Variation in rates of age-related change in skeletal tissue in a Romano-British population

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ABSTRACT

1228 skeletons from the Romano-British cemetery at Poundbury have been used to compare methods of assessment of age at death on archaeological material. The main aim was to evaluate the potential use of methods based on cortical bone structure and of the occurrence of degenerative joint disease in the spine.

Changes in cortical bone do not proceed linearly with age, so methods currently available, which are based of single regression equations, are inadequate. The measures of bone structure are found to be useful in conjunction with other methods in terms of calibration. The expectations of greater variation in bone structure measurements amongst males was not observed. In the case of cortical thickness exactly the reverse is found. Possible explanations are discussed.

Degenerative joint disease of the spine offers a promising means of age assessment as it is found to increase in incidence, severity and extent with age. Males show a faster rate of degeneration than females particularly in extent. The best measure indicated is the combined number of facet and disc joints affected in the lumbar region.

The Poundbury population is assessed as having a mean age of death around forty-five, with very few survivors beyond sixty. A greater proportion of males are found in the older age groups than females. The pattern of mortality given by the dental attrition method of age assessment is essentially validated, but in absolute values the method underestimates the ages.

VARIATION IN RATES OF AGE-RELATED CHANGE IN SKELETAL TISSUE IN A ROMANO-BRITISH POPULATION

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PART I

LITERATURE REVIEW

INTRODUCTION

The current methods for determining the age at death from skeletal remains make two general assumptions of uniformitariansim. First, it is assumed that biological processes of aging in the skeleton have the same relationship to actual chronological age in the past as in the present. Secondly, it is assumed that the methods derived from present day populations will be applicable across the entire population of the past, that is, that the variation in biological rates between individuals in a past population will be no greater than in the present.

There is a growing realization amongst both physical anthropologists and the archaeologists, dependent on their data, of a pressing need for some detailed evaluation of variations in biological aging rates between and within populations, between the sexes, between different age groups and between different parts of the skeleton.

This thesis is a contribution to the solution of the problem by comparing different current aging techniques and measures of age-related changes within one population. The current aging methods to be compared are those of dental attrition (Brothwell, 1972), pubic symphysis metamorphosis (McKern and Stewart, 1957; Gilbert and McKern, 1973) and

eleven different age estimates from changes in cortical bone structure (Kerley, 1965; Ahlquist and Damsten, 1969; Thompson, 1978; Samson, 1983). The measures of age-related changes to be investigated are the various parameters measured or counted from the cortical bone, the scores of dental attrition and pubic symphysis metamorphosis necessary to produce the age estimates to be tested.

In addition, an assessment is made of age-related degenerative changes in the vertebral column, that is osteoarthrosis and osteophytosis. The specific focus of interest is aimed towards assessing the potential use of the measures of cortical bone structure and of degenerative joint disease.

The collection of skeletons from the Romano-British site of Poundbury in Dorset is used for this study. The collection comprises 1228 individuals and is of good preservation.

The literature on skeletal aging methods, age-related changes in cortical bone structure and degenerative joint disease, with specific reference to the spine will be reviewed in part 1 of the thesis (chapters 1 to 4). Part 2 contains the details of the original research carried out on the Poundbury collection. Chapter 5 describes the sample, the methods of data collection and the analyses to be performed. Chapters 6 to 9 present the results of relationships between the various parameters and chapter 10

contains a discussion of the findings, picking out a few specific issues for attention.

Chapter 1. AN INTRODUCTION TO AGING.

1.1 The Phenomenon of Aging.

An aging organism is defined as one which experiences an increased probability of death with increasing age, that is the life expectancy decreases with age. Aging or senescent processes are age-related changes in the body components causing this decreased probability of survival. Although death itself is a sudden event, age-related deleterious changes are accumulated up to a threshold beyond which the body can no longer tolerate the level of functional impairment present in its senescent parts (Lamb, 1977).

Why organisms should age at all is a question which confronts evolutionary biologists (Hamilton, 1966; Kirkwood, 1977; Medawar, 1952; Williams,1957; Charlesworth, 1980; Moment,1978). The observation that comparative studies show species-specific life expectancies implies some level of genetic control over aging processes (Sinex, 1977; Brash and Hart, 1978). On the other hand, although there are a number of body functions, organ systems and their cells which are universally found in all multicellular animals, not all organ systems etc. age in the same way in different species (Rockstein et al., 1977). This illustrates the difficulty of producing a single theory of aging except for at the broadest level.

Theories for the evolution of aging can involve active or passive selection. The former instance proposes that there is a positive advantage to aging which is actively selected. The argument usually follows the commonsense sounding line that individuals must die to prevent overcrowding. This style of argument can be rejected on two grounds. First, the advantage derived from aging is for the good of the group and as yet there has been no evidence of mechanisms by which group selection can operate. Secondly, under natural conditions, mammals and early humans do not manifest their potential aging pattern as death is more likely to occur from accidents befalling individuals randomly at any age than from an age-related increase in susceptibility. Therefore, aging and age-related mortality is not seen in wild or natural populations to act as a regulatory mechanism on population growth.

Passive theories of the evolution of aging accept that faults will arise in the processes and functioning of any organism by the very nature of biological information transferal. The accumulation of such errors with time constitutes the aging process. Cutler (1978) calls these harmful effects biosenescent processes which can cover anything whether intrinsic or extrinsic to the organism. The occurrence of copying errors is recognized as an essential feature of the evolutionary process and an optimal level of error will have been selected for in any given organism. However, the expression of biosenescent

processes must be held back until after successful reproduction, so balancing processes, which Cutler terms antibiosenescent, evolve to counteract or delay the expression of aging.

In selection terms, genes having bad effects pre-reproduction will be selected against, whereas those acting post-reproduction will not be selected against and could become fixed in a population by stochastic events. Particularly important may be the effects of pleiotropy where genes with beneficial actions for survival early in life will be favourably selected regardless of the fact that the same genes may produce biosenescent effects later in life. Similarly genes having antibiosenescent behaviour early in life will be strongly selected whereas those not acting until later will not. The selection, therefore, can be direct through gene action or indirect promoting antibiosenescent processes through selection. A balance is ultimately found between the retention of order, and the tendency towards disorder, or entropy in terms of energy input or costs to retain order and the output or reward of doing so in evolutionary currency (Kirkwood, 1977). The variety of forms of life today and variety of maximum lifespan potentials reflects the many different compromises or balances evolved between these two opposing forces.

Biosenescent processes can arise from any biological process where possible errors can arise in information copying or interpretation, or where side products of chemical reactions are formed which similarly accumulate.

The early work of Rubner in the nineteen hundreds first established the existence of a strong correlation between the metabolic rate of a species and its maximum lifespan potential, with some notable exceptions. These exceptions, which include humans, have a larger ratio of brain to body size than the average relationship over the range of species (Sacher, 1978), which seems to play some part in the increased maximum lifespan potential. Sacher (1978) sees it as an expression of improved homeostatic control acting as an accompaniment of increased brain size, whereas Cutler (1978) sees the increase in both brain size and longevity as related back to the selective pressures for features to enable the development of an increased learning capacity.

Specific chemical reactions known to increase errors in DNA and other protein syntheses are those involving free-radicals. These are highly reactive by-products of various metabolic processes which can act as crosslinking agents between macromolecules, impeding the accurate breakdown or replication of these molecules in subsequent reactions (see Harman, 1956,1968,1971,1972,1973; Dormandy, 1983). These free-radicals can be particularly harmful if forming crosslinkages between DNA strands (Bjorksten,1974). One of the most dangerous free-radicals is that of oxygen, O_2^-, in mammalian cells, because of the great number of reactions involving oxygen and releasing this particular free-radical. Free-radicals are just one type of crosslinking agents, many of which exist in the organism. Many molecules

form crosslinkages with each other simply as the continuation of processes forming molecules and macromolecules in the first place. This is seen to be the case in collagen for example, as important component of connective tissues. The accumulation of crosslinkages with age is recognized as a powerful biosenescent process.

