657	B/C	?Female	25–34	caries; dentabs; sn		34
1374	3658	A/B	Female	15			36
1375	3659	A/B	Juvenile	5			36
1376	3638	A	Female	35	caries; dentabs	Ann Goy (d. 1825), coffin	36
1377	3655	A	Infant	0	co	coffin	36
1378	3660	B/C	Juvenile	14	pnb		34
1379	3623		Female	Adult	caries; dentabs	coffin	36
1380	3662	A	Infant	Neonate		coffin	36
1381	3663	A	?Male	13	eh; pnb		36
1382	3680	A/B	Infant	Neonate			36
1383	3653	A	Unknown	Adult	oa	coffin	36
1384	3681	A	Infant	Neonate		coffin	36
1385	3684	A	Infant	0	pnb	coffin	36
1386	3685	A	Infant	Neonate		coffin	36
1387	3687	A	Infant	Neonate		coffin	36
1388	3688	A	Infant	Neonate		coffin	36
1389	3691	A/B	Female	25–34	caries; dentabs		36
1390	3692	A/B	Female	22	caries; dentabs		36
1392	3693	A	Infant	0		?coffin	36
1393	3682	A				coffin	36
1394	3695	A/B	Infant	4			36
1395	3699	A/B	Infant	1			36
1396	3700	A/B	Male	22	caries; dentabs		36
1397	3698	A	Unknown	Adult		coffin	36
1398	3701	A	Infant	1	co	coffin	36
1399	4546	A	Unknown	45+	caries; dish; oa; osdiss	coffin	36
1400	4556	A/B	Unknown	30	caries; co	?coffin	36
1401	4726	A/B	Juvenile	13	pnb		36
1402	4559	A/B	Female	Adult	oa; osdiss; spond; stt	?coffin	36
1403	4727	B/C	Juvenile	8	trauma		34
1404	4558	B/C	Unknown	Adult			34
1405	4526	A	Juvenile	7			36
1406	3679	A	Infant	2		coffin	36
1407	3703	A/B	Male	13	pnb		36
1408	3706	A/B	Infant	2			36
1409	3694	B/C	Male	35–44	oa; polio		34
1410	4557	A	Infant	0	pnb	coffin	36
1411	4560	C/D	Unknown	Adult			33
1412	4561	C/D	Infant	1			33
1413	4562	D/E	Unknown	Adult			32
1414	4555	A	Male	22	eh	coffin	36
1415	4728	B/C	Juvenile	7			34
1416	4729	B/C	Juvenile	7			34

1417	4508	A	Juvenile	13		coffin	36
1417	4563	A	Juvenile	13		coffin	36
1418	4730	A/B	Juvenile	5			36
1419	4532	8	Female	Adult	oa	coffin	36
1419	4564	A	Female	Adult			36
1420	4565	A/B	Female	Adult	trauma	?coffin	36
1421	3709	A	Fetus	8 mths	pnb	coffin	36
1422	3710	A	Infant	0		coffin	36
1423	3707	A	Female	Adult	polio; sn; trauma	coffin, coin	36
1424	3713	A	Infant	0		coffin	36
1425	3683	A	Male	28	polio	Joseph Bromley Swallow (d. 1842) coffin, coin	36, 68
1426	3715	A	Infant	1		coffin	36
1427	3718	C–E	Unknown	25–34	caries		not on plan
1428	3719	A/B	Unknown	Adult			36
1429	3702	A	Female	Adult	caries; sn; trauma	coffin	36
1430	3702	A	Infant	0	pnb	coffin	36
1431	3721	A	Female	Adult		coffin	36
1432	3705	A/B	Unknown	17	caries	coffin	36
1433	3706	A/B	Female	45+	caries; oa; trauma	?coffin	36
1434	3716	A	Unknown	Adult	oa; op	coffin, earrings	36
1435	4568	B/C	Juvenile	10			34
1436	4731	A/B	Infant	0			36
1437	4732	A/B	Infant	1			36
1438	4582	B/C	Male	45+		?coffin	34
1439	4733	A/B	Infant	1			36
1440	4566	A/B	Unknown	13			36
1441	4581	A/B	Male	35–44	caries; dentabs		36
1442	4583	A/B	Female	15–24	disloc		36, 79
1443	4584	A	Infant	2		coffin	36
1444	4585	A/B	Female	Adult			36
1445	4734	A/B	Female	Adult			36
1446	3720	A/B	Female	25–34	eh		36
1447	3722	A	Infant	No age	pnb	coffin	36
1448	3723	A	Male	22	caries; dentabs; maligt; sn	coffin, textile, coins (3)	36, 91
1449	3696	A	Male	50+	oa	coffin	36
1450	3697	A	Female	55	dentabs; oa; pnb	Margaret Swallow (d. 1845) coffin, gold ring	36
1451	3712	A/B	Male	45+	dentabs; dish; oa; pdb; trauma		36
1452	3725	B/C	Female	Adult			34
1453	3726	A/B	Unknown	Adult			36
1454	3727	A/B	Male	35–44	dentabs; tb		36, 67
1455	3728	A/B	Juvenile	5–15			36
1456	3728	A/B	Infant	1			36
1457	3729	A	Infant	2			36
1458	4735	C/D	Unknown	Adult			33
1459	4736	C/D	Unknown	19	trauma		33
1460	4737	A/B	Male	20	caries; sn		36
1461	4738	A/B	Juvenile	14	co; eh		36
1462	4602	A/B	Male	35–44			36
1463	4739	B/C	Male	25–34	dentabs; sn		34
1464	4740	A/B	Infant	4	co		36
1465	4741	A/B	Unknown	17	caries		36
1466	4605	A	Female	25–34	trauma	coffin	36
1467	4742	B/C	Unknown	Adult	pnb		34
1468	4606	A/B/C	Unknown	Adult	sn	washed away by rain	not on plan
1469	4592	A	Infant	1		coffin	36
1470	4593	A	Infant	0		coffin	36
1471	3717	A	Female	Adult		coffin	36
1472	3730	B/C	Unknown	17			34
1473	3733	B/C	Male	25–34	pnb		34
1474	3732	B/C	Female	Adult			34
1475	3734	A	Infant	1		coffin	36
1476	3735	A	Infant	1	pnb rib	coffin	36
1477	3736	A	Infant	1	co	coffin	36
1478	3737	A	Female	Adult	caries		36
1479	3738	B/C	Male	Adult	pnb; trauma		34
1480	3741	A	Unknown	Adult		coffin	36
1481	3742	A	Female	45+	caries; sn; trauma	coffin, lead pendant, coin	36
1482	3743	B/C	Infant	2			34
1483	3744	B/C	Female	Adult	oa		34
1484	3750	A	Unknown	15	caries; pnb		36
1485	3748	B/C	Unknown	Adult	pnb		34
1486	3751	A/B	Juvenile	13			36

Sk. no.	*Context*	*Phase*	*Sex*	*Age*	*Pathology notes*	*Archaeology notes*	*Fig. no.*
1487	3704	B/C	?Male	Adult			34
1488	3752	B/C	Infant	1		buckle	34
1489	3636	A	Female	Adult	oa	coffin	36
1490	3731	C/D	Juvenile	10			33
1491	3731	B/C	Female	25–34	sn	BH 69	34
1492	3749	A	?Female	17	caries; dentabs; pnb	coffin	36
1493	4743	A/B	Unknown	Adult			36
1494	4744	B/C	Unknown	Adult	oa; trauma		34
1495	4608	A/B	Female	Adult		coffin	36
1496	4607	A	Male	35–44	caries; dentabs; oa; sinusitis; sn; spond	coffin	36
1497	4745	B/C	Female	Adult			34
1498	4586	B/C	Male	45+		coffin	34
1499	4746	A/B	Infant	4			36
1500	4747	C/D	Male	Adult			33
1501	4748	A/B	Infant	4			36
1502	4575	A	Male	20	caries; pnb rib; sn	coffin	36
1503	4749	B/C	Juvenile	14			34
1504	4750	C/D	Unknown	25–34			33
1505	4751	B/C	?Female	20	polio; sepa; sn		34
1506	4610	A	Female	Adult	oa	coffin	36
1507	4596	A	Infant	1	bent; co	coffin	36
1508	4589	A	Male	35–44	sn	coffin	36
1509	4621	A/B	Juvenile	9			36
1510	4622	A	Infant	0		coffin	36
1511	3757	B/C	Female	25–34	dentabs		34
1512	3636	A	Female	Adult	dish; osdiss; trauma	coffin	36
1513	3636	A	Infant	0		coffin	36
1514	3758	A	Female	20		coffin	36
1515	3762	A/B	Juvenile	12			36
1516	3764	B/C	Juvenile	5			34
1517	3768	B/C	Female	25–34	oa		34
1518	3769	A/B	Infant	1	caries		36
1519	3754	A/B	Unknown	Adult			36
1520	3760	B/C	Infant	0			34
1521	3761	A/B	Juvenile	5–15			36
1522	3770	A	Male	25–34			36
1523	3773	B/C	Female	Adult			34
1524	3753	A/B	Male	45+	caries		36
1525	3774	B/C	Infant	0			34
1526	3775	A/B	?Male	Adult	pnb		36
1527	3776	C/D	Unknown	Adult	trauma		33
1528	3778	C/D	Male	Adult			33
1529	3777	B/C	Female	20	caries; eh		34
1530	3799	A/B	Male	Adult	oa		36
1531	4752	B/C	Juvenile	9	caries; co		34
1532	4753	C/D	Juvenile	Adult		BH 75	33
1533	4754	C/D	Male	Adult			33
1534	4755	C/D	Juvenile	10			33
1535	7968	C	Unknown	Adult	oa		33
1536	3785	B/C	Male	25–34			34
1537	3787	B/C	Juvenile	10			34
1538	3788	B/C	?Male	45+			34
1539	3789	C/D	Unknown	Adult			33
1540	3790	B/C	Juvenile	5			34
1541	3791	B/C	Juvenile	13			34
1542	3792	C/D	Female	45+	caries		33
1543	5480	C	Male	Adult			33
1544	4756	D/E	Infant	1			32
1545	4757	B/C	Infant	1			34
1546	4612	A	Unknown	Adult		coffin	36
1547	4623	C/D	?Female	Adult			33
1548	4624	D	Male	45+	dentabs; oa; sn	BH 46	32
1549	4758	B/C	Juvenile	6			34
1550	4759	B/C	Unknown	Adult			34
1551	3796	C/D	Unknown	45+	sinusitis		33
1552	3797	C/D	Unknown	Adult			33
1553	3798	C	Infant	0			33
1554	3795	C/D	Male	Adult		?coffin	33
1555	3800	C/D	?Female	25–34			33
1556	4760	C/D	Male	45+			33
1557	4627	A	Juvenile	6	caries	?coffin	36
1558	4628	B/C	Female	25–34		?coffin	34

1559	4629	A	Female	Adult		coffin	36
1560	4761	B/C	Female	Adult			34
1561	3801	D	Unknown	Adult			32
1562	3802	C/D	Male	Adult	trauma		33
1563	3803	B/C	Unknown	Adult			34
1564	3804	D	Unknown	Adult			32
1565	3811	D	Female	Adult			32
1566	3812	B/C	Male	45+	sn; trauma		34
1567	3813	C	Infant	1			33
1568	3822	B/C	Female	Adult			34
1569	3818	C/D				did not reach finds shed	33
1570	4630	A/B	Unknown	Adult			36
1571	4631	C/D	Female	Adult			33
1572	4634	C/D	Female	25–34	caries; oa	BH 77	33
1573	4615	A	Male	Adult	trauma	coffin	36
1574	4635	D/E	Male	Adult	oa; trauma	coffin, BH 74	32
1575	4762	D/E	Female	Adult			32
1576	4763	D/E	Male	25–34			32
1577	4764	D/E	Male	25–34	sn; trauma		32
1578	4765	C/D	Unknown	Adult	dentabs		33
1579	4766	B/C	Infant	0			34
1580	4636	A/B	Male	Adult			36
1581	4637	B/C	Male	25–34		coffin	34
1582	4638	B/C	Female	15			34
1583	4639	B/C	Unknown	Adult		?coffin	34
1584	4767	B/C	Juvenile	5			34
1585	4633	B/C	Juvenile	Adult			34
1586	4768	C/D	Female	Adult	sepa		33
1587	4769	C/D	Female	16	osdiss		33
1588	4640	C/D	Female	Adult	caries		33
1589	4770	C/D	Male	Adult	oa; sn		33
1590	4641	A	Infant	1	co	coffin	36
1591	3820	A/B	Juvenile	11			36
1592	3817	C/D	Female	15	co; pnb		33
1593	3819	C/D	?Male	Adult	oa		33
1594	5479	C/D	Infant	0			33
1595	3823	C	Unknown	Adult			33
1596	3816	C/D	Male	25–34	caries		33
1597	3828	B/C	Infant	4	caries		34
1598	3814	B/C	Juvenile	15	caries		34
1599	3815	B/C	Female	25–34	caries		34
1600	3829	C/D	Unknown	Adult		roves (2), ?redeposited	33
1601	3830	C	Male	25–34			33
1602	4771	C/D	Unknown	Adult			33
1603	4772	B/C	Unknown	Adult	trauma		34
1604	4773	B/C	Infant	2			34
1605	4642	D/E	Unknown	Adult			32
1606	4643	D/E	Female	Adult		?coffin, BH 47	32
1607	4774	A/B	Juvenile	7	pnb		36
1608	4644	A	?Male	Adult		coffin	36
1609	4645	A	Male	Adult	eh; sn	coffin	36
1610	4775	B/C	Male	25–34			34
1611	4776	B/C	Unknown	Adult			34
1612	4777	B/C	Female	25–34	caries		34
1613	4617	A	Male	Adult		coffin	36
1614	4778	B/C	Infant	2			34
1615	4779	B/C	Male	45+			34
1616	3832	D	Female	Adult	dentabs		32
1617	3833	C/D	Male	20			33
1618	3835	C	Male	25–34		coffin, buckle, pot	33
1619	4646	A	Juvenile	6	eh	coffin, coin	36
1620	4618	A	Infant	2	caries	coffin	36
1621	4780	D/E	Female	Adult			32
1622	4649	C/D	Male	Adult	pnb; ra; trauma		33, 58
1623	4782	C/D	Juvenile	7			33
1624	4783	C/D	Unknown	Adult			33
1625	4784	C/D	Juvenile	8			33
1626	3844	B/C	Juvenile	8			34
1627	3849	A/B	Infant	0			36
1628	4785	B/C	Unknown	Adult	pnb		34
1629	4786	A	Unknown	Adult	trauma		36
1630	4787	A/B	Male	45+	maligt		36
1631	4788	C/D				may not have survived washing.	33
1632	4789	A/B	Unknown	Adult			36

Sk. no.	*Context*	*Phase*	*Sex*	*Age*	*Pathology notes*	*Archaeology notes*	*Fig. no.*
1633	4790	C/D	Unknown	Adult			33
1634	4650	A	Unknown	Adult			36
1635	4791	B/C	Juvenile	Adult			34
1636	3646	A	Female	Adult	oa	coffin	36
1637	3851	B/C	Juvenile	8			34
1638	3850	B/C	Infant	3	co		34
1639	3854	B/C	Male	25–34		coffin, pot	34
1640	3855	B/C	Female	17	dentabs; sn	coffin	34
1641	3856	A/B	Unknown	Adult	oa; trauma	coffin	36
1642	3857	B/C	Unknown	20	co		34
1643	4661	D/E	Unknown	No age			32
1644	4792	D	Male	25–34	oa; pnb; sn	BH 76	32
1645	4544	B/C	Male	Adult			34
1646	4662	A	Unknown	17		coffin	36
1647	4794	C/D	Juvenile	6			33
1648	4795	C/D	Unknown	Adult			33
1649	4663	A	Male	50+	dish; oa; trauma	coffin	36
1650	4796	B/C	Male	25–34			34
1651	4797	D/E	Male	Adult	oa		32
1652	4798	E	Juvenile	6	co		32
1653	4799	C/D	Unknown	Adult	pnb		33
1654	4800	B/C	Unknown	Adult			34
1655	4801	D/E	Female	Adult	oa		32
1656	3871	B/C	Juvenile	9			34
1657	3872	B/C	Unknown	Adult			34
1658	4802	D/E	Juvenile	13			32
1659	4803	D/E	Juvenile	9			32
1660	4665	D/E	Unknown	Adult	trauma	finds belong to 4545	32
1661	4654	C/D	Male	Adult	sn		33
1662	4670	A	Juvenile	12		coffin	36
1663	4804	B/C	Female	17	caries; dentabs; eh		34
1664	4805	B/C	Juvenile	8			34
1665	4806	B/C	Female	Adult	pnb		34
1666	4666	A	Female	Adult	oa	coffin	36
1667	4807	D/E	Female	Adult			32
1668	4808	C/D				may not have survived washing	33
1669	4667	A	Unknown	Adult	trauma	coffin	36
1670	4668	A	Juvenile	5		coffin	36
1671	3873	C/D	Unknown	Adult			33
1672	4652	B/C	Unknown	Adult		?coffin	34
1673	4653	B/C	Male	Adult	gout; sn; trauma	BH 48	34
1674	3875	C/D	Female	17		coffin	33
1675	3876	C/D	Unknown	Adult	hfi	coffin	33
1676	3877	B/C	Juvenile	12			34
1677	3878	C/D	Unknown	Adult	pnb; trauma		33
1678	4655	A/B	Male	25–34	fibdys; om		36
1679	4809	D				may not have survived washing	32
1680	4810	D	Unknown	Adult			32
1681	4811	C/D	Juvenile	11			33
1682	4669	A/B	?Male	25–34	caries; eh		36
1683	4812	B/C	Unknown	Adult			34
1684	4813	B/C	Juvenile	9			34
1685	4657	A	Unknown	Adult		?coffin	36
1686	4656	A	Male	Adult	oa	coffin	36
1687	4814	D/E	Unknown	Adult			32
1688	4815	D/E	?Male	19			32
1689	4816	C/D	?Male	21			33
1690	4658	A	Unknown	Adult		coffin	36
1691	3870	D/E	Male	50+	caries; trauma	coffin	32
1692	3887	C/D	Male	25–34	caries	pillow stones	33
1693	4817	C/D	Unknown	Adult			33
1694	4818	C	Male	25–34		BH 50	33
1695	4676	B/C	Female	45+	caries; dentabs; hfi; sn		34
1696	4677	D/E	Unknown	Adult		coffin	32
1697	4819	D/E	Unknown	Adult			32
1698	4820	C/D	Unknown	Adult			33
1699	4678	B/C	Female	20	caries; eh; sn	coffin	34
1700	4821	B/C	Unknown	Adult			34
1701	3893	B/C	Infant	4	co		34
1702	3640	A	Male	48	sn; trauma	William Goy (d. 1836), coffin	36
1703	3894	A/B	Unknown	Adult			36
1704	4659	B/C	Female	21			34
1705	4660	A	Unknown	Adult			36

1706	4671	C/D	Male	15			33
1707	3898	B/C	Male	45+	oa; trauma		34
1708	4840	C/D	Unknown	Adult			33
1709	3900	D/E	Infant	0		coffin, ear muff (1), pot	32
1710	4822	C/D	Female	Adult	oa		33, 52
1711	4823	D/E	?Female	Adult			32
1712	4824	D/E	Infant	3	pnb; rickets		32
1713	4679	A	Unknown	Adult		coffin	36
1714	4683	D/E	Female	20			32
1715	4684	E	Male	25–34	co		32
1716	4672	B/C	Female	Adult			34
1717	4685	A	Juvenile	8		coffin	36
1718	4691	A/B	Female	Adult		coffin	36
1719	3895	D/E	Male	25–34	caries; oa	coffin	32
1720	4723	E	Unknown	Adult	oa		32
1721	4673	A	Unknown	Adult		coffin	36
1722	3902	B/C	Female	25–34		coffin	34
1723	4841	0	Male	45+	trauma		not on plan
1724	4692	D/E				coffin, bones may not have survived washing	32
1725	4693	D/E	Female	Adult		coffin	32
1726	4694	E	Female	25–34			32
1727	4825	D/E	Juvenile	14			32
1728	3905	C/D				did not reach finds shed	33
1729	3906	C/D	Male	25–34	dentabs	?coffin	33
1730	4687	A	Female	Adult		?coffin	36
1731	4688	A	Unknown	Adult		coffin	36
1732	4690	A	Female	Adult	oa	coffin	36
1733	4826	D/E				did not reach finds shed	32
1734	3936	C/D	Infant	1			33
1735	4827	D/E	Male	Adult	oa		32
1736	4828	A/B	Male	Adult	pnb		36
1737	4829	A/B	Unknown	Adult			36
1738	4695	C/D	Male	15	caries; co	BH 78	33
1739	4696	C/D	Female	25–34	dentabs		33
1740	4689	D/E	Juvenile	7	co	?coffin	32
1741	4697	E	Juvenile	10		?coffin	32
1742	4698	E	Male	25–34	pnb	coffin, ear muff (1)	32
1743	4699	E	Unknown	Adult	caries; oa		32
1744	4711	C/D	Female	Adult			33
1745	4712	D	Female	Adult	oa	BH 49	32
1746	4701	E	Unknown	Adult			32
1747	4702	E	Unknown	Adult			32
1748	4713	C/D	Male	Adult	sn		33
1749	4830	E	Male	Adult			32
1750	4831	E	Female	Adult		?coffin	32
1751	3908	E	Unknown	Adult	caries; oa	coffin(T), pot, dendro	30
1752	3911	C/D	Unknown	17			33
1753	3912	B/C	?Female	13			34
1754	3979	E	Male	45+	caries; oa; spond	coffin(T), ear muff (1), dendro	30
1755	3858	B/C	Juvenile	5			34
1756	3915	B/C	Male	45+	caries; osdiss; sn	?coffin	34
1757	3917	C/D	Male	20	caries		33
1758	4704	E	Unknown	20			32
1759	4705	E	Male	45+	oa; sn		32
1760	4706	E	Male	25–34			32
1761	4707	E	Infant	2			32
1762	4832	E	Unknown	Adult			32
1763	4708	E	Female	Adult			32
1764	4833	E	Infant	4	caries; eh		32
1765	4834	E	Female	45+			32
1766	4835	E	Male	Adult	gout; oa; pnb		32
1767	4836	E	Unknown	Adult			32
1768	4717	E	Male	45+	caries		32
1769	4837	D/E	Male	45+	dentabs; oa		32
1770	4718	D/E	Female	Adult			32
1771	3918	B/C	Female	Adult	oa		34
1772	3919	B/C	Male	Adult			34
1773	3922	B/C	Male	25–34	co		34
1774	3923	B/C	Unknown	Adult			34
1775	3924	B/C	Male	45+	head wnd; oa		34
1776	3925	C/D	Juvenile	Adult			33
1777	3926	C/D	Juvenile	10	caries		33
1779	3927	D/E	Male	20			32

Sk. no.	*Context*	*Phase*	*Sex*	*Age*	*Pathology notes*	*Archaeology notes*	*Fig. no.*
1780	3930	C/D	Female	20	caries; trauma	BH 68	33
1781	3931	B/C	Unknown	Adult			34
1782	3916	E	Unknown	Adult		coffin	30
1783	3921	D/E	Female	Adult		coffin, ear muffs (2)	32
1784	3868	E	Female	25–34		coffin(T), wands (2), ear muffs (2), dendro	30
1785	3868	E	Infant	0		in same coffin as sk 1784	30
1786	3932	C/D	Juvenile	5	co		33
1787	3933	D/E	Unknown	45+			32
1788	3934	E	Male	35–44	spond	coffin	30
1789	3935	B/C	Unknown	Adult			34
1790	3937	C/D	Female	Adult			33
1791	4719	E	Male	17	dentabs		32
1792	4720	D/E	Unknown	Adult			32
1793	4724	E	Male	25–34			32
1794	4838	E	Male	45+	pdb		32, 87
1795	4839	D/E	Female	35–44	caries; dentabs		32
1796	4725	E	?Male	Adult	oa; sn		32
1797	7990	E	Male	25–34	dentabs		30
1798	7991	D/E	Unknown	25–34	caries; eh; trauma		32
1799	7989	D/E	Unknown	Adult			32
1800	7988	E	Female	Adult		?coffin	30
1801	3883	D/E	Unknown	25–34	pnb	coffin(T), dendro	32
1802	3938	B/C	Unknown	17		coffin	34
1803	3939	D/E	Unknown	15		coffin(T)	32
1804	3940	C/D	Female	Adult			33
1805	3941	D/E	Male	35–44	caries; oa; trauma	coffin	32
1806	3943	C/D	Juvenile	5			33
1807	3944	C/D	Male	Adult			33
1808	3945	C/D	Male	25–34			33
1810	4729	–	Unknown	Adult		coffin	not on plan
1811	7987	D/E	Infant	0	co		32
1815	7985	D/E	Male	Adult	disloc; oa		32
1817	7986	D/E	Unknown	Adult			32
1818	7992	E	Male	25–34	eh		30
1819	3869	E	Male	45+	caries; dentabs; dish; oa; trauma	coffin(T), wand, dendro	30
1820	3947	D/E	Juvenile	15		?coffin	32
1821	3948	C/D	Female	25–34	co		33
1822	3949	B/C	Male	25–34			34
1823	3950	B/C	Infant	1			34
1824	3951	E	Male	45+	oa	coffin	30
1825	3946	D/E	Infant	3	co	coffin(T), boar tooth amulet, BH 11 (dendro)	32
1826	3954	B/C	Unknown	Adult			34
1827	3955	D/E	Infant	4	co	coffin	32
1828	3958	B/C	Infant	No age			34
1830	7980	D/E	Female	17			32
1831	7981	C/D	Unknown	25–34			33
1832	7996	E	Unknown	Adult	trauma		30
1833	7997	E	Unknown	Adult	oa		30
1834	7998	E	Juvenile	Adult			30
1835	7994	E	Unknown	Adult			30
1836	3959	E	Infant	1		re–burial in ditch fill	not on plan
1837	3961	C/D	Unknown	Adult			33
1838	3962	E	Unknown	Adult		roves (2), 3962 mostly cut away by 3965	30
1839	3964	D/E				may not have survived washing	32
1840	3965	E	Female	25–34	caries	roves (30), ?from G3962	30
1841	7993	E	Unknown	Adult		with sk 1842	30
1842	7993	E	Infant	No age		with sk 1841	30
1843	3966	E	Male	45+	oa; trauma	coffin, red ear muffs (2)	30
1847	3969	C/D	Unknown	25–34			33
1848	3970	E	Male	25–34	caries	coffin	30
1849	3972	C/D	Male	45+	oa		33
1850	3973	C/D	Male	25–34	sn		33
1852	3976	C/D	Male	25–34	caries		33
1853	3977	D/E	Unknown	Adult			32
1854	3978	D/E	Juvenile	6			32
1855	7984	C/D	Juvenile	12			33
1856	7995	C/D	Unknown	35–44			33
1857	7982	C/D	Infant	1			33
1858	7983	C/D	Female	Adult			33
1859	3974	E	Female	Adult	oa	coffin, wicker–work base	30
1861	3967	E	Juvenile	6		coffin(T)	30