Antibiosenescent process must therefore counteract such reactions. Repair mechanisms for DNA and the other proteins exist, but, of course, at no time is it economical to invest the energy required to repair all the damage present. All that is necessary is a level of repair sufficient to maintain the integrity of the organism long enough to successfully reproduce. Energy can be more valuably invested elsewhere, for example in reproduction itself. Some of the recognized agents of antibiosenescent processes are superoxide dismutases, antioxidants and free-radical scavengers and the selective removal of abnormal proteins. Delaying the rate of development and slowing the metabolic rate are antibiosenescent processes evolved by particular species including humans.

1.2 Mortality in Humans.

A distinction is drawn by Pianka (1978) between the kinds of strategies adapted by different species as regards reproduction and rearing investments; r selected strategists have short maximum lifespans potentials, high metabolic rates, early reproduction, a high frequency of young

produced at any one birth and very little subsequent investment in the offspring. Primates have a k selected strategy, investing greater energy in each single offspring both before and after birth, with a longer development period in offspring and a greater learning capacity. This is most developed as a strategy in humans who display extended development time through neoteny and slow metabolic rate. This is accompanied by energy investment in antibiosenescent mechanisms for repair to allow the length of life necessary to successfully raise the offspring.

The human maximum lifespan potential is given as ninety-five (Weiss, 1981) but obviously very few actually attain such a ripe old age. As mentioned earlier, wild animals do not experience aging as an important factor leading to death, since death from hazards of the environment are of the greatest importance, for example, accidents, attacks, birth, starvation, disease, poisoning, etc. This is thought to have most likely been true for humans also throughout the majority of our history. Vastly different patterns of mortality can be seen in different populations, both those of today and in the past. The most important influence on mortality patterns is seen as environmental circumstance, taking this to embrace both the ecological situation and societies' own patterns of activity and responses to accidents or aging.

Assessments of mortality patterns in the past tend to indicate the same general trend. Early human populations

are thought to be characterized by high infant mortality and a mean adult age of death around 30 to 40. The mean age of death could vary considerably between populations and between the sexes, but is generally considered to have been low. Childbirth is presumed to have accounted for female deaths in the twenties and thirties in the majority of the population, possibly giving a lower mean age at death in females. Males would be more prone to death from accidents, or attacks occurring at any age of life, perhaps giving a more even distribution of age at death in males. Average longevity is not reported to increase noticeably towards modern levels of the western world until after the Industrial Revolution, not, as was expected, after the Neolithic Revolution (Brothwell, 1972; Angel, 1947; Weiss, 1981). Weiss (1981) also examines the variation in age at death. This he finds to be far greater in earlier populations than in post-industrial societies of Europe, reflecting the greater likelihood of death at any age in earlier times.

Studies of past demographic patterns have to depend on fragmentary information from early documentations, inscriptions of tombs or monuments, settlement sizes, quantities of debris left and analogy with present day so-called 'primitive' societies. Obviously, the most direct information on mortality can come from examination of skeletal remains. Many of the mortality trends described above are derived from or supported by skeletal studies. The importance of reliable aging methods or at least some idea of the extent of their unreliability is crucial for any

discussion of demographic trends based upon this evidence.

1.3 Anthropological Methods for Aging Skeletons.

A distinction is drawn between biological age, that is the extent of development or degeneration of any particular feature and the chronological age, that is the number of years the individual has been alive (Benjamin, 1947). These are shortened to bioage and chronoage here.

The development of a method for assessing the age at death of an individual from the skeleton requires two steps. First, a clear, uncomplicated way of measuring the bioage of the particular anatomical feature under study must be devised and secondly, the relationship of change in bioage to the chronoage must be established and some way of converting the measure to an absolute age estimate provided together with an assessment of the estimate's accuracy or resolution.

The development of most methods for aging skeletons draw on epidemiological research in the medical fields of growth and senescent changes. Such studies, which are usually based on living populations, provide ranges of bioages in relation to the controlled chronoage. The anthropologist takes this relationship and turns it around to get a chronoage range from a known bioage. However, there are a number of statistical pitfalls in the development of a method for anthropological use which have not always

been avoided. The provision of a means of measuring or grading the bioage is not a problem, and although for certain anatomical features a number of different means of bioage measurement exist, the researcher's choice of method should be based on the soundness of the conversion process rather than the means of measurement.

First, many bioage measurements do not use interval scales but grade the feature from a visual assessment using an ordinal scale. If a number of components of the anatomical feature are to be graded, it is inadmissible to then treat these grades as if they were an interval scale and take a mean average value. If an average is to be taken, the median or mode should be used (Siegel, 1956). If a wider range of numbers is required for finer discrimination, then a summation of the various grades can be used. The use of mean averages from ordinal scores puts into question the foundations on which the aging method is based.

Secondly, distortion of the population's assessed age structure, particularly in the middle range, will result if the same table is used to get the chronoage from known bioage as that which expresses the bioage range in relation to the known chronoage. Separate tables should be given for each of the two relationships (Masset, 1971). This means that tables published in medical journals on paediatrics or geriatrics giving ranges of bioage in relation to the known chronoage should not be used directly. Access to raw data would allow the anthropologist to draw

up the necessary table for the opposite relationship.

Thirdly, and of the greatest importance, the age and sex structure of the reference population from which the method has been developed will affect the conversion process. Therefore, the reference population should be similar in age and sex distribution to the archaeological population under study. Although, of course, this is the unknown, the details of which are to be found out, the gross structure of most skeletal samples can be assumed to have come from a population of more or less equal sex ratio and with a distribution of ages from neonates to the elderly. The age distribution of a reference population should therefore cover this range, preferably with a reasonable sample size over the ages, or else specify the use of the method for a limited sector of the range. This sector of the population would then have to be identified first by other aging criteria.

Bearing these considerations in mind, a review of current aging methods can be made. The methods for aging subadults are touched on but briefly, with a fuller coverage of the methods for aging adults since this is the problem area in which this thesis is located.

1.4 Aging Subadults.

The main skeletal processes used for bioage assessment and subsequent estimation of chronoage from subadult

skeletons are as follows:

Appearance of ossification centres

Growth of bones in length

Fusion of epiphyses

Development of teeth

Eruption of teeth

The appearance of ossification centres occurs in the foetal stage and can be used to estimate foetal age if such is found in excavation. There is a vast literature in paediatrics on childhood growth rates, variation within normal limits and effects thereon of nutritional status, disease, physical activity, racial differences etc. The use of teeth is the best age indicator for juveniles, first, because it is the most commonly preserved element and secondly, because tooth development is less affected by sex differences than is bone growth (Hunt and Gleiser, 1955). The simplest methods of aging from teeth relate eruption times to absolute age (Brothwell, 1972). A more precise method considers in detail the development of the tooth components, the crown and root pre- and post-eruption (Demirjian et al., 1973). This method involves the use of radiography which accounts for its lack of popular use.

Long bone lengths give age if tables are used based on a populations relevant for the one under study. Once full growth is reached in any osteological element, the epiphyseal plates will gradually fuse to the main unit. This again is well-documented and can provide a good estimation of age in late adolescence. The various epiphyses fuse at different

ages so the possible range of age can be narrowed down using all the bones available (Bass, 1971).

The fusion of the bones of the skull cover a wide range of ages between them, the latest being the spheno-occipital synchondrosis in which total fusion may not be complete until the mid-twenties (Melsen, 1972; Redfield, 1970).

In both the assessment of growth measurements and epiphyseal union, there is a difference between the sexes at which age these occur, a difference which can be several years. Although this is nothing compared to the inaccuracies of adult age determinations, it is quite considerable in proportion to the age under discussion. Therefore, it is important to try to obtain the best tooth age determinations on which to callibrate other age assessments of both juveniles and adults. The use of X-rays in assessment of tooth development could prove worth the extra time investment.