1862	3981	C/D	Unknown	Adult			33
1863	3968	E	Male	45+	dentabs; oa; trauma	coffin(T), dendro, grass pillow & ear muffs (3), wands	24, 30
1864	3982	D/E	Male	25–34		coffin	32
1865	4842	0	Unknown	Adult	trauma	no skeleton sheet	not on plan
1866	3985	D/E	Female	25–34		coffin, rove (1)	32
1867	3980	D/E	Female	25–34	sn	coffin(T), ear muffs (2), wand, dendro	32
1868	3986	D/E	Juvenile	6	caries; dentabs	coffin	32
1869	3971	D/E	Male	45+	caries; dish; oa	coffin(T), dendro	32
1870	3987	E	Male	25–34	caries; eh; gout	coffin, ear muff (1), ?pot, flint	30
1871	3989	D/E	Male	45+	oa; osdiss; sn; spond		32
1872	3990	D/E	Infant	1			32
1873	3991	D/E	Male	Adult			32
1874	3992	B/C	Female	Adult	caries; dentabs		34
1875	3994	D/E	Female	45+		coffin(T)	32
1876	3995	E	?Female	16	pnb; sn	coffin	30
1877	3996	D/E	Female	25–34	dentabs; discitis	coffin	32
1878	3998	D/E	?Male	25–34		coffin	32
1879	3999	D/E	Unknown	Adult	caries	coffin(T), dendro	32
1880	5001	E	Male	25–34	caries	ear muffs (2 small ones)	30
1881	5024	D/E	Infant	1		coffin(T), pillow (1), dendro	32
1882	5004	E	Male	25–34	sn	board cover	30
1883	5005	E	Male	17	caries; co; pnb; trauma		30
1884	5006	E	Male	25–34			30
1885	3983	D/E	?Female	25–34		?coffin	32
1886	5007	D/E	Female	25–34	caries		32
1887	5008	D/E	Unknown	Adult	hallvag		32
1888	5009	E	Infant	2	co	pillow stones (7)	30
1889	5003	E	Female	Adult			30
1890	5011	D/E	Infant	2			32
1891	5012	D/E	Juvenile	6		coffin(T)	32
1892	5013	E	Female	45+		coffin, roves (2 – ?stray)	30
1893	5015	E	Juvenile	5	co	?coffin	30
1894	5014	E	Infant	1			30
1895	5016	D/E	Male	25–34		river cobbles	32
1896	5017	E	Unknown	Adult			30
1897	5018	D/E	Male	45+	caries	coffin, rove (1) & nails	32
1898	5020	E	Male	Adult			30
1899	5021	E	Juvenile	8			30
1900	5023	D/E	Female	25–34	caries	coffin	32
1901	5025	D/E	?Female	Adult			32
1902	5026	D/E	Female	25–34		roves (21)	32
1903	5027	E	Female	20		coffin	30
1904	5002	E	Male	25–34	co; sn	board lining (T), dendro	30
1905	5028	E	Female	Adult	sn	coffin, ear muff (1)	30
1906	5029	E	Female	25–34	spond	ear muff (1)	30
1907	5031	D/E	Infant	No age		coffin(T), rove (1), dendro	32
1908	5030	C/D	Juvenile	11			33
1909	5032	E	Juvenile	8		part of multiple burial	11, 30
1910	5032	E	Male	45+	caries; dentabs; disloc; oa; om	ditto, BH 38	11, 30, 80
1911	5032	E	Male	45+	bent; eh; trauma	ditto, BH 14	11, 30
1912	5032	E	Juvenile	7	eh	ditto	11, 30
1913	5032	E	Juvenile	12		ditto	11, 30
1914	5036	D/E	Infant	1			32
1915	7999	E	Male	Adult			30
1916	5037	E	Female	25–34	caries	roves + nails, ear muffs (2), BH 41	30
1917	5038	E	Male	25–34		coffin	30
1918	5040	E	?Male	25–34	sepa; oa; trauma	coffin	30
1919	5046	D/E	Juvenile	Adult		coffin, ear muff (1)	32
1920	5047	D/E	Male	45+	caries; dentabs; oa; spond	coffin	32
1921	5049	D/E	Infant	0		coffin	32
1922	5050	E	Male	45+	dentabs; oa; pnb rib; sn; spond		30
1923	5051	D/E	Unknown	Adult		coffin, ear muffs (2)	32
1924	5052	E	Unknown	22		coffin(T), ear muffs (2), dendro	30
1925	5044	E	Male	45+	caries; dentabs	coffin(T), wands (3), dendro	30
1926	5045	E	Male	45+	caries; dentabs; dish; oa; trauma	coffin(T), wands (2), BH 12, dendro	30
1927	5061	E	Female	25–34		coffin(T)	30
1928	5079	C/D	Juvenile	5	co		33
1929	7000	A	Infant	0		coffin	36
1930	7000	A	Infant	0	pnb	coffin	36
1931	7012	A	Infant	0	pnb	coffin	36
1932	7001	A	Infant	0		coffin	36
1933	7008	A	Female	17	caries	coffin	36
1934	7002	A	Female	Adult	caries	coffin, hair comb	36

Sk. no.	*Context*	*Phase*	*Sex*	*Age*	*Pathology notes*	*Archaeology notes*	*Fig. no.*
1935	7016	A	Infant	0		coffin	36
1936	7016	A	Female	17	caries	coffin, hair comb	36
1937	7009	A	Male	45+	caries; ea; oa	coffin	36, 62
1938	7004	A	Female	Adult	caries; op; trauma	coffin	36
1939	7003	A	Female	Adult	pnb; scoliosis; oa; trauma	coffin	36
1940	4993	B/C	Unknown	Adult			34
1941	7008	A	Juvenile	6			36
1942	4994	A/B	Female	Adult	dentabs; oa		36
1943	7013	A	Male	25–34	caries; dentabs; oa; trauma	coffin	36
1944	7020	A/B	Unknown	16		?coffin	36
1945	7019	A	Male	50+	caries; dish	coffin, small AE bell	36
1946	7022	A	Infant	0		coffin	36
1947	7024	A	Infant	0		coffin	36
1948	7025	A	Juvenile	11	sinusitis	coffin	36
1949	7023	A	Male	Adult	caries; dentabs; oa; pnb; sn	coffin, coin	36
1950	7033	A	Female	25–34		coffin	36
1951	7021	A	Female	Adult	caries; scoliosis; trauma	coffin	36
1952	7032	A	Female	Adult	eh; op	?coffin, finger-ring	36
1953	7029	A	Infant	0			36
1954	7030	A	Male	Adult	dish; oa	coffin	36
1955	7017	A	Juvenile	6	caries; co	coffin	36
1956	7005	A	Infant	1	rickets; pnb	coffin	36
1957	7039	A	Male	45+	caries; oa	coffin	36
1958	7029	A	Juvenile	9	co	coffin	36
1959	7040	A	Female	17	caries	coffin	36
1960	5106	A	Juvenile	12		coffin	36
1961	5107	A	Infant	0		coffin	36
1962	5108	A	Female	17	caries	coffin	36
1963	5112	A	Male	25–34	caries	coffin	36
1964	5102	A	Male	Adult	polio	coffin, coin, textile	36
1965	5109	A	Female	Adult	caries	coffin	36
1966	5122	A	Female	Adult	caries; oa; op; osdiss	coffin	36
1967	5111	A	Infant	0		coffin	36
1968	5110	A	Infant	0		coffin	36
1969	5117	A	Male	Adult	caries; oa; pnb; sn	coffin	36
1970	7014	A	Male	Adult	pnb	coffin	36
1971	7018	A/B	?Male	Adult	caries; oa; op		36
1972	5104	A	Male	Adult	sn; trauma	coffin	36
1973	5129	A	Male	25–34	caries	coffin	36
1974	5133	A	Female	Adult	caries; co; dentabs	coffin	36
1975	5127	A	Male	45+	oa; sn	coffin	36
1976	5150	A	Infant	0		coffin letters 'J' & 'H'	36
1977	5114	A	Male	17	pnb rib	coffin	36, 70
1978	5152	A	Male	12	caries; co	coffin	36
1979	7035	A	Infant	1	co	coffin	36
1980	7042	A	Infant	0		?coffin	36
1981	7034	A/B	Infant	0	co		36
1982	7046	A	Infant	2		coffin	36
1983	7027	A	Female	20	caries	coffin	36
1984	7037	A	Female	35–44	caries; hfi	coffin	36
1985	7047	A	Female	Adult		coffin	36
1986	7051	A	Infant	1		coffin, part of sk. 2009	36
1987	7048	A	Female	17		coffin	36
1988	7035	A	Female	17	caries	coffin	36
1989	7052	A/B	Juvenile	Adult		coffin	36
1990	5155	A	Infant	0	pnb	coffin, letter 'H' & no. '4'	36
1991	5153	A	Infant	1		coffin	36
1992	7036	A	Male	Adult	sepa; oa; trauma	coffin	36
1993	5160	B/C	Male	35–44	caries; oa; trauma		34
1994	5161	A	Infant	0	pnb	coffin	36
1995	5111	B/C	Male	45+	dentabs; ea; oa		34
1996	5115	A	Male	Adult	caries; dentabs; dish; oa; trauma	coffin	36
1997	5162	A	Infant	0		coffin	36
1998	5154	A	Unknown	Adult	pnb	coffin	36
1999	5167	A/B	Juvenile	6			36
2000	5168	A/B	Male	25–34	caries; trauma		36
2001	5177	A	Infant	0		coffin	36
2002	5169	A	Infant	0		coffin	36
2003	5178	B/C	Female	33			34
2004	5179	A	Infant	0		coffin	36
2005	5180	A	Infant	0		coffin	36
2006	7054	A	Juvenile	11		coffin	36
2007	7055	A	Female	22		coffin	36

2008	7061	A/B	Juvenile	5	caries; co		36
2009	7065	A				coffin, part of sk. 1986	36
2010	7059	A	Male	45+	caries; dentabs; spond; trauma	coffin	36
2011	7061	A/B	Juvenile	5–15	caries; pnb		36
2012	7066	A/B	Unknown	Adult		?coffin	36
2013	7070	B/C	Female	Adult	caries; sinusitis		34
2014	7072	A/B	Unknown	Adult	caries; dentabs	?coffin	36
2015	7073	A	Female	17	caries; dentabs; rickets	coffin	36
2016	7063	A	Male	45+	caries; osdiss; pdb; pnb	coffin, bell	36
2017	7069	C/D	Juvenile	10		?coffin	33
2018	7067	A/B	Unknown	Adult	caries	?coffin	36
2019	7074	A	Male	Adult	trauma	coffin	36
2020	7077	A	Infant	0		coffin	36
2021	7056	A	Infant	1	pnb		36
2022	5182	A	Juvenile	7		coffin, lace ends (2)	36
2023	5166	A	Unknown	Adult		coffin	36
2024	5189	B/C	Infant	0			34
2025	5183	A	Infant	0	bent	coffin	36
2026	5185	A	Male	45+	caries; dish; hfi; pnb	coffin	36
2027	5163	B/C	Male	35–44	oa		34
2028	5187	A	Juvenile	12		coffin	36
2029	5188	A	Infant	1		coffin	36
2030	5181	B/C	Female	25–34	caries; co; dentabs		34
2031	5190	A	Infant	1		coffin	36
2032	5192	A				coffin, coin	36
2033	5193	A	Infant	0		coffin	36
2034	5190	A	Juvenile	8		coffin	36
2035	5189	B/C	Juvenile	6			34
2036	5194	A	Unknown	Adult		coffin, textile	36
2037	5195	A/B	Male	35–44	hfi; oa		36
2038	3843	C/D	Female	25–34	caries	coffin	33
2039	7076	A	Female	Adult	caries; dentabs	coffin	36
2040	7058	A	Male	45+	caries; dentabs; hfi; op	coffin	36
2041	7078	A	Infant	1			36
2042	7079	A	Female	18	caries; dentabs	coffin	36
2043	7053	A	Male	35–44	caries; sn	coffin	36
2044	7083	B/C	Unknown	Adult			34
2045	7080	A	Male	45+	caries; oa	coffin	36
2046	7068	B/C	Male	Adult	oa		34
2047	7081	A	Male	45+	oa	coffin	36
2048	7084	B/C	Juvenile	14			34
2049	7085	A/B	Unknown	Adult		?coffin	36
2050	7086	A/B	Female	Adult	caries; dentabs		36
2051	7089	A	Infant	1		coffin	36
2052	7090	B/C	Female	45+	caries		34
2053	7092	A	Unknown	Adult			36
2054	7440	0	Infant	1	pnb		not on plan
2055	7100	A	Infant	0		coffin	36
2056	7096	B/C	Male	25–34	sn; trauma	coffin	34
2057	7102	A/B	Unknown	Adult			36
2058	7088	A/B	Unknown	Adult			36
2059	7103	B/C	Female	Adult			34
2060	5200	A	Male	35–44	caries; dentabs; eh	coffin	36
2061	5196	A	Infant	0	caries	coffin	36
2062	5197	A	Infant	0		coffin	36
2063	5205	A	Infant	1		coffin	36
2064	5203	C/D	Female	Adult			33
2065	5204	A/B	Infant	0			36
2066	5201	C/D	Male	15		coffin	33
2067	5207	C/D	Male	25–34	trauma		33
2068	5206	A	Unknown	Adult	dentabs	coffin	36
2069	5214	A/B	Male	Adult	pnb		36
2070	5223	B/C	Juvenile	6			34
2071	7094	A	Unknown	Adult			36
2072	7093	A	Male	45+	oa; sn		36
2073	7091	A/B	Female	Adult			36
2074	5215	C/D	Unknown	Adult	oa		33
2075	5213	B/C	Infant	0	pnb		34
2076	5216	B/C	Male	25–34	dentabs; sn		34
2077	5217	D	Female	Adult			32
2078	5218	A/B	Male	Adult	oa		36
2079	5219	A/B	Juvenile	6	co		36
2080	7105	A	Infant	2	co; pnb; trauma		36
2081	7099	A/B	Female	Adult	caries		36