1.5 Aging Adults.

The age-related processes drawn upon to assess chronoage after epiphyseal union fall into three categories.

a) Continuing development:

fusion of cranial sutures

metamorphosis of the pubic symphysis

remodelling of bone

b) Mechanical:

wear on dental occlusal planes

c) Degenerative:

osteoanthrosis

osteophytosis

osteoporosis

1.5.1 Cranial Suture Closure.

The cranial sutures separate the plates of bone of the cranium, thereby allowing growth of the skull and the enclosed brain. These slowly fuse throughout life until the final stage where the line of the suture is no longer visible. The sutures can be examined from the outside of the skull-case, ectocranially, or from the inside, endocranially. Most methods of age estimation from cranial suture closure are based on the pioneering work of Todd in the twenties. On a collection of known aged males of 267 Todd and Lyon (1924, 1925) studied the extent of suture closure in relation to age. They provide a detailed description of the progressive fusion of the sutures both endo- and ecto-cranially, giving the mean age at which progressive stages of fusion occur. However, this is not presented as a clear method for aging with a conversion chart but rather as a description of characteristic ages for any stage in cranial suture closure. Therefore, as Masset (1971) points out, their paper was not an attempt to provide an aging method, but merely a preliminary step towards one by examining the age-related changes at a descriptive level. He claims any

method based on their original publication is not based on a secure foundation. In addition, since ectocranial observations are much more easily performed than endocranial, most methods of cranial suture aging are done ectocranially. Todd and Lyon (1924) themselves state that ectocranial closure is more variable than endocranial closure. Even of endocranial closure, which they did use themselves in a test run to age a small sample of skeletons, they say that their findings do not justify 'uncontrolled' use of suture closure for age determination.

Aging by cranial suture closure has gained a reputation for inaccuracy in more recent publications (Brooks, 1955; Singer, 1953). Masset (1971), although critical of the widespread use of Todd and Lyon's original descriptions, is more optimistic of the usefulness of cranial suture aging as a method in conjunction with other age indicators. He claims systematic errors are introduced into the methods, avoidance of which can improve the reliability of the age relations. In addition to the misuse of Todd and Lyon's descriptions, the problem mentioned above of using separate tables for the two directions of relationship between age and suture closure must be made available. Thirdly, the similarity of age-related patterns of suture closure in the sexes when expressed as modal patterns obscure less obvious but important differences about variation, dispersion, skewness etc. Again, as mentioned before many papers do not present information beyond the average in relation to age which falls short of the full details

required. Masset (1971) claims use of the same tables for males and females causes an underestimation of female age and distortion of the overall age structure of the female population.

The Hungarian workers, Acsadi and Nemeskeri (1970) have themselves examined a sample of 352 individuals of known age and sex - 208 male and 144 female aged between 15 and 89. They studied the age-relationships of both ectocranial and endocranial suture closure. They therefore avoid Masset's criticism of misusing Todd and Lyon's publication by presenting their own results. They also present different tables for the two directions of age to suture closure relationship. However, they grade the three main sutures (coronal, sagittal and lambdoid) in a total of sixteen sections, then take a mean value over these sixteen, thereby misusing an ordinal scale as if it were an interval scale. They do publish the ranges of ages in relation to the suture stages and they feel confident in using endocranial suture closure as one of the methods in their complex method of aging, a method which incorporates a number of parameters to get an age estimate. They introduce the idea of limiting ranges using aging methods to state that no individual over or under a certain age would display a certain level of age-related change. By combining this practise over a number of parameters they can narrow down the estimated age range.

1.5.2 Pubic Symphysis Metamorphosis.

Using changes on the articular surfaces of the pubic symphysis as age indicators was also pioneered in the 1920s by Todd. He studied the pubic symphyses of a sample of known age, sex and race:

306	male	white	18 - 70
90	male	negro-white hybrid	17 - 45
47	female	white	16 - 74
22	female	negro-white hybrid	16 - 45

The white male sample provided the main description and the age changes of the other groups were compared to this main group for differences. He distinguishes ten stages through which the surfaces change from young adulthood through into the fifties and over. The alterations of the pubic symphysis had previously been commented on by Aeby (in Todd, 1920), who, from measurements made, considered the changes to represent continuing ossification i.e. a process of growth. Todd (1920,1921) by contrast, interprets his measurements as representing a process of metamorphosis rather than growth. Todd (1921) compares the female and negro-white hybrid group for differences and notices slightly different rates at which the stages are passed, but, nonetheless finds they do go through the same stages of metamorphosis. However, since his sample size for these groups is small on the whole, particularly for the females, this is not developed further than the

level of observation. The pattern of changes observed in the pubic symphyses pass from a ridge and furrow formation in the adolescent/ young adult, through the development of a dorsal margin with ventral bevelling in the twenties. The thirties see the development of a ventral rampart. In turn this changes to a flat symphysial face in the forties with the development of a rim around the face. This in turn breaks down after fifty and pitting and osteophyte formation may be observed.

Todd's observed stages of age-related metamorphosis in the pubic bone has been used widely either as presented by Todd, or developed further by subsequent researchers. Brooks (1955) using Todd's ten stages finds a very good correlation with known age for males and a slightly lower one for females but still good.

It is often suggested that possible damage to the pubic bone from pregnancy and childbirth could cause incorrect age assessment in females by imitating the later stages of pubic metamorphosis. In pregnancy the stresses on the central abdominal muscles stretches these and causes reinforcement of the sites of muscle attachment. Similarly at childbirth the arcuate and interpubic ligaments are stretched. Tears in the muscles and ligaments give rise to knots of disintegrated fibrocartilage which become pushed into the ligament lifting it from the periosteum, especially on the dorsal (inner) surface. The bone reactions are similar to those of osteoarthrosis with pitting of

the pubic symphysis articular surface and lipping on the margins (Ullrich, 1975; Angel, 1969). The female metamorphosis of the pubic symphysis lags behind the male in the ages at which the various stages are attained. Therefore, mistaken grading of a pubic symphysis damaged by parturition into an old age group could cause a great overestimation of the female age structure. Todd was unable to detect any differences in rates at which the stages are passed in females known to have born children compared to those known to have had none. However, he does not discuss whether problems of wrong identification from damage were encountered (Todd, 1921).

Acsadi and Nemekeri(1970) have a known age sample of 1045 skeletons aged from 18 - 84 of both sexes. They reduce Todd's ten stages down into a more easily defined set of five stages. They incorporate the use of the pubic symphysis metamorphosis into their complex method.

McKern (1956) and McKern and Stewart (1957) have devised a slightly different grading method based on the same observations of metamophosis. They describe the changes in terms of three components involved in the overall appearance of the pubic symphysis. The three components are:

I The dorsal demiface

II The ventral rampart

III The symphysial rim

They divide each of these into five stages of metamorphosis. The sum of the three grades is then taken and correlated

with the age ranges found in the sample. The division of the overall appearance into three separate components allows for flexibility in the development of the symphysial face and recognises that all the changes as described by Todd may not totally coincide in their stage of metamorphosis. However, there are problems with the interpretation stage because McKern and Stewart (1957) were unable to obtain a widespread age distribution in their original reference population. The method was developed on a large sample compared to Todd (1920,1921) and Acsadi and Nemeskeri (1970) of 349 males, dead from the Korean war which only covered an age range from 17 - early 40s. This leads to the final stages of symphysis metamorphosis being assigned to a group defined as 36 plus. A lack of ability to discriminate amongst the older age groups leads to an age structured population with the bulk of the population having died before forty. Since reaching the anatomically modern form of H. sapiens sapiens , humans have had the potential to live up to the nineties. Certainly, the majority may well have died in the thirties and forties but some will have survived into old age (Biesele and Howell, 1981) , and there is a need to try to identify these individuals and not to concertina the age distribution of past populations into the narrow range from birth to forty (Molleson, pers. comm.). Although the McKern and Stewart method is attractive in its grading method, it should be limited in application to populations which have a similar demographic structure. A military cemetery for example would provide

an ideal situation in which to use the method. In applying the method to a mixed sample the authors noticed anomalies which on closer analysis proved to be the females, supporting Todd's earlier suggestion of sex differences in the metamorphic rates. Gilbert (1971, 1973) and Gilbert and McKern (1973) then presented a form for application to females based on a total sample of 120 from age 19 - 97. They claimed no regular changes were identifiable after 55 once the final breaking down stage was attained, so limited their sample to those between 13 - 57, numbering 103 in all. Unfortunately, it is questionable whether putting the two methods together allows the age identification of a mixed population. Apart from the age limitation of the male sample mentioned above the two methods are not comparable one to another for the same reason of age distribution differences, the females being distinguished up to 55, whereas all males over 36 are clumped together. Ideally, the method needs to be tried and tested and/or developed on a single mixed sample of a wide age distribution. However, in spite of its drawbacks and questionable validity in terms of its reference populations, these two methods have gained widespread use in preference to the Todd method, utilising a simpler identification of stages, based on large reference sample size and devised specifically for calculating ages and presenting the defined age ranges in a clear fashion.