Sk. no.	*Context*	*Phase*	*Sex*	*Age*	*Pathology notes*	*Archaeology notes*	*Fig. no.*
2082	7098	A	Infant	1	co	coffin	36
2083	7107	D/E	Infant	1		charred board/coffin	32
2084	7087	A	Juvenile	7	caries; co	coffin	36
2085	7108	B/C	?Female	Adult			34
2086	7109	B/C	Juvenile	Adult			34
2087	7110	B/C	Unknown	Adult			34
2088	7101	A	Female	Adult	caries; dentabs	coffin, AE ring	36
2089	7111	A	Female	17	caries; spond	coffin	36
2090	5220	A	Infant	0		coffin	36
2091	5221	A	Infant	0		coffin	36
2092	5222	B/C	Unknown	Adult	oa; pnb		34
2093	5224	B/C	Infant	0			34
2094	5225	A	Infant	0		coffin	36
2095	5226	B/C	Male	25–34	caries; sn		34
2096	5228	D	Juvenile	6			32
2097	5202	B/C	Female	25–34	sn; trauma		34
2099	5231	C/D	Infant	1		?coffin	33
2100	7117	B/C	Male	22		?coffin	34
2101	7441	B/C	Juvenile	Adult		?coffin	34
2102	7442	B	Unknown	Adult	stt	?coffin	34
2103	7443	B/C	?Female	Adult		coffin	34
2104	7444	A/B	Infant	0		coffin	36
2105	7445	A/B	Infant	1			36
2106	7113	A	Infant	1		coffin	36
2107	7114	A	Infant	2		coffin	36
2108	7446	B/C	Male	Adult	spond		34
2109	7447	B/C	Female	Adult		?coffin	34
2110	7448	B/C	Male	45+	dentabs; sn		34
2111	7449	B/C	Unknown	Adult		?coffin	34
2112	7450	B/C	Unknown	Adult			34
2113	7451	B/C	Male	45+			34
2114	7116	A	Juvenile	9		coffin	36
2115	7101	A	Female	Adult	dish; oa	coffin	36
2116	7119	A	Female	Adult	hfi; op	coffin	36
2117	7452	B/C	Female	Adult	caries; dentabs; oa		34
2118	7453	A/B	Infant	0			36
2119	7454	B/C	Female	17			34
2120	7455	B/C	Unknown	Adult			34
2121	7456	B/C	Juvenile	12	sn		34
2122	7457	B/C	Unknown	Adult	pnb		34
2123	7106	A	Juvenile	12	co; eh	coffin (?gabled)	36
2124	7458	A/B	Male	15			36
2125	7459	B/C	Infant	3			34
2126	7460	A/B	Infant	0			36
2127	7045	A	Female	Adult		coffin	36
2128	7461	B/C	Juvenile	7			34
2129	7120	A	Unknown	Adult		coffin, coin	36
2130	5235	B/C	Female	Adult	sn		34
2131	5236	C/D	Male	Adult			33
2132	5237	C/D	Infant	1			33
2133	5238	B/C	Juvenile	9	caries; co		34
2134	5240	A/B	Male	Adult			36
2135	5242	A	Male	35–44	dentabs; sn; spond		36
2136	5241	A/B	Juvenile	10			36
2137	5245	D	Infant	0	caries		32
2138	5246	D	Infant	2	co		32
2139	5246	D	Male	Adult	pnb; spond; oa; sn; trauma	BH 53, BH 53A	32
2140	7125	A	Female	Adult	op; spond; oa; trauma	coffin	36
2141	7462	0	Unknown	Adult			not on plan
2142	7128	B/C	Female	Adult	caries; dentabs; oa		34
2143	7463	B/C	Juvenile	5			34
2144	7123	A	Infant	1		coffin	36
2145	7121	B/C	Female	25–34	caries; dentabs	with sk. 2146 and ?2147	34, 43
2146	7121	B/C	Fetus	0			34
2147	7121	B/C	Infant	0			34
2148	7129	C/D	Male	25–34	caries		33
2149	7464	A/B	Juvenile	5		coffin	36
2150	7465	B/C	Unknown	Adult			34
2151	7466	B/C	Unknown	Adult			34
2152	7467	0	Unknown	Adult			not on plan
2153	7131	B/C	Male	Adult	pnb rib		34
2154	7468	B/C	Unknown	Adult		?coffin	34
2155	7469	A/B	Unknown	Adult		?coffin	36

2156	7133	B/C	Female	Adult	osdiss; sn	?coffin	34
2157	7470	B/C	Male	Adult			34
2158	7134	C/D	Male	45+	pdb; spond; oa; trauma		33
2159	7471	B/C	Juvenile	12	co; om		34, 64
2160	7135	B/C	?Female	Adult	oa; trauma		34
2161	7136	B/C	Male	25–34	dentabs; osdiss		34
2162	7472	B/C	Unknown	Adult	pnb	?coffin	34
2163	7137	A	Female	Adult	caries; co; dentabs; pnb	coffin	36
2164	7138	B/C	Juvenile	8			34
2165	7473	C/D	Male	Adult	dish; oa		33
2166	7474	A	Male	35–44	caries; dentabs; oa		36
2167	7475	B/C	Unknown	Adult			34
2168	7476	B/C	Juvenile	10			34
2169	7477	0	Female	25–34	co; sepa; sn		65
2170	5247	B/C	Female	25–34	co; hfi		34
2171	5248	D	Juvenile	7			32
2172	5249	C/D	Infant	1			33
2173	5250	C/D	Female	25–34			33
2174	5251	B/C	Female	Adult			34
2175	5253	B/C	Male	Adult			34
2176	5254	D	Female	45+	caries; dentabs		32
2177	5478	D	Infant	1			32
2178	7126	B/C	Female	20	co; sn		34
2179	7478	B/C	Unknown	Adult			34
2180	5256	B/C	Unknown	Adult	gout		34
2181	5257	D	Juvenile	13			32
2182	5258	A	Infant	0		coffin	36
2183	5261	D	Juvenile	5			32
2184	5263	D	Male	Adult			32
2185	5264	A/B	Unknown	Adult	osdiss		36
2186	5285	B/C	Juvenile	12	co		34
2187	5274	D	Female	45+	pnb		32
2188	5275	D	Female	Adult			32
2189	5277	D	Female	Adult			32
2190	7479	B/C	Male	Adult			34
2191	7480	B/C	Infant	0			34
2192	7481	B/C	Infant	0			34
2193	7140	C/D	Male	35–44	pnb rib; oa; trauma		33
2194	7142	B/C	Unknown	Adult			34
2195	7482	B/C	Male	Adult	caries		34
2196	7139	B/C	Male	45+	head wnd; spond		34
2197	7483	B/C	Juvenile	9			34
2198	7144	B/C	Infant	3	co	coffin	34
2199	7484	B/C	Juvenile	8			34
2200	7485	B/C	Male	45+	caries; sn		34
2201	7486	B/C	Female	Adult	trauma		34
2202	7487	0	Unknown	Adult			not on plan
2203	7488	0	Male	45+	oa; osdiss; pnb		not on plan
2204	7489	B/C	Male	25–34	caries; hfi; oa		34
2205	7145	0	Juvenile	Adult			not on plan
2206	7490	B/C	Male	45+			34
2207	7148	B/C	?Male	Adult			34
2208	7491	B/C	Unknown	Adult			34
2209	7492	B/C	Male	25–34	spond; trauma		34
2210	5282	D	Male	35–44	caries; dentabs; stt	ear muffs (2)	32, 82
2211	5252	D	Female	25–34	caries; dentabs; hfi	AE band	32
2212	7493	B/C	Unknown	Adult			34
2213	5271	D	Juvenile	Adult	sn		32
2214	7494	B/C	Infant	2			34
2215	5288	D	Male	Adult			32
2216	5293	D	Female	45+	caries; oa; pnb		32
2217	5294	B/C	?Male	45+			34
2218	5304	B	Infant	2			34
2219	5318	D	Male	35–44	oa; trauma		32
2220	7495	B/C	Male	Adult			34
2221	7152	B/C	Infant	0		coffin	34
2222	7496	C/D	Female	20	caries; spond		33, 97
2223	7497	B/C	Unknown	Adult			34
2224	7156	B/C	Female	Adult	caries		34
2225	7498	A/B	Juvenile	10			36
2226	7499	C/D	Male	25–34	eh		33
2227	4870	B/C	Female	Adult	spond		34
2230	4871	C/D	Infant	0			33
2231	4872	B/C	Male	Adult	co		34

Sk. no.	*Context*	*Phase*	*Sex*	*Age*	*Pathology notes*	*Archaeology notes*	*Fig. no.*
2232	4873	B/C	Juvenile	5			34
2233	7160	C/D	Juvenile	12	co; sn		33
2234	4874	B/C	Female	Adult			34
2235	4875	B/C	Unknown	Adult			34
2236	4876	B	Female	Adult	op		34
2237	7163	B/C	Female	Adult	oa	coffin	34
2238	4877	C/D	Male	25–34		BH 80	33
2239	4878	C/D	Female	25–34			33
2240	7166	C/D	Infant	0			33
2241	4879	B/C	?Female	Adult	op		34
2242	7161	D	Male	22	bent; sn	BH 56	32
2243	4880	C	Female	25–34		BH 81	33
2244	7166	C/D	Male	35–44	spond		33
2245	4881	B/C	Female	Adult			34
2246	4882	C/D	Unknown	Adult	oa		33
2247	7097	C/D	Infant	2	rickets	?coffin	33
2248	7167	C/D	Male	35–44	oa; pdb		33
2249	4883	A/B	Infant	1			36
2250	7168	C/D	Juvenile	10	pnb		33
2251	7154	B/C	Male	45+	dish; oa; om; trauma		34
2252	7165	B/C	Male	22	caries; om; pnb; trauma	coffin	34
2253	4884	A/B	Infant	2			36
2254	4885	A/B	Infant	1			36
2255	7172	C/D	Male	Adult			33
2256	7173	D/E	Juvenile	7		?coffin	32
2257	4886	B/C				may not have survived lifting	34
2258	4887	C/D	Female	Adult			33
2259	7204	D/E	Female	Adult	co; oa	BH 57	32
2260	7174	C/D	Female	Adult		?coffin	33
2261	7176	E	Unknown	Adult	dentabs		30
2262	7178	B/C	Juvenile	7	co		34
2263	4888	C/D	Unknown	Adult	pnb		33
2264	4889	C/D	Unknown	Adult			33
2265	4890	B/C	Unknown	Adult			34
2266	4891	B/C	Male	Adult			34
2267	7179	B/C	Unknown	Adult			34
2268	4892	C/D	Male	Adult	co; dentabs		33
2269	7185	C/D	Juvenile	7	co	?coffin	33
2270	4893	C/D	Female	Adult			33
2271	7182	A	Female	20	caries	coffin	36
2272	4894	C/D	Female	Adult			33
2273	7184	D/E	Female	Adult	caries; oa; pnb	coffin	32
2274	7183	B/C	Male	45+	caries; dish; oa; sinusitis; sn; trauma	coffin	34, 69
2275	4895	C/D	Female	Adult		?coffin	33
2276	7186	C/D	Male	15			33
2277	7187	C/D	Female	Adult		coffin	33
2278	4896	B/C	Male	Adult			34
2279	7189	B/C	Male	25–34	caries; co; sn; trauma		34
2280	7188	C/D	Juvenile	Adult			33
2281	7190	B/C	Female	25–34	caries; oa		34
2282	4897	D/E	Male	Adult	oa; sn	BH 51	32
2283	7196	C/D	Female	45+	oa; trauma	?coffin	33
2284	7197	D/E	Female	45+	caries; trauma		32
2285	4898	A/B	Infant	4			36
2286	7198	B/C	Infant	4			34
2287	7201	B/C	Infant	2			34
2288	7203	B/C	Female	Adult			34
2289	7202	D/E	Male	35–44	dish; pnb		32
2290	5321	E	Male	Adult			30
2291	5296	C	Female	25–34	caries; sn		33
2292	5322	E	Unknown	Adult	trauma		30
2293	5323	E	Unknown	Adult	pnb		30
2294	5327	C	Male	45+	caries; sn		33
2295	5325	C/D	Female	25–34	caries		33
2296	5324	C	Female	20	caries; dentabs; sn		33
2297	5311	D/E	Male	45+	caries; dentabs; oa; trauma	coffin(T), dendro	32
2298	5316	D/E	Male	45+	trauma	coffin, ear muffs (2)	32
2299	5330	C/D	Infant	0			33
2300	7225	C/D	Juvenile	12			33
2301	7226	C/D	Male	Adult	dish		33
2302	4977	D/E	Juvenile	7	head wnd	coffin	32
2303	4977	D/E	Unknown	Adult	oa		32
2304	4901	C/D	Male	45+		BH 52	33

2305	7214	B/C	Female	Adult			34
2306	4902	B/C	Unknown	Adult	oa		34
2307	7211	B/C	Male	Adult			34
2308	7210	C/D	Female	Adult			33
2309	5326	E	Male	45+	caries; oa; sn	coffin	30
2310	7215	C/D	Unknown	Adult	pdb		33
2311	4900	C/D	Infant	0			33
2312	7216	B/C	Unknown	Adult			34
2313	7217	B/C	Female	Adult			34
2314	7219	C/D	Unknown	Adult	oa		33
2315	4903	B/C	Unknown	Adult			34
2316	4904	B/C	Unknown	Adult			34
2317	4905	B/C	Juvenile	6			34
2318	4906	B/C	Juvenile	15			34
2319	4907	C/D	Juvenile	Adult			33
2320	5317	D/E	Male	45+	caries; oa; osdiss; pnb	coffin	32
2321	5331	C	Juvenile	7	co;trauma		33
2322	5328	E	Male	25–34	dish; sn; spond	coffin(T), dendro, AE strap-end	30
2323	5332	C	Infant	0	pnb		33
2324	7227	C/D	Female	Adult	pnb	?coffin	33
2325	5338	C	Infant	1			33
2326	5339	D	Infant	1			32
2327	5310	B/C	Male	45+	spond		34
2328	5341	C	Juvenile	6	co		33
2329	5353	C/D	Infant	1			33
2330	4908	E	Female	25–34			30
2331	4909	B/C	Unknown	Adult			34
2332	4910	A/B	Unknown	Adult	co; sn		36
2333	7221	B/C	Female	Adult	caries; trauma	coffin	34
2334	7222	B/C	Unknown	Adult			34
2335	7218	D/E	?Female	Adult		coffin	32
2336	7223	D/E	Juvenile	6		pot	32
2337	7224	D/E	Male	45+		coffin	32
2338	4911	C/D	Male	45+	oa; spond		33
2339	4912	B/C	Infant	1			34
2340	7234	D/E	Male	20	caries; sfe; sn		32
2341	7228	B/C	?Male	Adult			34
2342	7229	D/E	Infant	2		coffin	32
2343	7209	C/D	Female	25–34			33
2344	7230	C/D	Juvenile	13	sn		33
2345	7233	C/D	Male	35–44			33
2346	4913	D/E	Infant	1			32
2347	4914	C/D	Female	25–34	caries; hfi		33
2348	5335	C/D	Female	Adult		BH 54	33
2349	5334	B/C	Infant	1			34
2350	4915	A	Unknown	Adult			36
2351	7232	C/D	Male	45+	co; dentabs		33
2352	7220	C/D	Female	25–34	dentabs; head wnd; oa	BH 79	33
2353	4916	C/D	Female	25–34	caries; dentabs		33
2354	4917	C/D	Male	Adult			33
2355	4918	C/D				part of sk. 2541.	33
2356	7235	C/D	Female	45+	caries; sn; spond		33
2357	7236	A/B	Juvenile	5			36
2358	4919	C/D	Unknown	Adult		?coffin	33
2359	7231	B/C	Male	25–34	head wnd; sn		34
2360	7231	B/C	Infant	0		coin	34
2361	4920	C/D	Male	25–34		?coffin	33
2362	4921	C/D	Juvenile	10			33
2363	7237	C/D	Juvenile	7			33
2364	4922	B/C	Unknown	20			34
2365	7239	C/D	Unknown	Adult			33
2366	4923	B/C	Juvenile	Adult			34
2367	4924	B/C	Juvenile	13			34
2368	4925	B/C	Infant	3			34
2369	7229	D/E	Juvenile	6		coffin	32
2370	4926	A/B	Male	18			36
2371	7243	C/D	Female	25–34			33
2372	7243	C/D	Juvenile	Adult			33
2373	4927	0	Infant	0			not on plan
2374	7244	C/D	Infant	1½–2			33
2375	4928	C/D	Juvenile	12			33
2376	7245	D/E	Male	45+	caries; co; dentabs; spond; trauma	?coffin, pot	32
2377	7245	D/E	Infant	1	pnb		32
2378	7246	C/D	Male	25–34		buckles (2)	33

Sk. no.	*Context*	*Phase*	*Sex*	*Age*	*Pathology notes*	*Archaeology notes*	*Fig. no.*
2379	7247	B/C	Male	16	pnb; sn		34
2380	5355	C/D	Infant	0			33
2381	5356	E	Unknown	Adult			30
2382	5366	E	Male	25–34	caries; trauma		30
2383	5367	C	Unknown	Adult	co; pnb		33
2384	5362	E	Female	17			30
2385	5363	E	Infant	1	rickets	?coffin	30
2386	5361	E	Female	45+	oa; trauma		30
2387	7262	D/E	Juvenile	7		?coffin	32
2388	7249	D/E	Male	25–34		coffin	32
2389	4929	E	Male	Adult	scoliosis		30, 94
2390	4930	C/D	Unknown	Adult			33
2391	7248	C/D	Infant	1		pot	33
2392	7253	D/E	?Male	Adult		coffin	32
2393	7253	D/E	Infant	0		coffin	32
2394	7256	E	Female	25–34		roves (2 rows)	30
2395	7254	C/D	Infant	1			33
2396	4931	C/D	Unknown	Adult			33
2397	7255	C/D	Juvenile	13	co		33
2398	4932	B/C	Unknown	13			34
2399	7258	E	Infant	0			30
2400	7259	C/D	Infant	1		pot	33
2401	7414	D/E	Female	25–34			32
2402	7265	E	Male	45+	hfi	roves (3), ?coffin (silt round bones)	30
2403	4933	C/D	Infant	4			33
2404	7268	C/D	Unknown	Adult		coffin	33
2405	7270	B/C	Female	Adult			34
2406	4934	B/C	Unknown	Adult	hfi		34
2407	7272	C/D	Unknown	Adult		coffin	33
2408	7273	D/E	Female	45+			32
2409	7264	D/E	Female	25–34		coffin	32
2410	7258	E	Male	25–34	caries; sn	coffin	30
2411	7267	E	Male	45+	hfi; oa; trauma		30
2412	4935	D/E	Infant	1			32
2413	7271	E	Male	45+	caries; oa	coffin	30
2414	7279	E	Juvenile	Adult		coffin	30
2415	4936	B/C	Female	Adult			34
2416	7277	D/E	Female	Adult	hfi	coffin, ear muff(1)	32
2417	7278	D	Female	Adult		BH 70	32
2418	4937	B/C	Female	15	pnb		34
2419	7281	E	Female	15		roves(3)	30
2420	5379	C	Infant	2		chalk lumps over head & feet	33
2421	5364	E	Infant	1	caries	coffin	30
2422	5382	C/D	Female	25–34	caries; dentabs	BH 55	33
2424	5385	C/D	Juvenile	8	co		33
2425	5385	C/D	Infant	2	co		33
2426	5388	D/E	Male	25–34		a re-burial, AE object	not on plan
2427	5389	E	Female	17	eh; ?tb	coffin	30
2428	3884	E	Female	35–44			30
2429	5393	E	Male	Adult		?coffin, BH 19	30
2430	7283	D/E	Juvenile	6		coffin	32
2431	7274	D/E	Unknown	Adult		coffin	32
2432	7287	E	Male	Adult			30
2433	7282	E	Unknown	25–34		coffin (roves in fill ?from G7256)	30
2434	7288	D/E	Infant	1		coffin, layer of mortar below coffin	32
2435	7289	D/E	Juvenile	5	co; eh	coffin, pot	32
2436	7290	C/D	Male	45+	oa	BH 82	33
2437	7293	E	Infant	1		?coffin	30
2438	7263	D/E	Female	25–34	caries; sn	?coffin, slab cover & disartic. bones	32
2439	7294	C/D	Infant	1			33
2440	7276	D/E	Infant	0		?coffin (displaced bones), ?roves	32
2441	7295	D/E	Male	Adult	sn		32
2442	7296	A/B	Infant	0		?coffin	36
2443	7297	D/E	Unknown	Adult		coffin	32
2444	4938	B/C	Juvenile	11			34
2445	7305	C/D	Infant	0	pnb		33
2446	7306	D/E	Unknown	Adult			32
2447	4939	D/E	Infant	0			32
2448	7310	D/E	Infant	0		coffin	32
2449	7238	0	Male	15			not on plan
2450	7311	E	Male	45+	oa	ear muffs (2)	30
2451	7314	D/E	?Male	35–44	dentabs	coffin	32
2452	7313	C/D	Male	45+	oa	coffin	33

2453	7315	D/E	Female	Adult		?coffin	32
2454	7269	E	Female	17		?coffin, pillow stone (1), same as sk. 2483	30
2455	7316	D/E	Infant	0			32
2456	7317	D/E	Juvenile	5			32
2457	7319	E	Female	45+	caries; hfi	?coffin (large grave), ?rove (stray?)	30
2458	7299	D/E	Female	45+	hfi	coffin, ?rove (1)	32
2459	7322	D/E	Unknown	Adult			32
2460	5404	C/D	Infant	0	co		33
2461	4940	0	Unknown	Adult			not on plan
2462	4941	D/E	Unknown	Adult			32
2463	7323	D/E	Infant	2		coffin	32
2464	7324	C/D	Unknown	Adult			33
2465	7321	E	Female	45+		coin, BH 58	30
2466	7327	E	Male	45+	caries; oa	roves (19), animal bones on feet	30
2467	7298	C/D	Unknown	Adult			33
2468	7298	C/D	?Female	Adult			33
2469	7328	E	Male	Adult		coffin, pot (Roman)	30
2470	5357	E	Female	25–34	co	coffin(T), dendro	30
2471	5402	E	Male	45+	caries; dentabs	coffin(T), wand (1), dendro, organic?	30
2472	5416	D	Female	25–34		coffin	32
2473	5418	D	Female	25–34		coffin	32
2474	5419	D	Juvenile	12		coffin	32
2475	5421	D		Adult	oa		32
2476	5420	D	Female	45+		?coffin	32
2477	7360	D/E	Male	12		coffin	32
2478	5425	E	Male	45+	dentabs	coffin(T), dendro	30
2479	5436	D	Female	25–34		?coffin	32
2480	7329	D/E	Infant	1			32
2481	7333	D/E	Infant	2			32
2482	7335	D/E	Infant	1			32
2484	7337	C/D	Male	Adult		coffin	33
2485	7338	D/E	Male	35–44	oa	bronze object	32
2486	7340	D/E	Male	Adult		?coffin, BH 59	32
2487	7339	D/E	Unknown	Adult		?coffin	32
2488	7334	E	Female	Adult		coffin, pot (Saxon)	30
2489	7326	D/E	Unknown	17		?coffin, ear muffs (2)	32
2490	4942	C/D	Infant	1			33
2491	4943	B/C	Male	45+	dish; oa; trauma		34
2492	4944	B/C	Juvenile	7			34
2493	7206	A	Unknown	Adult		coffin	36
2494	4945	C/D	Female	25–34	co; discitis; sn		33
2495	7207	E	Male	45+	oa; sn		30
2496	4946	B/C	Unknown	Adult			34
2497	4947	B/C	Infant	3			34
2498	7208	D/E	Female	25–34		coffin	32
2499	7212	C/D	Male	35–44	oa	half coin, BH 83	33
2500	7346	C/D	Unknown	Adult			33
2501	7348	D/E	Male	Adult	trauma	BH 18	30
2502	7341	E	Unknown	45+		coffin	30
2503	7343	D/E	Female	45+	caries; dentabs; pnb; stt		32
2504	7344	E	Unknown	Adult	oa; osdiss	roves (1 row)	30
2505	7349	D/E	Juvenile	8	co		32
2506	7350	D/E	Juvenile	6		?coffin	32
2507	4948	B/C	Male	20			34
2508	4949	B/C	Male	Adult	oa		34
2509	7351	C/D	Female	Adult		BH 71	33
2510	7320	D/E	Infant	1			32
2511	4950	D/E	Infant	0			32
2512	7354	D/E	Juvenile	6		?coffin	32
2513	7355	D/E	Female	25–34	dentabs	?coffin	32
2514	7331	D/E	Male	45+	dentabs	coffin	32
2515	4951	D/E	Infant	0			32
2516	7356	D/E	Infant	0		charred board/coffin?	32
2517	7357	D/E	Male	25–34			32
2518	7342	D/E	Male	Adult	caries		32
2519	7358	E	Male	25–34		coffin	30
2520	7353	E	Male	50+	caries; dentabs; oa; sn	coffin, ?rove (1 – stray?)	30
2521	7359	E	Female	45+		coffin	30
2522	7361	D/E	Unknown	Adult			32
2523	7200	A	Male	Adult		coffin	36
2524	4952	D/E	Infant	1			32
2525	7365	D/E	Infant	1	co	?coffin	32
2526	7366	D/E	Juvenile	6		coffin	32
2527	7370	D/E	Unknown	Adult		coffin	32

Sk. no.	*Context*	*Phase*	*Sex*	*Age*	*Pathology notes*	*Archaeology notes*	*Fig. no.*
2528	7367	D/E	?Male	Adult	dentabs	?coffin	32
2529	7368	D/E	Female	25–34		?coffin	32
2530	4953	D/E	Juvenile	8			32
2531	4954	E	Infant	4			30
2532	7373	E	Male	25–34	oa	coffin	30
2533	4955	B/C	Unknown	Adult		?coffin	34
2534	4956	B/C	Male	Adult	oa	?coffin	34
2535	4957	B/C	Unknown	Adult		?coffin	34
2536	7377	D/E	Male	25–34	caries; co; dentabs; oa; sn	coffin, ear muffs (2)	32
2537	4958	0	Unknown	Adult	oa		not on plan
2538	7371	E	Male	25–34	co; trauma	coffin	30
2539	7380	D/E	Male	Adult	caries; stt		32
2540	7379	D/E	Female	Adult		?coffin	32
2541	7378	C/D	Juvenile	13			33
2542	4959	E	Unknown	Adult			30
2543	4960	C/D	Unknown	Adult			33
2544	4961	D/E	Unknown	Adult			32
2545	7382	E	Female	Adult	hfi	coffin, BH 39	30
2546	7385	D/E	Infant	1			32
2547	7387	D/E	Juvenile	9		pot	32
2548	7417	D/E	?Male	Adult	oa		32
2549	7376	E	Male	Adult		?coffin	30
2550	5438	D	Juvenile	7	co	coffin	32
2551	5439	D	Juvenile	10	pnb; trauma		32, 78
2552	7393	D/E	Female	17		roves (39)	32
2553	7393	D/E	Infant	0			32
2554	7409	C/D	Infant	0			33
2555	4962	B/C	Infant	4			34
2556	7393	D/E	Unknown	Adult	sn	coffin	32
2557	7415	E	Female	25–34	caries; pnb		30
2558	4963	B/C	Female	17	caries		34
2559	7416	E	Male	17		?coffin	30
2560	4964	B/C	Unknown	Adult			34
2561	7392	D/E	Infant	0			32
2562	4965	E	Juvenile	5			30
2563	7391	D/E	Unknown	Adult			32
2564	7390	D/E	Infant	2			32
2565	7388	D/E	Juvenile	7		coffin	32
2566	7384	E	Unknown	Adult		coffin	30
2567	4966	E	Male	Adult	oa		30
2568	7397	E	Infant	0			30
2569	7398	E	Male	45+	caries	coffin, BH40	30
2570	7362	E	Male	45+	caries; oa; sn	coffin	30
2571	7402	E	Infant	1			30
2572	4967	D/E	Female	Adult			32
2573	4968	E	Female	45+	hfi; oa		30
2574	7405	E	Female	Adult	spond; oa	ear muffs (2)	30
2575	4969	C/D	Female	Adult			33
2576	4970	E	?Female	Adult	oa	BH 90	30
2577	7406	E	Male	Adult	caries; sepa; oa; trauma	coffin	30
2578	7408	E	Infant	0			30
2579	7399	D/E	Infant	1			32
2580	7410	E	Male	25–34		?coffin (silt), roves (3)	30
2581	7412	E	Female	25–34		coffin	30
2582	7407	D/E	Juvenile	5		coffin	32
2583	7395	E	Male	45+	caries; dentabs; disloc; oa; sn	coffin	30
2584	4971	C/D	Unknown	Adult			33
2585	4972	A/B	Male	45+	caries; dentabs	?coffin	36
2586	4973	B/C	Unknown	Adult		?coffin	34
2587	7418	D/E				may not have survived washing	32
2588	4974	A	Infant	1		coffin	36
2589	4975	B/C	Juvenile	8			34
2590	7419	E	Male	17			30
2591	4976	0	Infant	1		coffin, skeleton missing	not on plan
2592	7268	C/D	Male	Adult	oa		33
2593	4978	E	Female	Adult	caries; hfi; trauma		30
2594	7420	E	Male	45+	discitis; sn		30
2595	4979	B/C	Male	45+	caries; co; dentabs; hfi; oa	?coffin	34
2596	4980	C/D	?Male	Adult		?coffin	33
2597	4981	B/C	Unknown	Adult		?coffin	34
2598	4982	B/C	Male	15		?coffin	34
2599	4983	B/C	Male	Adult			34
2600	7421	E	?Male	Adult	oa		30