Therefore, there exist four possible methods of

determining age from the pubic symphysis; Todd, Nemeskeri, McKern and Stewart, Gilbert and McKern. Masset (1976) reviews all of these and again emphasises the importance of the age structure of the reference population in influencing the stages described, the age calculations derived therefrom and the resulting age structure of the population under study. Masset (1976) claims the best way to select from the methods on offer is to pick the one with best suited reference population age structure. In the majority of skeletal samples where probably the living population was of mixed sexes and of all ages, this age distribution is best represented by Todd (1920, 1921) or Acsadi and Nemeskeri's (1970) reference populations. Unfortunately, there has so far been little opportunity to test any of the methods' applicability to another population of known age and sex. The methods of McKern and Stewart (1957) and Gilbert and McKern (1973) have been tested on the same reference populations by other researchers not knowing the actual age of the individuals. But this only proves the replicability of the grading process, not the applicability of the methods across populations. The nearest alternative in the absence of many large samples of known age and sex which cover all age ranges in sufficient numbers is to correlate one aging method against others to see how each stands in relation to the overall bioage of the skeleton. A test of this kind was performed by Doukladal (see Masset, 1976) on a Czeck population where little was known except that the population ranged between 18 - 69. As expected from Masset's argument of the importance of the reference

population's age structure, the most suitable results came from the use of Todd's method, where the reference population also most closely resembles Doukladal's Czecks.

As regards the problem raised earlier of possible mistaken grading on female pelves, Gilbert and McKern (1973) claim that "only infrequently" was damage to the pubic bone such as to prevent age assessment at all. They do not expand on exactly how infrequently this occurred. They do, however, acknowledge that the dorsal margin could be damaged such that the dorsal surface becomes pitted resembling stage V in component I (the dorsal demiface) and that it can be difficult to assess whether this state represents components II and III in their developing phase or breaking down phase. So where there is evidence of damage from childbirth, caution and careful observations have to be exercised in assessing the stage of metamorphosis.

As regards possible racial differences in rates of metamorphosis across populations, Todd (1920, 1921) suggests,from his small sample, that there might be a slight difference between whites and negro-whites. Gilbert and McKern (1973) report no evidence of racial differences but again this is based on a very small sample of ten, and they appreciate the possibility that populations living under totally different conditions could vary in metamorphic rate. The only comparative study so far is one by Hanihara (1952) on a Japanese sample. He reports that Todd's ten phases are totally applicable to the Japanese skeletons

as regards the stages through which the Japanese pubic symphysis was observed to pass. However, the changes occur consistently about 2 - 3 years ahead of Todd's sample. It is difficult to know whether this should be seen as a true difference in age-related metamorphosis between the populations or as a constant observer error in interpretation of Todd's descriptive stages. Todd, himself, did recognize that both pubic symphysis metamorphosis and cranial suture closure could be retarded or accelerated by conditions of nutrition, disease etc. so it is to be expected that further work could produce evidence of different rates of metamorphosis between populations. The problem to be solved will be whether these represent purely differences in living circumstances or whether genetically determined differences also exist.

1.5.3 Dental Attrition.

Asseement of age from the degree of wear on the occlusal surface of the teeth, particularly the molars has been long recognized as a good indication in all animals. ("never look a gift horse in the mouth") There are many methods published of grading the extent of tooth wear (Miles, 1963; Brothwell, 1972; Molnar, 1971, 1972; Gustafson, 1950).

The rate at which an occlusal surface wears down can be affected by a host of varied agents e.g. soft enamel, malocclusion, diet, teeth used as tools, tooth grinding etc.

(Cruwys, pers. comm.). Dietary differences stand out as potentially the most importantcause of variation in rates of wear. For reasons of diet, studies of wear in contemporary western populations are often very little use as controls for developing a method of aging on wear. Modern populations have far softer diets than formerly and suffer from dental caries in general rather than dental wear. It has, therefore, proved difficult to find controlled samples for tooth wear studies. Most of the popular methods in use have been developed on prehistoric population samples where age has been estimated from other criteria (Brothwell, 1972; Miles, 1963). This has led to tooth wear grades often being interpreted into very loose age categories of a wide range (Brothwell, 1972). Vast differences in wear patterns have been shown to exist between various prehistoric populations and between present-day non-soft food eating populations such as the hunter-gatherer groups (Lunt, 1978). Eskimos repeatedly turn up in publications as manifesting the highest rates of tooth wear, a feature commonly attributed to the high meat content of their diet, but could equally well be a result of the custom of preparing skin and sinew for use through chewing. Preagricultural groups are shown to exhibit greater wear and lower caries frequency than post-agricultural populations presumably as a result of a heavier wearing diet in contrast to the soft carbohydrate based diets of most agricultural communities (Cassidy, 1972; Campbell 1939). Unfortunately, it is difficult to compare wear rates in many prehistoric populations since the wear rates themselves have been used

as age indicators. The wear rates have to be plotted against some unrelated age assessment in order to gain a true comparison. Even within a population dietary differences can play a highly significant role in wear rates. Older individuals may have different diets to younger; the sexes may differ in their diets and, if the population is socially stratified. the different social levels will almost certainly have differing access to resources e.g. in the amount of meat contained in the diet (Schoeninger, 1979).

Since the influence of diet on rates of wear is very obvious, aging methods based on tooth wear tend to avoid making definite claims about the relationship between chronoage and tooth wear age. Gustafson (1950) presents a very thorough examination of the tooth as a whole, using six criteria, one of which is the degree of tooth wear. He gives 4 grades of tooth wear from 0 (none) to 3, when the pulp cavity is exposed. The other criteria used are the amount of secondary dentine in the pulp cavity, the amount of periodontosis, the amount of cementum, the degree of translucency of the root and the extent of root resorption. The scores for each of these six criteria are added up and a regression equation given for conversion into a chronoage estimate. Using a number of different criteria avoids too great a dependancy on wear alone or on any other one of the criteria but still presents an absolute relationship. Miles (1963) compares the Gustafson method on a sample of 200 teeth of known age with age estimates made by intuitive

guess from their visual appearance. He did not find the method offered any significantly greater accuracy over the visual guess of the experience observer: 34% were good estimates i.e. within 3 years of the real age, by the intuitive method and only 38% by Gustafson's method.

Brothwell, in "Digging up Bones", presents a chart of stages of tooth wear, giving up to six stages. He then gives an interpretation chart grouping together stages of wear on the three molars into large overall age categories:
17 - 25; 25 - 35; 35 - 45; 45 +
All the older individuals are classed together in a single bracket with no means of distinguishing between them, again giving a possibly false contraction of the age distribution or at least an impression thereof.

The Miles method af aging presents the best solution by avoiding absolute relationships altogether (Nowell, 1978). Since wear starts as soon as the tooth has erupted Miles argues that the rate at which wear is proceeding in a population can be gauged be measuring the amount of wear on M_1 by the eruption time of M_2. The comparison of M_2 with M_3 can also be made although this is less reliable since the eruption time of M_3 is so variable. Miles develops his method on a series of Anglo-Saxon skulls numbering 190 in all and treats the 38 with immature dentition as those of known age in which the scale is built up. Although the rate of wear may well change throughout life, the attempt to callibrate the wear rates in early life at least sets

a standard for any particular population in relation to other populations. Any of the schemes of tooth wear grading can be used and calibrated in the way Miles suggests. Again there is a need for a skeletal sample of known age and sex with a wide age distribution to test out the applicability of the method. There is a need for more information of the variation between populations and within populations on wear rates, e.g. the time taken to pass from one stage to another may not be constant across populations, the sexes or social levels. Both Brothwell and Miles report no evidence of sex differences in wear patterns but this is certainly not always going to be true.