2601	7422	E	Unknown	Adult			30
2602	7423	D/E	?Male	25–34		?coffin	32
2603	4984	B/C	Male	45+	oa; trauma		34
2604	4985	C/D	Unknown	Adult	trauma	?coffin	33
2605	4986	B/C	Infant	1			34
2606	4987	A/B	Infant	4			36
2607	4988	B/C	Unknown	Adult		?coffin	34
2608	4989	C/D	Juvenile	5			33
2609	7426	E	Unknown	Adult	hfi		30
2610	7435	D/E	Male	Adult			32
2611	7427	D/E	Unknown	Adult		roves (3 rows)	not on plan
2612	3984	D/E	Male	45+	co	grave shaped around head	32
2613	7429	E	Female	25–34	caries	coffin, pillow stones (4)	30
2614	7428	E	Unknown	Adult	osdiss		30
2615	4991	0	Juvenile	Adult		skeleton missing	not on plan
2616	5473	E	Male	35–44	dentabs; oa; trauma	coffin(T), dendro	30
2617	7431	D/E	?Male	25–34		coffin	32
2618	4992	E	Unknown	Adult			30
2619	7432	E	Male	Adult		coffin	30
2620	7433	E	Unknown	25–34		coffin, ?pillow stone (1)	30
2621	7436	E				coffin	30
2622	7437	E	Infant	1		coffin, pot	30
2623	5474	D/E	Juvenile			coffin(T), BH 13 (dendro), bones now missing	16–17, 32
2624	5475	E	Female	45+	dentabs; oa; pnb; sn	coffin(T), wand(1), dendro	16, 30
2625	7529	A/B	Female	Adult			36
2626	7511	A	Male	Adult		coffin	36
2627	7640	A	Male	45+	caries; dentabs; eh; pnb rib; sn; trauma		36
2628	7509	A	Male	45+	pnb; oa; trauma	coffin, coin	36
2629	7505	A	Female	Adult	hfi; oa	coffin	36
2630	7516	A				coffin, bones may not have survived washing	36
2631	7513	A	Female	Adult	caries	coffin	36
2632	7517	A	Female	45+	oa	coffin	36
2633	7641	A	Female	Adult	oa	coffin	36
2634	7520	A	Female	Adult	hfi	coffin	36
2635	7642	A/B	Infant	1			36
2636	7522	A	Female	Adult	caries	coffin	36
2637	7523	A	Unknown	Adult		coffin	36
2638	7524	A	Male	45+	dish; oa	coffin	36
2639	7643	A/B	Female	Adult	oa; op		36
2640	7644	A/B	Male	Adult	oa		36
2641	7525	A	Unknown	13	caries	coffin	36
2642	7645	A/B	Unknown	Adult			36
2643	7646	B/C	Unknown	Adult			34
2644	7647	A/B	Unknown	Adult	pnb	coffin	36
2645	7648	A	Unknown	Adult	pnb; trauma		36
2646	7649	A	Unknown	Adult	oa		36
2647	7650	A/B	Juvenile	12	caries		36
2648	7651	A/B	Unknown	Adult			36
2649	7527	A	Female	25–34		coffin	36
2650	7652	B/C	Juvenile	12	co		34
2651	7653	B/C	Female	Adult			34
2652	7654	A/B	Male	Adult			36
2653	7655	B/C	Female	Adult			34
2654	7656	A/B	Unknown	Adult			36
2655	7657	A/B	Unknown	Adult	sn		36
2656	7658	A	Unknown	Adult			36
2657	7659	A	Unknown	Adult			36
2658	7660	A/B	Unknown	Adult	trauma		36
2659	7661	A/B	Unknown	Adult			36
2660	7662	A/B	Juvenile	9			36
2661	7531	A/B	Male	45+	caries		36
2662	7663	A/B	Juvenile	13			36
2663	7532	A/B	Female	Adult	pnb		36
2664	7664	B/C	Unknown	Adult			34
2665	7665	B/C	Unknown	Adult	pnb		34
2666	7666	B/C	Female	Adult			34
2667	7533	A/B	Male	25–34			36
2668	7534	A/B	Male	45+	caries; sinusitis; sn		36
2669	7535	B/C	Male	25–34	trauma		34
2670	7667	B/C	Juvenile	7			34
2671	7668	A/B	Juvenile	13			36

Sk. no.	*Context*	*Phase*	*Sex*	*Age*	*Pathology notes*	*Archaeology notes*	*Fig. no.*
2673	7537	A/B	Juvenile	14			36
2674	7536	B/C					34
2675	7669	B/C	Unknown	Adult		?coffin	34
2676	7670	B/C	Female	45+	oa	?coffin	34
2677	7671	B/C	Unknown	Adult			34
2678	7672	B/C	Infant	0			34
2679	7526	B/C	Infant	3			34
2680	7673	B/C	Juvenile	13			34
2681	7674	B/C	Juvenile	5			34
2682	7539	B/C				may not have survived washing.	34
2683	7528	B/C	Female	17			34
2684	7528	B/C	Male	45+	caries; oa		34
2685	7675	B/C	Juvenile	9			34
2686	7676	B/C	Female	Adult			34
2687	7677	A	Female	Adult			36
2688	7678	B/C	Juvenile	13			34
2689	7679	A/B	Unknown	Adult			36
2690	7544	B/C	Juvenile	13	pnb		34
2691	7541	B/C	Female	Adult	caries; oa		34
2692	7543	A/B	Female	25–34	caries; dentabs; discitis; oa; pnb; spond; trauma		36
2693	7680	A/B	Juvenile	12	sfe		36
2694	7681	B/C	Juvenile	14	stt		34
2695	7545	B/C	Juvenile	11			34
2696	7540	C/D	Female	Adult	trauma	coffin	33
2697	7682	B/C	Juvenile	7			34
2698	7683	A/B	Unknown	Adult			36
2699	7547	B/C	Male	Adult	dish		34
2700	7552	B/C	?Female	45+	caries; pnb; oa; trauma		34
2701A	7551	C/D	Female	25–34		coffin, BH 60	33
2701B	7551	C/D	Infant	0		coffin	33
2702	7548	C/D	Juvenile	15		coffin	33
2703	7684	B/C	Juvenile	Adult			34
2704	7685	B/C	Infant	0			34
2705	7557	B/C	Female	45+			34
2706	7558	B/C	Unknown	45+	caries; dentabs; dish; oa		34
2707	7686	B/C	Unknown	Adult			34
2708	7559	C/D	Female	Adult		coffin	33
2709	7687	B/C	Male	Adult			34
2710	7688	B/C	Female	Adult			34
2711	7561	C/D	Female	45+	dentabs; psa; oa; pnb	coffin	33
2712	7689	B/C	Juvenile	12	pnb		34
2713	7562	B/C	Male	Adult			34
2714	7563	C/D	Male	45+	caries; dentabs; oa; dish	coffin	33
2715	7690	C/D	?Female	Adult			33
2716	7691	B/C	Unknown	Adult	trauma		34
2717	7692	B/C	Unknown	Adult			34
2718	7564	B/C	Female	45+	co	coffin	34
2719	7693	C/D	Unknown	Adult			33
2720	7566	C/D	Male	45+	caries; trauma	coffin	33
2721	7567	C/D	Male	50+	oa		33
2722	7586	D/E	Female	Adult			32
2723	7568	C/D	Male	25–34	caries; dentabs; head wnd	coffin (charred)	33
2724	7694	C/D	Unknown	Adult	oa		33
2725	7574	C/D	?Male	15	co		33
2726	7572	D/E	Unknown	Adult	trauma	coffin	32
2727	7571	D/E	Female	45+	caries		32
2728	7573	D/E	Juvenile	8		coffin	32
2729	7581	D/E	Juvenile	8			32
2730	7576	C/D	Male	45+	oa; sn; trauma	coffin	33
2731	7579	D/E	Male	35–44	oa; trauma		32
2732	7577	D/E	Female	45+	oa; trauma		32
2733	7575	D/E	Male	25–34	sn; trauma	?coffin	32
2734	7695	D/E	Infant	2	co		32
2735	7582	D/E	Infant	0		coffin	32
2736	7583	D/E	Male	25–34	caries; pnb	?coffin	32
2737	7696	D/E	Infant	0			32
2738	7697	D/E	Infant	0			32
2739	7584	D/E	Male	45+	caries; pnb; spond	coffin	32
2740	7585	D/E	Unknown	Adult			32
2741	7698	D/E	Juvenile	7	caries; co		32
2742	7588	D/E	Juvenile	6			32
2743	7590	D/E	Juvenile	5	co	?coffin	32

2744	7591	D/E	Infant	2			32
2745	7593	D/E	Female	25–34	spond	BH 61	32
2746	7594	D/E	Female	25–34	dentabs		32
2747	7592	D/E	Male	25–34	pnb	?coffin	32
2748	7595	D/E	Male	Adult			32
2749	7699	D/E	Unknown	Adult			32
2750	7700	D/E	Juvenile	10			32
2751	7589	D/E	Female	45+	caries		32
2752	7596	D/E	Female	Adult	oa	pot	32
2753	7598	D/E	Juvenile	12		coffin, bronze stud	32
2754	7599	D/E	Male	25–34	caries; om; spond		32
2755	7701	C/D	Unknown	Adult	oa		33
2756	7702	D/E	Juvenile	9			32
2757	7601	D/E	Unknown	Adult		coffin	32
2758	7600	D/E	Male	45+	hfi	coffin, glass bead	32
2759	7602	D/E	Male	35–44	dentabs	coffin	32
2760	7703	D/E	Unknown	Adult			32
2761	7606	D/E	Infant	4	caries; co		32
2762	7603	D/E	Juvenile	13		coffin	32
2763	7704	D/E	Unknown	Adult	oa		32
2764	7705	D/E	Male	Adult	oa; trauma		32
2765	7706	D/E	Juvenile	Adult			32
2766	7607	E	Male	Adult		?coffin	30
2767	7608	D/E	Male	Adult	sn	coffin	32
2768	7605	E	?Male	25–34	oa	rove (1)	30
2769	7609	D/E	Female	Adult			32
2770	7610	E	Unknown	Adult	op		30
2771	7611	E	Unknown	35–44	pnb; sn; spond		30
2772	7707	E	?Female	Adult			30
2773	7612	D/E	Male	Adult		coffin	32
2774	7613	E	Male	17		coffin	30
2775	7614	E	Female	45+	caries; dentabs; discitis; oa; spond		30, 83
2776	7615	D/E	Female	25–34	caries	coffin, ear muffs (2)	32
2777	7616	E	Female	17	caries		30
2778	7708	E	Male	Adult			30
2779	7617	D/E	Infant	1		coffin	32
2780	7604	E	Female	Adult			30
2781	7619	D/E	Juvenile	8			32
2782	7620	D/E	Male	45+	co; dentabs	coffin	32
2783	7621	E	Female	Adult		coffin	30
2784	7618	E	Male	45+	sn; spond	coffin, ?roves (2)	30
2785	7624	D/E	Female	25–34	caries; fibdys; om	coffin	32
2786	7625	D/E	Male	45+			32
2787	7709	E	Unknown	Adult			30
2788	7623	E	Unknown	Adult		coffin	30
2789	7626	E	Unknown	25–34	dentabs	coffin, BH 15	30
2790	7627	E	?Male	25–34		coffin, ear muffs (2)	30
2791	7710	D/E	Unknown	Adult			32
2792	7711	D/E	Male	Adult			32
2793	7712	D/E	Juvenile	5			32
2794	7713	E	Infant	0		coffin	30
2795	7630	E	Juvenile	9		coffin	30
2796	7628	E	Unknown	Adult			30
2797	7629	E	Unknown	Adult		?roves (2)	30
2798	7631	D/E	Male	45+	caries; dentabs		32
2799	7632	E	Male	35–44	caries; fibdys; om	pot	30
2800	7634	E	Unknown	Adult		?roves (2)	30
2801	7635	E	Female	25–34	oa		30
2802	7636	E	Infant	2			30
2803	7637	E	Unknown	Adult		?coffin, ?nails/roves	30

Notes to Chapters

Chapter 1 (pp. 1–14)

1. See generally, Bryant 1994. For a gazetteer of archaeological sites and finds in the area, see Loughlin and Miller 1979.
2. For an account of the archaeology of Roman Lincolnshire, see Whitwell 1970. Again, Barton itself receives fuller discussion in Bryant 1994.
3. The archaeology of Anglo-Saxon settlement on Humberside has been catalogued by Eagles (1979), and early Anglo-Saxon evidence in north Lincolnshire has been usefully summarized by Leahy (1993; 1998; 1999). For a general and wide-ranging account of Anglo-Saxon Lincolnshire, see Sawyer 1998.
4. For example, grave 5 was orientated west–east and contained the skeleton of a woman who was laid in a supine position, and was not accompanied by any formal grave-goods. The only associated item was an iron dress-hook, which conveys no religious overtones. Similarly, the female buried in grave 15 was accompanied only by a very plain dress pin and two lace tags. The only implication is that she was interred in simple clothing: her burial was not furnished with grave-goods. A radiocarbon determination on grave 5 yielded a date of Cal. A.D. 600–660 (UB–4643), and on grave 15 of Cal. A.D. 655–695 (UB–4644), both at 95% confidence range.
5. Although not precisely analogous to Barton, the case of Sancton, Yorkshire, is of interest in this context (Faull 1976).
6. The gift was recorded by Bede, *A History of the English Church and People*, bk. IV, chap. 3.
7. The complex evidence has been succinctly summarized and discussed by Bryant (1994, 58–68).
8. There are, however, unresolved problems regarding the origins of St Mary's church. Although there is a record preserved in the Cartulary of Bardney Abbey that it was founded as the chapel of All Saints, around the beginning of the twelfth century, the present structure overlies the foundations of an earlier and undocumented church: that is almost certainly Anglo-Saxon. The evidence is discussed at length in Volume 1.
9. There were also two Domesday-period ferries at Grimsby, and one at Winteringham.
10. Brown (1908) ended his history in 1377, and very little has been written about the town between this date and the late seventeenth century (WEA 1980).
11. This reference occurs in the seventh (1769) edition of Defoe's travels. Although Defoe was writing in the early 1720s, his observations were collected over a period of forty years, and close dating of individual observations is not therefore possible.
12. Thomas Tombleson recorded the following in one of his voluminous notebooks (vol. 5, p. 123); although undated, the relevant notebook was evidently compiled in 1899 (private possession).