1.5.4 Bone Structure.

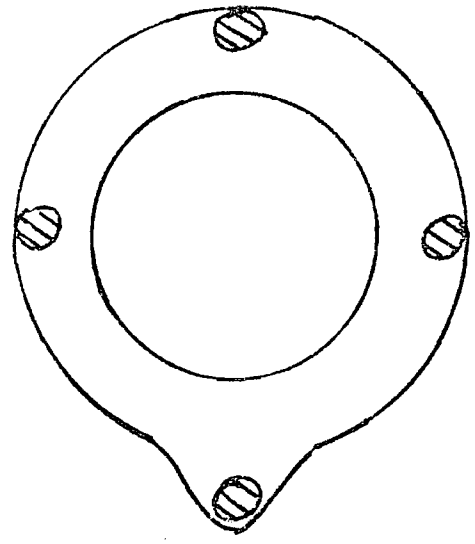
Acsadi and Nemeskeri (1970) have examined changes in the appearance of the head of the femur and humerus. They present five stages of reduction in amount of trabeculae within the bone head as seen by radiography. The grading is used in association with their other aging parameters to form the complex method of age assessment. Distinguishing the five stages from radiographs may be more difficult in practise than suggested in theory (Molleson, pers. comm.).

A number of methods have been developed from measures of cortical thickness and internal cellular structures of cortical bone.

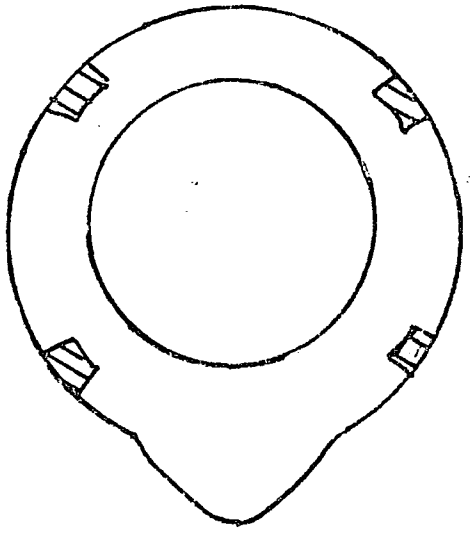
Kerley (1965, 1969) presented the first method of estimating age from bone sections. He had a sample of 126 individuals ranging in age from birth to ninety-five and he studied sections from the femur, tibia and fibula. Four parameters were measured in each of four fields located on the periosteal edge of the section (see Fig. 1.1). The four parameters were the number of osteons, number of osteon fragments, number of non-haversian canals and percentage of circumferential lamellar bone. A regression equation for converting each of the measures to an age estimate is given, together with the range of accuracy. The best single parameter is the number of osteon fragments in the fibula. Using all four measures in conjunction, the four estimates for any one bone, or more if more than one bone is used, can be considered together and the overlap region of the estimates used to provide a more precise age assessment. Kerley tested his method on samples of known age and was able to estimate the age of all of them to within ten years. He found no sex differences in any of the four measures.

Ahlquist and Damsten (1969) published a modification of Kerley's method, simplifying the method by focussing on a single parameter in the femur alone. They discuss the difficulties encountered using Kerley's method, particularly in distinguishing osteons from osteon fragments in the rough estimation of percentage of circumferential lamellar bone by eye and in identifying a structure on the edge of the field without moving the

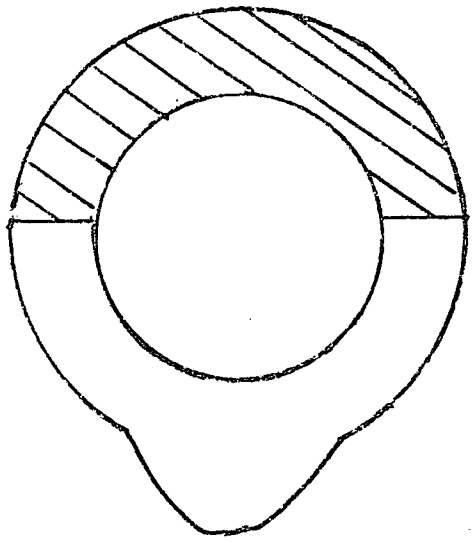
FIG. 1·1 FIELDS USED IN THE METHODS OF AGE ASSESSMENT FROM BONE STRUCTURE



KERLEY



AHLQUIST AND DAMSTEN



SINGH AND GUNBERG

field. As Kerley's parameters can be grouped into those of increasing frequency with age, that is the numbers of osteons and osteon fragments, and those of decreasing frequency, that is the number of non-haversian canals and the percentage of circumferential lamellae, Ahlquist and Damsten measure only the percentage of the field occupied by osteons and osteon fragmennts, an inverse measure of Kerley's percentage of circumferential lamellae. Instead of estimating this percentage roughly by eye, they make use of a 10 x 10 square grid micrometer and count the number of squares out of the hundred more than half covered by haversian systems. This gives a more accurate estimate of the percentage, allows peripheral structures to be identified and does not necessitate any discrimination between osteons and fragments. They make use of four fields from femoral cross-sections, located slightly differently to Kerley, but still on the outer periosteal edge of the bone (see Fig. 1.1). Tested on a sample of 20 of known age, they estimate the accuracy of the method as ± 6.71, which is better than most of Kerley's ranges for any single parameter. The authors feel that in comparison to Kerley's results using all the parameters in which he was able to place all his test cases within 10 years, their method would be inferior. Nevertheless, the greater simplicity, speed and lack of necessity for clear details in the section give the method great practical advantages (Ubelaker, 1974).

The third method is presented by Singh and Gunberg (1970) based on sections from the femur, tibia and mandibular ramus using three parameters. In the long bones a section is taken from the midshaft anterior portion (see Fig. 1.1). The measurements are made on two fields taken at random from the outer third of the periosteum. The total number of osteons from the two fields are counted and the mean haversian canal diameter calculated. The third parameter is the mean number of lamellae per osteon. This last parameter demands very clear details in the sections in order to count the lamellae and unfortunately most archaeological specimens do not present such clarity of detail. Singh and Gunberg's sample of 59 is mainly male (number = 52) and, although the studies of Kerley showed no sex differences for his parameters, the application of the method to females has not been truly tested. In addition the sample was all over fifty years of age with a mean age in the sixties. The regression equations allow the parameters to be used singly or in combination. The method cites a resolution of estimate of ± 3 years.

The method developed by Thompson (1978, 1980) is a result of a thorough analysis of age-related changes in bone looking at a total of nineteen variables on the femur, tibia, ulna and humerus. He presents a series of step-wise equations for the sexes together and apart for each of the bones studied. The five important variables for age estimation from the femur with the sexes together, are cortical thickness, number of osteons, percentage area

of osteons, aggregate osteon perimeter and the individual osteon perimeter. The resolution of the age estimates are from ±9 years to ±7 years. The reference sample consists of 116 individuals of both sexes in equal proportions. However, the ranges of age are predominantly in the older age groups with mean ages in the seventies for both sexes. This, therefore, does not offer a good sample across all ages.

The most recent method has been developed by Samson (1983). His reference sample suffers from the same preponderence of old individuals, particularly in females. He makes use of the measures of the number of secondary osteons and the mean haversian canal diameter. His method is applicable to males only, as he found no measures, or combination thereof, provided an adequate age estimate in females. The method gives a resolution of ±6 years. This method again does not demand great clarity of detail from the section. Samson (1983) applied his method to the males from two archaeological samples and found good correspondence with the general age categories provided from other criteria of aging.

1.5.5 Degenerative Joint Disease of the Spine.

The growth of bony lips or osteophytes from the margins of the vertebral bodies is well documented to correlate with chronological age. However, osteophytosis, as this particular form of degenerative joint disease is

often called, is known to be influenced by many other possible local factors, particularly mechanical stresses on the spine. In the absence of other methods for aging older individuals the development of osteophytes could offer a means for making some differentiation amongst the older age groups, even if only into large general categories comparable to those given by Brothwell for dental attrition.

Stewart (1958) studied a large sample of spines of known age from the Terry collection and the Korean war dead:

87	male	38 - 84	368	male	17 - 50
17	female				

Unfortunately, as so often, there was only a very small sample of old individuals, and even fewer females. However, Stewart was able to make some assessment of its potential use and limitations. It was recognized that a small proportion of a population will never develop osteophytes even by the eighth decade and some individuals may develop severe forms unusually young. Stewart notes that an absence of his second severity grade will most likely indicate an individual under thirty while the presence of grades 2 and 3 will be characteristic of the forty plus age group. The intensification of the osteophyte development is not, however, necessarily manifested by an increased severity indicated by larger osteophyte formation but rather by an increase in the number of segments involved. Stewart (1958) makes this interesting observation but does

not follow it up as a means of aging but continues to refer to severity. Stewart (1958) also mentions that the facet joints of the spine become more and more involved after forty, but again does not follow this observation up in specific discussions of using the degenerative changes for aging purposes.

In studying spondylolysis in Eskimo spines, Stewart (1953) ages his sample into rough age categories on the basis of osteophytic formation. He does, however, assume an age of onset, comparable to a white population, of 30 and a moderately severe stage as occurring by 40. His categories are broad and do not differentiate individuals beyond 40. It may be possible to adapt a Miles-style strategy of callibrating the age of onset against other aging methods in younger individuals to assess the rate of development specific for the study population. The idea of increased involvement of joint number could be followed up to make a wider scale, and more specific ideas of the variation within a population of the age-related degenerative changes.

1.6 Overview of Aging Methods.

Various assumptions are made in the development of methods of age determination and their application across a range of populations. Aging methods based on quantitative data provide regression equations to compute the chronoage from the bioage measures. This is the case in the age

determination methods based on measures of bone structure. The use of regression equations assumes the age-related changes of bone structure proceed regularly throughout all stages of life, known not to be true, but seen as an acceptable compromise losing some accuracy in favour of simplicity.

The qualitative methods based on visual assessment suffer from a similar problem. The length of time necessary for a sufficient degree of change to occur that visibly distinguishes one category from another can be assessed in the modern control population without forcing a false pattern of steadily continuous change as in the quantitative methods. However, the equivalent problem is that the relative times taken for the changes to occur from category A to category B to category C may be totally different in populations other than the reference population. This is seen in the use of dental attrition and pubic symphysis metamorphosis. The need for some means of internal callibration for any population along the lines of the Miles method of dental attrition is thus emphasized. This would apply to any use of degenerative joint disease also.

Many of both the qualitative and quantitative methods make no distinction between the sexes. Again the assumption of identical biological aging processes across both sexes in some cases is justified by simplicity if differences are not great. However, because there are no great differences in the modern control sample there is no guarentee that this

is equally true in another population. Again, some means of callibration against age estimates from the more accurate subadult measures could be developed.

The various methods make use of different skeletal elements from one another. The relative rates of aging of these different parts one to another will not necessarily be a constant across populations nor across the sexes. This means that the method with the best correlation of bioage measures with chronoage in the control sample may be totally inadequate in another situation and population. However, comparisons of one method with another could provide a means of assessing population specific rates of change of any single measure thus providing some level of callibration, even if only relatively, in older groups. Alternatively, the use of many methods can provide a number of ranges of age estimates from which a narrower range can be derived through overlap.

Most reprts of mortality in a population appear to use only one or two methods. The complex method of Acsadi and Nemeskeri (1970) is an exception to this, using four measures of age, cranial sutures, pubic symphysis, and the proximal epiphyses of the humerus and femur. The complex method was recommended by the European conference (1981) as a common practice in an attempt to promote some kind of consensus internationally in approach to age assessment.

In summary, a broader scan of age estimates would provide the needed information on differences of biological aging measures between the sexes and to one another, can indicate the reliability of any particular method for the specific population and can complement one another, thus reducing the resolutions of an age estimate.

Chapter 2. BONE STRUCTURE AND AGE-RELATED CHANGES.

2.1 Anatomy.

Bone structure can be studied at two levels. The external level concerns features of gross morphology such as size, shape, weight and overall density, whereas the internal level involves parameters of the microscopic structures within the bone such as measures of cellular structures (Villanueva et al., 1963).

There are two forms of bone in the skeleton: cortical or compact bone made of dense solid units such as that found in the tubular shafts of the long bones, and cancellous or trabecular bone, which has a more open structure with bone formed in strands called trabeculae. This kind of bone is found particularly in the long bone heads and in the vertebral bodies. The bones most widely used for age assessments from their structure are the long bones. These have been studied for age-related changes at both external and internal levels. The long bones of the skeleton are the femur, tibia, fibula, humerus, radius, ulna and clavicle and are all built on the same basic design of a long tubular structure.

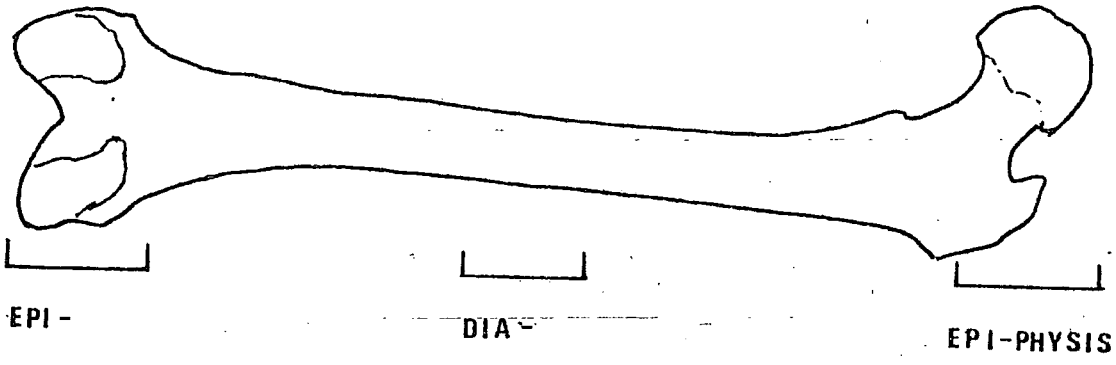
In the embryo the long bones are first represented by a cartilaginous model which then ossifies from the centre

outwards in both directions length-wise. Other centres of ossification in the embryo arise in the epiphyses, which are bone caps at each end of the bone, separated from the bone by a cartilage endplate. At birth, the cartilage model of the long bone has completely degenerated and been replaced by bone. The bones then continue to expand in length by bone deposition beneath the cartilage endplates until the final adult length is attained in adolescence. At this point, the epiphyseal bone caps fuse onto the main bone shaft and lengthwise growth ceases.

The middle portion of the bone shaft is termed the diaphysis and the areas between the diaphysis and epiphyses are called the metaphyses (see Fig. 2.1). The bulk of the bone of the shaft is the dense, cortical bone. There is only a small amount of cancellous bone located mainly at the epiphyseal ends. The central cavity houses the bone marrow and is called the medullary cavity. In cross-section the cortical bone of the shaft is seen as sandwiched between the outer and inner borders referred to respectively as the periosteal and endosteal surfaces (see Fig. 2.1).