1801	St Peter's parish	1,057
	St Mary's parish	679
	Total	1,733

1812	St Peter: males 552; females 676	1,228
	St Mary: males 462; females 514	976
	Total	2,204

1818	St Peter:	1,500
	St Mary:	1,200
	Total	2,700

The last set of figures is clearly only an approximation. Tombleson's labelling of St Peter's and St Mary's as separate 'parishes' is erroneous, although they were popularly so called in the nineteenth century.
13. The census returns chart the population growth of Barton through the nineteenth century as follows: *1801* – 1,709; *1811* – 2,204; *1821* – 2,496; *1831* – 3,233; *1841* – 3,475; *1851* – 3,866; *1861* – 3,797 (decrease); *1871* – 4,332; *1881* – 5,319; *1891* – 5,226 (decrease); *1901* – 5,761.
14. For details of population movement in the mid-nineteenth century, see WEA 1978.
15. See generally, W.E. Varah 1928; G.H. Varah 1965; Bryant 1984 and 1994; Pevsner *et al.* 1989, 121–5.
16. In the Church Terrier of 1730. Its predecessor was described in terriers of, *inter alia*, 1578 and 1622.
17. For the vicars of Barton, see Volume 1; also Brown 1908, appendix II. Churchwardens' accounts for St Mary's survive (partly in transcript) from 1640, but the list of churchwardens is complete only from 1819. However, a list of St Peter's churchwardens survives from 1650–51.
18. The chantry chaplains are listed in Brown 1908, appendix I.
19. Transcripts from lost registers include the following: St Peter's burial register for 1561–62 and 1565–66; baptisms, marriages and burials at St Peter's in 1562–63; and marriages at St Mary's in 1566.

Chapter 2 (pp. 15–32)

1. Eight examples of wooden boxes were identified by various fittings, but these were all grave goods, not receptacles for corpses (Drinkall and Foreman 1998, 296–8).
2. The occurrence of nails was as follows: grave 6 (3 clenched nails); grave 31 (1 nail); grave 38 (2 nails); grave 94 (1 nail); and grave 166B (1 copper-alloy nail).
3. Graves 53 and 187 yielded diamond-shaped roves, which were not recognized as such in the published report.
4. The last recorded tombstone for a new grave plot was for the Rev'd James Knight, in 1867. However, some existing graves continued to receive additional relatives, and the latest for which there is a tombstone record is 1875.
5. The southern churchyard was formally established in 1850, but the earliest surviving tombstone is dated 1849. Some earlier deaths are mentioned on stones commemorating certain individuals, but that does not confirm the whereabouts of the interments.
6. The burial register for St Mary's shows regular interments (several per month) down to October 1855. There was a single further entry in December 1857, presumably an addition into an existing grave-plot. By this time, Nonconformists were already being buried in their own chapel cemeteries.
7. The last entry in St Peter's burial register occurred in December 1867.
8. Subsequent investigation has shown that numerous errors in names and dates occur in the schedule, and the inscriptions have mostly been abbreviated or paraphrased.
9. During post-excavation work it was found logistically necessary to assign notional feature numbers to the undefined graves.

10. These do not include the empty graves under the tower and annexe. The corporeal remains were exhumed from at least 29 graves here in the late Saxon period.
11. Since there was effectively no graveyard to the east of the church, from the thirteenth century onwards, the south side was favoured.
12. A major imponderable affecting the calculations is the proportion of burials which took place in and around St Mary's church. We do not know when interment began there, it being only a chapel-of-ease to St Peter's. Nor is there any reliable information regarding the respective catchment areas of the two churches prior to the eighteenth century.
13. This contrasts markedly with the Roman period, for example, when nails were extremely commonplace. The use of pegs and other forms of fixing, to save on nails, is a phenomenon which has continued into modern times in communities which do not have ready access to a supply of iron. Thus in the Channel Islands, nails were rarely used in construction work before the mid-eighteenth century.
14. The suggestions advanced in Rodwell and Rodwell 1982, 291, were misinformed and should now be disregarded.
15. Graves F4019, 4040, 4064, 4067, 4100, 4106 and 4152.
16. The exhumed graves are referred to only by their feature numbers. There are no skeleton numbers associated with these, and therefore they do not appear in the burial statistics cited in this volume.
17. The chancel could count as an appendage, especially if there were no lateral *porticus* which could receive interments.

Chapter 3 (pp. 33–39)

1. Registration of births and deaths in England and Wales became compulsory following the passage of the *Births and Deaths Act, 1836*; the Act also established the office of the Registrar General. Registration of marriages was required by the *Marriage Act* of the same year. Registration began on 1 July 1837, and from then on the annual reports of the Registrar General provide data that can be used for calculating birth and death rates. Earlier records, such as parish records and the *London Bills of Mortality* provide numbers of burials and deaths, in the case of the former, and deaths in the case of the latter. These sources cannot be used to calculate rates, however, unless the size of the living population can be estimated from other sources.
2. The total number of burials recorded archaeologically was slightly in excess of 2,800, but some skeletons were not lifted and others were so decayed that no useful bone survived.
3. The crude death rate in England and Wales during the eighteenth century has been estimated to have been *c.* 25 per thousand (Woods 2000, 17–18). The median number of burials at St Peter's per year during the period covered by the parish records was 22. This suggests that it is reasonable to use the crude death rate above to determine the number of deaths expected.
4. There are many publications detailing methods that can be used for ageing and sexing. One of the most useful is Bass 1995. Another very helpful reference is Ferembach *et al.* 1980. Other useful sources include: Jurmain 1986; Katzenberg and Saunders 2000; and Reichs 1997. The outpourings from the myriad of physical anthropologists who are interminably seeking better and better ways of ageing and sexing the skeleton are to be found especially in the pages of the *American Journal of Physical Anthropology*; the fact that so many appear suggests that none is much improvement on what has gone before.
5. The jaws that Miles used for this work can be seen in the Hunterian Museum at the Royal College of Surgeons in Lincoln's Inn Fields, London.
6. There is often a small excess of young females in skeletal assemblages, most likely resulting from deaths relating to pregnancy or child-birth.
7. The still birth rate (SBR) is defined as:

$$IMR = \frac{\textit{Number of deaths in first year of life}}{\textit{Number of live births}}$$

The infant mortality rate (IMR) is defined as:

$$SBR = \frac{\textit{Number of stillbirths}}{\textit{Number of stillbirths} + \textit{number of live births}}$$

8. The numbers equate to 31% of males in the total sample and 36% of females; again, not a significant difference.
9. The autopsy came into prominence in medical practice during the eighteenth century, particularly with the work of Morgagni. It developed further in the succeeding century through the efforts of Bichat, Rokitanski and Virchow and it was at this time that clinical signs were correlated with autopsy findings especially in the schools of Paris, Vienna and Berlin. Virchow's (1876) book is interesting in taking the understanding of pathology to the cellular level. For further details see King and Meehan 1973; Burton 2001.
10. For the techniques of constructing historic life tables see: Ascádi and Nemeskéri 1970; Hassan 1981.
11. The expectation of life at birth is simply the mean age of death of the total assemblage. The nomenclature in the life table is a good example of how to make a simple concept appear complicated, something that epidemiologists and demographers seem to excel at.

Chapter 4 (pp. 41–48)

1. There have been many interpretations of Wolff's law but it is generally taken to indicate that bone is laid down in sites subject to stress and resorbed from sites where there is little stress; in other words, the shape of a bone reflects its function (Wolff 1891).
2. These values correspond to a mean of 5 ft 7 ins (range 5 ft–6ft 1 in.) for the males and for the females, 5 ft 2¼ ins (range 4 ft 10 ins–5 ft 6½ ins). A quick way to convert metres to inches is to multiply the metric measurement by 40.
3. Two of the most important historical studies are Komlos 1989 and Floud *et al.* 1990. For a later study see Alter *et al.* 2002.
4. For example, the height of young men entering the Post Office rose by 4 cm between 1881 and 1903 (from 1.67 m to 1.71 m) owing to improvements in the diet (Inter-Departmental Committee on Physical Deterioration, *Report, Vol. 1 and Appendix, Cd 2175*, London, HMSO, 1904).
5. Short stature seems particularly to be associated with death from heart and lung disease (Nwasokwa *et al.* 1997; Davey Smith *et al.* 2000. For iconoclastic views see Samaras and Elrick 1999; Samaras and Storms 2002; also Riley 2000 and references therein.
6. From birth to age one, growth takes place at the rate of 18–25 cm per year, from one to two years at 10–13 cm per year, while the pubertal growth spurt takes place at the relatively stately rate of 6–13 cm.

7. Strictly speaking a cross-sectional study such as this cannot be used to follow dynamic trends; that should be done by following up – in this case – a group of children as they grow. In palaeo-epidemiology, however, one must settle for second best, otherwise it would be scarcely possible to study anything!
8. For a modern example of this phenomenon see Checkley *et al.* 2003.
9. The so-called stress markers include enamel hypoplasia, cribra orbitalia, periostitis and Harris' lines.
10. Body weight (kg) at 18 = 3.383 × maximum head diameter (mm) – 85.8.
11. These means are equivalent to 12 stone 12 lbs for the males, and 9 stone 4 lbs for the females.

Chapter 5 (pp. 49–52)

1. The commonest of these include osteogenesis imperfecta, cleidocranial dysplasia, pyknodysostosis, congenital hypothyroidism and hypophosphatasia; the mnemonic for remembering them all is PORK CHOPS (Kaplan *et al.* 1991.
2. In her study of the skeletons from the *Mary Rose*, Ann Stirland found a high prevalence of os acromiale which she thought might have been caused by the use of the long bow (see Stirland 2000).
3. The various forms of patella partita can be found in Köhler and Zimmer 1968.

Chapter 6 (pp. 53–63)

1. There have been many studies of the rate of clinical error, in many countries; the rate found is seldom less than 10% and may be as high as 60%. See, for example, Cameron and McGoogan 1981; Burton *et al.* 1998; Coombes *et al.* 2004; Ferguson *et al.* 2004.
2. This phenomenon had been known about for very many years before this paper was written, but may have been forgotten; for example it was mentioned over fifty years earlier by Stockman (1920, 113 and fig. 92).
3. Eburnation may be found in other joint diseases that destroy the articular cartilage – rheumatoid arthritis, for example – but it is easy to differentiate osteoarthritis from these other diseases and no confusion should arise (see Rogers and Waldron 1995, introduction, n. 3).
4. Although there are studies which show that, for example, miners have a greater risk of developing OA of the knee or spine, and farmers OA of the hip, the great majority of those who get OA at these sites are neither miners nor farmers. It would be possible to tell the occupation of an individual from his or her pattern of OA if the pattern were unique to a particular occupation. For further details, see Waldron 1994.
5. The prevalence of a disease is related to the incidence by the following approximation:

$$P \approx I \times D$$

where P = prevalence, I = incidence, and D = duration of the disease. Where the duration is short, P and I are very similar; where the duration is long – as in OA – the prevalence may be several times the incidence.
6. A number of special studies of osteoarthritis were carried out using the Barton assemblage; for details of these see Shepstone *et al.* 1999; 2000; 2001; Rogers *et al.* 2004.
7. In the text that follows, all significant differences can be taken to mean that $p < 0.05$, unless otherwise stated.
8. When making comparisons between the number of sites affected, compound joints such as the knee or the elbow are counted singly, no matter how many compartments are affected. Similarly multiple joints, such as the proximal or distal inter-phalangeal joints of the hand, are counted as a single instance, no matter how many individual joints are involved.
9. Unfortunately, the relationship between OA of the TMJ and tooth wear could not be investigated here since tooth wear was not recorded. A case-control study was undertaken, however, to see whether those with TMJ disease had lost more teeth than those without. Data about the number of teeth present were available for 10 of the 12 cases with TMJ. They were randomly matched with 40 controls without OA of the TMJ and the number of teeth lost during life was expressed as a percentage of the total number extant. Six of the cases had lost less than 50% of teeth, and four had lost more than 50%. The corresponding numbers for the controls were 33 and 7, respectively. This difference is not statistically significant (Fisher's exact test $p = 0.13$). The result suggests that in this assemblage, at least, tooth loss is not related to OA of the TMJ.
10. The association between obesity and OA of the hip is much weaker than with either the knee or the hand, which suggests that stress on a joint is not necessarily a major precipitant of the disease (Lievense *et al.* 2002.
11. The differences that are noted in Tables 26 and 30 are due to the fact that the denominators for calculating the prevalence rates are not the same in each case, being the number of joints present, not the number of individuals.
12. For comparative purposes it is necessary to determine the age- and sex-specific prevalence. Unfortunately, this was not possible here because so many of the skeletons could not be assigned an age or sex, leaving too few adult skeletons for further detailed analysis.
13. These are (from anterior to posterior in the order in which they insert into the humerus): subscapularis, supraspinatus, infraspinatus and teres minor.