Thin cross-sections can be taken from the bone shaft, ground down and examined microscopically for details of the internal bone structure (Frost, 1958). The first bone, formed in the embryo is called woven bone. This has relatively large vascular spaces and the bone cells are randomly arranged in the bone (see Fig. 2.2). This form is soon replaced by bone formed in layers, or sheets called

FIG. 2·1 LONG BONE ANATOMY



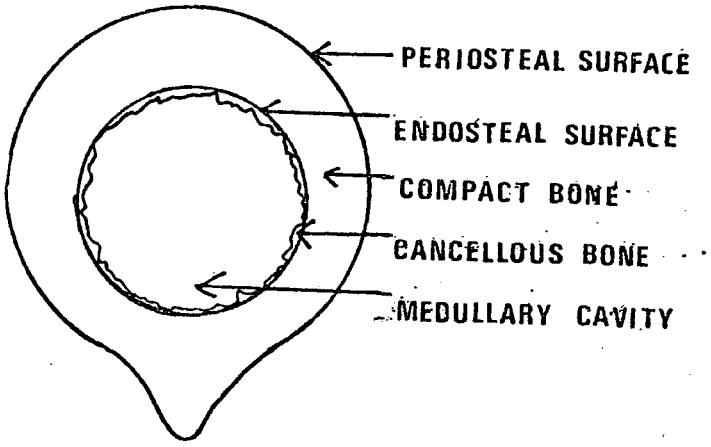
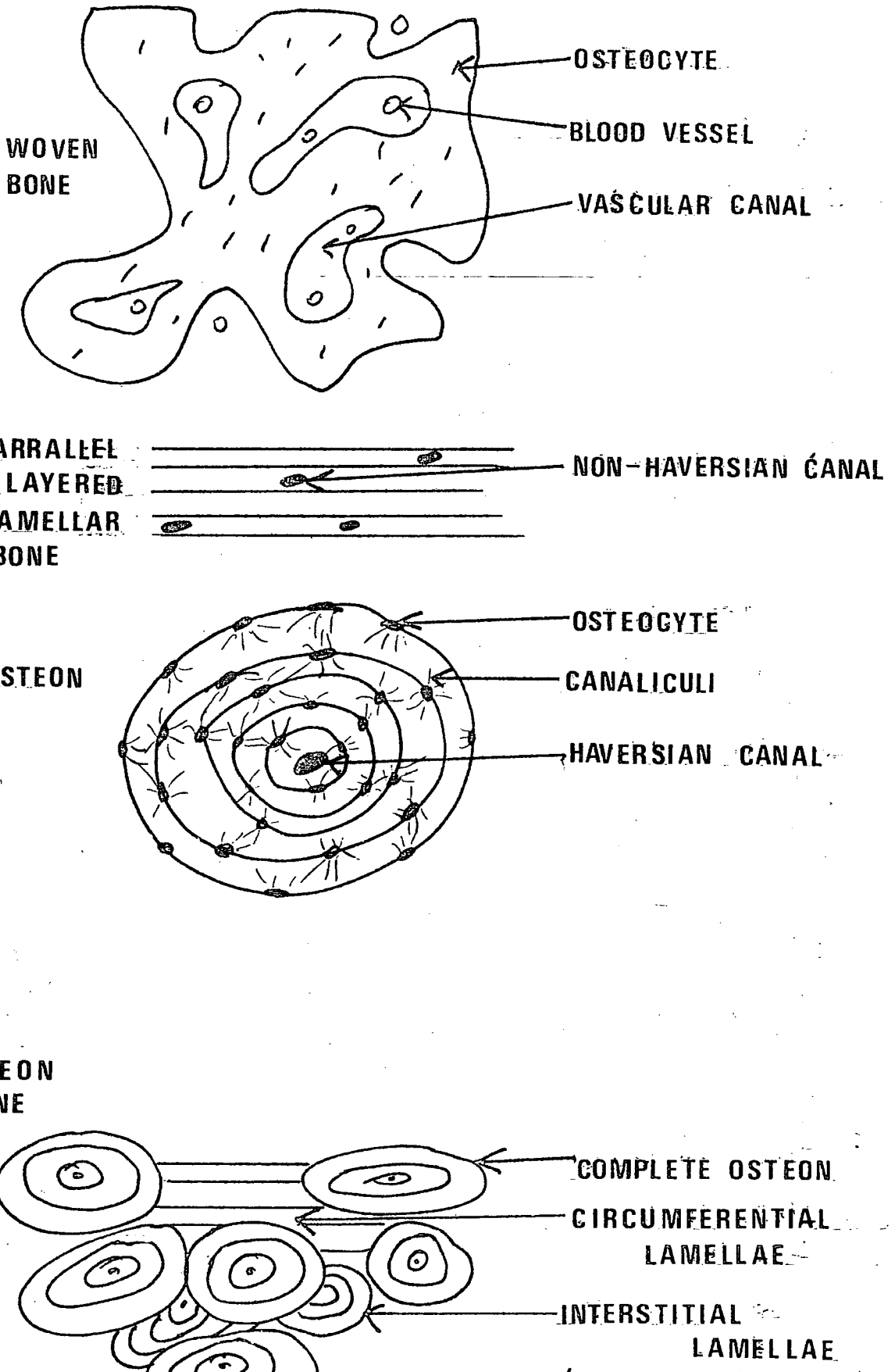


FIG. 2·2 INTERNAL BONE STRUCTURE



lamellar bone. In the human long bones woven bone is totally replaced by lamellar bone by two to three years of age. The layers of sheets of bone run parallel to one another and are visibly distinct as the orientation of the collagen fibres is different in adjacent layers or lamellae (Portigliatti et al., 1983). The first lamellar bone is laid down in straight, parallel sheets with vascular canals, known as non-haversian canals, scattered throughout the bone (see Fig. 2.2).

The characteristic adult human bone which replaces the straight-lined lamellae of childhood, has vascular canals called Haversian canals with the parallel lamellae and bone cells arranged concentrically around it forming the Haversian system or secondary osteon (see Fig. 2.2). In adult bone Haversian systems can be seen along with remnants of the childhood straight-lined lamellae, referred to as circumferential lamellae (Kerley, 1965,1969). Similarly complete Haversian systems are found together with remnants of previous Haversian systems that have been partially resorbed where the new system has been formed. These remnants are called interstitial lamellae or osteon fragments (Kerley, 1965,1969) (see Fig. 2.2).

The processes called bone remodelling, whereby bone is resorbed and new bone deposited, begin in the embryo as soon as the first woven bone has been formed and would appear to serve at least three functions. Primarily, it permits alterations in the bone structure to respond dynamically

to mechanical demands placed upon it both during the course of growth and subsequently throughout adult life. Secondly, the bone provides a mineral store from which minerals can be resorbed into the blood stream when required (Urist, 1964). Finally, areas where remodelling occurs are often those showing natural, non-pathological bone necrosis. Remodelling, thus, allows replacement or regeneration at these sites (Enlow, 1962).

The first of these functions draws upon the principle known as Wolff's Law. This, simply stated, proposes that bone will be resorbed where pressure is placed against it, that is under compressions and will be formed in response to pull, that is under tension (Lovejoy et al., 1976). Evidence supporting this hypothesis comes from many studies of bone structure and shape at the external level (Lovejoy et al., 1976; Jungers and Minns, 1979; Currey, 1968; Amtmann, 1971). There is a growing body of evidence that the Haversian systems are also involved in response to stress at the internal level. Portigliatti et al. (1983) studied the distribution in femoral cross-sections of Haversian systems classified into types by the predominant orientation of their collagen fibre bundles, an important component of all connective tissues. Those with longitudinally arranged bundles appear dark under polarizing light, whereas those transversally orientated appear light. Longitudinally orientated collagen fibre bundles are resistent to compression. Similarly, compressive forces operate on the femur bone shaft medially and

posteriorly while tensile forces operate laterally and anteriorly. A prevalence of transverse types of collagen bundles were found in the compression regions and the longitudinal type in the areas of tension, thus supporting the hypothesis that internal structure also responds to mechanical demands. In addition, the level of calcification in cross-section was found to be lower in the areas of tension rather than in the compression areas. Low calcification is usually interpreted as representing new bone formation, so Wolff's Law is vindicated by evidence of new bone formation being stimulated by tensile rather than compressive forces in internal structure (Portigliatti et al., 1983).

The second function of bone as a storage system for minerals directly explains a function of the Haversian system rather than bone in general. The study of the evolution of bone through examination of structure of the earliest skeletal animals, suggests that the first were those with exoskeletons. These did not have a capacity for resorbing bone, although the form of bone was of a lamella nature (Frost, 1964). Without resorption, a reliance for minerals is placed on a chemical continuity with the external environment forming what is termed the open-cycle chemical system. This is all very well if the external environment contains the required minerals. Even then much energy is put into filtering out the needed minerals as against anything else present. The use of resorption from the skeletal store appears to offer a more economical means

of gaining minerals, particularly rare ones such as phosphorous. The bone forms a continuum with the body fluid in this arrangement, the two operating as a single physiological unit in a closed-cycle system (Urist, 1964). The primacy of resorption over formation, in most modern vertebrates, in the sense that resorption always preceds the formation of bone further points to the evolution of remodelling as a means of getting at minerals and then replacing the store where possible (Frost, 1964).