Chapter 7 (pp. 65–72)

1. *Doit-on admettre une nouvelle espèce de goutte sous la denomination de goutte asthénique primitive?* An unabridged version of the text of the thesis can be found in: 'The first description of rheumatoid arthritis. Unabridged text of the doctoral dissertation presented in 1800', *Joint Bone Spine*, 2001, **68**, 130–43. Those who have presented MD theses in recent years will be amazed at the size of this one.
2. It is interesting to follow the changes in the understanding of joint diseases in the text books of rheumatology. In W.S.C. Copeman's, *Textbook of the Rheumatic Diseases* (E. & S. Livingstone, Edinburgh) the rheumatoid factor appears first in the third (1964) edition; the first and second editions appeared in 1948 and 1955, respectively. Not until the fourth (1969) edition, however, is the nature of RF 'so well established that it is no longer necessary to discuss the several arguments against it' (p. 187); it is in this edition also that the term sero-negative first appears. Many of the sero-negative arthropathies are included in the early editions as variants of RA. It is important to recognize the changes that have taken place in understanding rheumatic diseases – and in their nomenclature – over time when trying to compare the frequency, or the first appearance, of joint diseases in earlier bone reports as they may well be described under different rubrics from the present day. How many synonyms were used for OA in earlier publications can be understood from reading an old text book such as Stockman (1920).
3. This form of arthropathy is associated, for example, with chronic inflammatory gut disease such as ulcerative colitis and Crohn's disease (Holden *et al.* 2003). The pathological

changes are similar to those in Reiter's or reactive arthritis but there are no means whereby this type of arthropathy could be distinguished in the skeleton.
4. The major histocompatibility complex (MHC) is part of the immune system. It comprises a series of proteins that bind to fragments of antigens. The Class I MHC proteins present antigens to cytotoxic T lymphocytes. An allotype is the protein product of an allele which may be detected as an antigen in an animal of the same species. For further details see Hughes 1997.
5. Erosions are areas of bone loss resulting from the action of inflammatory cytokines and osteoclasts. They are characterized by the loss of bone cortex and exposure of the underlying trabeculae. The term is employed here only to refer to lesions accompanying joint disease and should best be restricted to that use. They may be confused with other holes in bones such as vascular foramina and the openings of cysts, but in non-pathological holes the cortex is always intact so long as there is no *post mortem* damage. Differentiating *post mortem* damage from true erosion can be difficult, but the former is seldom symmetrical and may well extend beyond the confines of the joint. Fresh damage to bone is usually easy to distinguish because the colour of the broken bone differs from the remainder. Some of the erosive joint diseases are accompanied by local osteoporosis and are easily damaged; this may confuse the picture. True erosions sometimes have undercut edges which never occur in an artefactual lesion and on X-ray a true erosion will frequently be surrounded by a white line – what the radiologists refer to as sclerosis – which is the result of remodelling around the lesion. For further details see Rogers and Waldron 1995, introduction, n. 3.
6. It would be possible to confirm a diagnosis of RA, if RF could be extracted from the bones of putative cases. When we tried this on bones from other sites, including one in which RA was almost certainly the correct diagnosis, we obtained no positive results using a commercially available kit. This may have been too insensitive to detect the small concentrations of RF that may have survived and it would be interesting to repeat the experiment using a much more sensitive method of analysis (D. Antoine and T. Waldron, unpublished data).
7. The denominator used for the calculation was the number of adult skeletons with either hands or feet present (n = 426).
8. This skeleton has also been described elsewhere (Rogers 1988).
9. In the French literature the condition is still more often referred to as Fiessinger–Leroy, or Fiessinger–Leroy–Reiter syndrome.
10. Gout may be associated with other conditions and it may be precipitated by a number of drugs.
11. Valgus refers to the angulation of one part of the body away from the mid-line of the body relative to the part immediately proximal to it. Its opposite, varus, refers to the converse; that is when one part of the body is toward the mid-line relative to the part immediately proximal to it. A bunion may be present at the fifth mtp joint, the so-called tailor's bunion, or bunionette; in this case there is a valgus deformity of the fifth proximal phalanx.

Chapter 8 (pp. 73–81)

1. It has been considered that the bovine bacterium was the first to infect man and that the human bacterium subsequently evolved from it. Recent DNA studies, however, suggest that this is very unlikely and the inference is that both have infected man for many thousands of years (Brosch *et al.* 2002).
2. Pott's disease is of considerable antiquity and has been recognized in human remains at least as far back as ancient Egypt: Palfi *et al.* 1999). A well known case was described by Elliot Smith and Ruffer (1910) in a mummy from the twenty-first dynasty.
3. Tuberculosis can be diagnosed with certainty if bacterial DNA can be recovered from the bone, or soft tissues if dealing with a mummy. This has been achieved on a number of occasions (see, for example, G.M. Taylor *et al.* 1999; Konomi *et al.* 2002). The bacterium has a waxy coat which contains mycolic acids and these too can be recovered from human remains (Gernaey 2001). With both techniques it is possible to determine whether the infection has been caused by the human or the bovine bacterium.
4. According to John Graunt (1662, 14), those recorded as having died of consumption were individuals whose bodies were 'very lean and worn away'.
5. Polio is another disease of considerable antiquity and the depiction on the stele for Ruma (or Rem) at the sanctuary of the goddess Astarte at Memphis dating to *c.* 2000 B.C. is well known (Major 1954, 43).
6. The diagnosis of the cause of periosteal new bone in the living depends to a large extent upon its radiological appearance (Ragsdale *et al.* 1981). Many of the cases of pnb seen in the skeleton are not amenable to radiological interpretation because the quantity of new bone is too slight to show up on X-ray.
7. The extent of the pleura on both sides of the chest and the relation to the ribs is well known to clinicians, as is the relation of the various lobes of the lungs to the overlying ribs. A pleural effusion would be expected to gravitate towards the bottom of the chest and involve the lower ribs on one side; if the individual were bed-bound then the effusion might settle towards the back of the chest. Pneumonia affecting the left upper lobe of the lung would be deduced from finding pnb on the first four ribs, for example. Diagrams of the lung and pleural markings can be found in any text on clinical anatomy and used to map out the areas affected if the ribs are sufficiently well preserved.
8. Periosteal new bone as a normal phenomenon in children has been recognized for at least 40 years (see, for example, Shopfner 1966); but it may still cause difficulty in diagnosis (de Silva *et al.* 2003).

Chapter 9 (pp. 83–94)

1. Further information on types of fractures, their causes and their complications may be found in, for example Koval *et al.* 2002.
2. In some cases fractured bones are not displaced. This is true with fractures of the ribs where the fractured rib or ribs will be held in place by the rest of the rib-cage unless the damage is very extensive, when it is likely that the individual will not survive. A fracture of either the ulna or the radius which leaves the other bone intact will, similarly, generally not be displaced because the fractured bone will be splinted by its intact neighbour. The same is true of a fracture of the fibula in which the tibia is undamaged.
3. The Schatzker classification has been used here; see Müller *et al.* 1990.
4. Type 2 and type 3 fractures are the result of relatively low impact energy, such as a fall, but the type 5 injury, involving the medial plateau, is much stronger than the lateral and is

the result of a high energy impact, such as a blow to the knee by a fast-moving object.
5. In this condition, the nerve roots from the fifth and sixth cervical roots, which contribute to the superior trunk of the plexus, are involved. The arm is adducted and internally rotated, while the forearm is pronated and extended; the hand points backwards in the so-called 'waiter's tip' position; see: Piatt 2004.
6. The pattern of inheritance in this condition is not clear but the underlying genetic disorder is becoming understood (Kaplan *et al*. 2004).
7. This is also referred to as Legg–Calvé–Perthes' disease.
8. The aetiology of this condition is not known but there is a clear association with poverty, suggesting the environmental factors are important in its causation (see, for example, Barker and Hall 1986).
9. There is a well-recognized congenital form of spondylolysis that affects the cervical spine (Yochum *et al*. 1995).

Chapter 10 (pp. 95–99)

1. Or men, of course, but among the present-day population, it is women who predominate in screening programmes for osteoporosis.
2. Strictly speaking, this is the definition of primary osteoporosis. Secondary osteoporosis may occur as a concomitant of a number of other diseases and with prolonged use of some drugs, particularly corticosteroids.
3. The activation results from the addition of two hydroxyl groups, the addition in the kidney being regulated by parathyroid hormone (PTH).
4. Rickets was a common cause of obstructed labour in the eighteenth, nineteenth and early twentieth centuries (Loudon 1986).
5. The osteoclasts in Paget's disease contain viral-like nuclear inclusion bodies and produce IL-6, a resorptive cytokine which is responsible for the increased resorption of bone (Roodman 1995; Bender 2003).
6. The specific fault appears to be in the sequestrosome 1 gene on chromosome 18q (Noor and Shoback 2000; Rousiere *et al*. 2003; Eekhoff *et al*. 2004).
7. When Juliet Rogers calculated the prevalence, using the old phasing data, she found that there was actually an increase in the prevalence from the early to the late periods, from 1.7% to 3.1% and this was reported in the literature (Rogers *et al*. 2002). After re-allocating the skeletons to their new phases, the difference has disappeared and an erratum has been placed in the journal in which the original report appeared (Waldron 2004). Interestingly, an earlier study of the prevalence when about 1,700 of the skeletons had been examined came to the conclusion that there had been *no* change in the prevalence over time (Rogers 1996a).

Chapter 11 (pp. 101–104)

1. In the rare condition of situs inversus, in which the aorta lies on the right-hand side of the thoracic vertebrae, the changes in DISH are found on the left-hand side, which certainly indicates that the presence of the aorta is a significant factor in preventing the ossification from occurring (Ciocci 1987).
2. The females were excluded from this analysis because the numbers were too small.
3. Fourteen sites were routinely inspected; the list can be found in Rogers *et al.* 1997, 86, n. 12.
4. The reason that all the cases of DISH (and early DISH) were not included among the bone-formers as would be expected is that many of the skeletons with DISH were not sufficiently intact for the sites of entheses to be inspected.
5. The hyperostosis may spread beyond the confines of the frontal bone in some cases, sometimes even to include much of the internal table.
6. It might be argued that some of the cases of HFI that were not detected could have had DISH or eDISH, contradicting the conclusions reached here. There is no reason to suppose, however, that the skulls of skeletons with DISH or eDISH would be less likely to break than those without, and so no bias against finding DISH or eDISH with HFI exists.

Chapter 12 (pp. 105–110)

1. The process whereby tumours arise is sometimes referred to as neoplasia, and tumours as neoplasms; this is simply medical jargon for new growth.
2. Resnick also said that 'In no other area of musculoskeletal disease is the cooperation of the othopedic surgeon, radiologist, and pathologist more important. Any of the three specialists working independently of the other is more likely to err.' (p. 3602). Bone specialists beware!
3. Surprisingly, no cases of meningioma were found. These tumours are by no means uncommon in skeletal material, although they are usually not detected in the intact skull unless it is X-rayed for some other purpose, or if the skull is examined with an endoscope.
4. There is a form of multiple enchondromatosis which presents, *inter alia*, with café au lait spots on the skin; this is referred to as Ollier's disease. In another variant, known as Maffucci's syndrome, haemangiomas are also present (Ahmed *et al*. 1999).
5. Fibrous cortical defects (FCD) are often referred to in the literature as non-ossifying fibromas or fibroxanthomas. The two last terms are synonymous but FCD differ on the basis of size and natural history; they are <30 mm in size and most heal spontaneously although they may on occasion enlarge and evolve into fibroxanthomas. Both FCD and fibroxanthomas are to be differentiated from cortical avulsive injuries that are related to repetitive stress at the site of muscle insertions (Resnick and Greenway 1982).
6. Multiple osteochondromatosis is an hereditary condition, often caused by a mutation in the EXT1 gene (Trebicz-Geffen *et al.* 2003).
7. Osteomas are commonly found in the external auditory meatus of those who swim or dive in cold water (Harrison 1962).
8. Other examples were found in the disarticulated material, but the exact number has not been recorded.
9. The notes on which this description of the study is based do not give an indication of why this number of skulls was randomly selected in the first place.
10. The volume is calculated as being equal to $(L \times D \times W)/2$ mm^3 where L = the maximum lateral length; D = the maximum lateral depth; and W = the maximum width on the a/p projection. The method used and the upper limit of normal are described by Di Chiro and Nelson 1962.
11. This lesion is a fluid-filled remnant of Rathke's pouch which grows up in the embryo from the primitive mouth to form the anterior lobe of the pituitary gland.

Chapter 13 (pp. 111–115)

1. There is another rare condition in which changes similar to those seen in fibrous dysplasia are noted in the cortex of the long bones; this was first described as a separate entity in

1976 and termed osteofibrous dysplasia (Campanacci 1976). Recent molecular studies have shown that the genetic abnormalities in fibrous dysplasia are not present in osteofibrous dysplasia and this strongly suggests that the two conditions have a separate aetiology (Sakamoto *et al.* 2000).
2. The Cobb angle is calculated by measuring the angle subtended between the superior end plate of the uppermost end vertebra of the curve, and the inferior end plate of the lowermost end vertebra of the curve on a straight X-ray.
3. In this case nine thoracic vertebrae were present and in such poor condition that they could not be identified. Two were fused anteriorly with left-sided scoliosis; two other vertebrae had sustained crush fractures and at least four ribs were fractured.
4. For a view contrary to the mainstream, see Häusler *et al.* 2002.

Chapter 14 (pp. 117–120)

1. For some of the special studies carried out using the Barton teeth, see Wollaston 1995; Sengupta *et al.* 1998; Sengupta *et al.* 1999.
2. There were 1,940 adult skulls with the maxilla and/or mandible present, each of which should have yielded 32 teeth giving a total of 62,080 teeth expected.
3. This is probably an over-simplistic view of caries and it ought to be considered a multi-factorial disease (see Fejerskov 2004).
4. The one which is probably most commonly used is that of Moore and Corbett (1971); for a more extensive and more systematic approach see Hillson 2001.
5. This may be the reason why so many people were reported as having died from 'teeth' in the *London Bills of Mortality* (Clarke 1999).

Chapter 15 (pp. 121–128)

1. *An Act for the Better Regulating and Preserving Parish and Other Registers of Birth, Baptisms, Marriages, and Burials in England, 1812.*
2. *The Marriage Act, 1836* and *The Births and Deaths Registration Act, 1836.*
3. The actual work of transcribing the records and putting them into an electronic database was carried out by David Parry, to whom we are extremely grateful.
4. The best known recent investigation of the accuracy of ageing skeletons was the Spitalfields project. When the crypt at Christ Church, Spitalfields, was cleared a number of coffins were found with the coffin plates intact and the ages of death on the plates were compared with those derived from the skeletons, with the expected results. This much-written-about and frequently quoted study, however, suffers from a serious methodological flaw in that a single observer made all the age estimations; it would have been a very much improved study if two or three observers had been used after suitable inter-observer comparisons. Such opportunities as this are rare and it was a pity that this was lost. For further details see Molleson and Cox 1993.

Chapter 16 (p 129)

1. Henry VIII's great ship, the *Mary Rose*, sank on the morning of Sunday 19 July 1545, with the loss of most of the men on board. Their remains were examined and reported on about 450 years later by Ann Stirland (2000).

Bibliography

Abbreviations

BAR	British Archaeological Reports (British Series)
CBA	Council for British Archaeology
HMSO	Her Majesty's Stationery Office
Sk.	Skeleton
WEA	Workers' Educational Association, Barton-upon-Humber Branch

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