The association observed by Enlow (1962) between necrosis and remodelling strongly suggests the third role of remodelling as a regenerative process.

Chemically, bone has two main components: the organic which comprises the bone matrix and the inorganic of bone minerals. In the process of bone formation, the bone matrix is laid down first, forming the basic framework within which mineralization takes place, producing the characteristic rock-hard appearance of bone. The combination of these two parts, organic and inorganic, acts as a composite material in the same way as fibre-glass, conferring properties of elasticity and strength to the bone beyond those of either component individually (Currey, 1962).

The bone matrix is composed of 2 substances, the major portion being the fibrillar protein collagen, and the rest the so-called ground substance. Collagen represents

about 95% of the bulk of the bone tissue and it is the collagen framework more than any other part of the bone matrix which gives a bone its shape. Collagen is formed by aggregations of the macromolecule, tropocollagen. This is made up of three polypeptide chains which spiral to the left around each other forming a triple helix. The entire helix in turn spirals to the right around a common central axis. Two of the chains are the same and are referred to as alpha-one chains whereas the third has a higher proportion of basic amino acids and is known as the alpha-two chain. The amino acid residues of the polypeptide chains can be charged positive, negative or neutral. The distribution of charge along the tropocollagen molecule is asymmetrical giving it a top and a tail and creating five bonding and 4 non-bonding zones along its length. The tropocollagen molecules aggregate side-by-side length-wise with the charged regions being aligned. Any one bonding zone of one molecule can bind with any one of the adjacent molecules allowing some flexibility to the protein's morphology. This alignment gives collagen its fibrillar nature.

Analysis of the ground substance breaks it down into a non-collagenous protein and a complex of mucopoly-saccharides, mainly chondroitin-4-sulphate, chondroitin-6-sulphate, keratan sulphate and sialic acid. It is believed that changes take place in the ground substance in areas of the bone matrix where mineralization is about to commence and where bone matrix is to be resorbed. So although the

function of the ground substance is not yet understood, it would appear to play some kind of role in the mineralization of bone. It may also serve to provide molecular bridges between collagen fibrils, a role demonstrated for ground substance in the cornea which may prove true ubiquitously in collagen of connective tissues. A further suggestion is that as the ground substance seems the best candidate for holding and releasing water because of its all-pervading nature, it could serve as the agency through which the water content of bone matrix is regulated.

The mineral portion of the bone is made up of calcium, phosphate and hydroxyl ions and the structure of the needle-shaped bone crystals resembles that of the apatites. Bone mineral is variable in its structure for a number of reasons. First, calcium can be replaced by other elements such as strontium, barium or magnesium and likewise hydroxyl can be replaced by bicarbonate or fluoride. Secondly, the crystals formed are exceptionally small and thus have a large surface area relative to their mass which allows various ions to attach to the surface, again giving a variable composition to the bone. Nevertheless, the basic formula for the bone matrix can be given as hydroxyapatite:

$$Ca_{10}(PO_4)_6(OH)_2$$

and the process of mineralization can be viewed as one essentially of calcification. There is a time lag between the deposition of bone matrix and its calcification. The bone crystals develop within the collagen framework surrounding and impregnating the collagen fibres and there is

some evidence of direct co-valent bonds between the two. The factors initiating and regulating the development of the crystals are little understood as yet. It seems likely that collagen plays a large part, but the exact role, whether active or passive is unknown.

The factor which must be seen as of prime significance is the cell responsible for bone formation, the osteoblast. This bone-forming cell is believed to secrete bone matrix and possibly orientate the collagen fibres during this process. Again its exact role on the calcification process is unknown. As bone deposition is in progress, osteoblasts may become trapped in the growing bone and are then known as osteocytes. These cells, set within the bone in lacunae, at relatively great distances from the blood vessels would appear to face difficulties in obtaining nutrients and in disposing of waste products. However, they appear to communicate with one another and ultimately with the blood vessels by small processes called canaliculi. The appearance of osteocytes varies from those which greatly resemble the osteoblast to those which have very little development of the cellular organelles. The more deeply placed and presumably oldest osteocytes are those with the least developed organelles and it is assumed that the younger, more developed osteocytes have osteoblast functions of secretion, whereas the older have become more inactive. It has also been suggested that osteocytes may change to perform an opposite function to the osteoblast, that is a resorption of bone matrix and mineral (Ortner, 1975;

Belanger et al., 1963). The bone cells recognized as responsible for bone resorption are referred to as osteoclasts (Blackwood, 1964; Bourne, 1971; Frost, 1964; Hancox, 1972; MacLean and Urist, 1968).

The processes of remodelling at the two levels, although interrelated exhibit some degree of independent action, as discussed by Villanueva et al. (1963). External gross shape from growth is completed around 17 - 20 years of age and although remodelling from endosteal and periosteal actia continues to provide modifications these are very minor compared to the changes in shape and length of childhood growth. The age of fusion of the epiphyses at early adulthood marks the end of the growth phase and constitutes a major point of change in types of external remodelling. Internally, by contrast, there is no such dramatic change at 17 - 20, but a later point at around 35, where a reversal in remodelling trends is observed (Villanueva et al., 1963). On the other hand, observed changes in gross density throughout life could be explained by both external alterations and internal processes, indicating a direct relationship between internal and external - density being counting as an external parameter (Thompson, 1980). However, most find it useful to treat the two levels separately for analytical purposes and this convention will be followed here.

2.2 Internal Remodelling.

At internal level, the processes of resorption and formation of bone can be studied at the level of the cells responsible for these activities. There exist a population of precursor cells, or osteoprogenitor cells mesenchymal in nature which do not resorb or form bone themselves but carry both these potentials and can be stimulated into action. In the formation of a Haversian system, bone is first resorbed, the osteoprogenitor cells stimulated into resorptive activity as osteoclast cells. Resorption is focal in nature and it is thought that the activity of a resorptive focus will inhibit production of new foci in the vicinity in a negative feedback system (Sedlin et al., 1963). Resorption occurs either from osteoclasia moving centrifugally in a pre-existing Haversian canal, or else proceeds into the cortex from the periosteal or endosteal surface. The resorptive activity progresses rapidly, once begun, to form a large space the size of the future osteon (Sedlin et al., 1963). Then there follows a transitional period of inactivity between the completion of resorption and the start of new bone matrix formation. The final stage is the onset of the apposition of the new bone matrix. Bone is formed by osteoblast cells, derived from the osteoprogenitor population. Matrix is formed first followed, about fourteen days later, by the commencement of mineralization (Villanueva et al., 1963). The layer of unmineralized bone matrix is referred to as the osteoid seam and is characteristic of forming osteons.

The mechanism by which osteoprogenitor cells are stimulated into osteoclastic or osteoblastic activity is not at all understood. Basset (1964) stresses the necessity to have both stimulus and available nutrients for osteoblastic activity. Stimuli may come from biomechanical action or from chemical sources. Bone crystals share with other crystals the property of piezoelectricity, so that stimulation of bone remodelling could arise from stress or injury induced electric potentials (Korostoff, 1977; Jendrucko et al., 1977; Pfeiffer, 1977a, 1977b; Bassett, 1964). This would fit neatly with the observed increased remodelling in areas of tensile stress and greater bone formation. The cells appear to have a negative feedback process whereby resorption halts after a certain degree has occurred. It is suggested that the stimuli to osteoblastic action could come directly from the decrease in activity of the osteoclastic cells, Resorption and formation are not normally seen simultaneously in the same focus, except in the case of the so-called waltzing cell, where resorption is occurring on one side of the system while new bone is being formed on the other (Sedlin et al., 1963). Enlow (1962), as mentioned earlier, suggests dead cells may have the potential to stimulate remodelling.

Hormonal influences can be seen to be involved at the level of providing nutrients for bone formation and may well be responsible for stimulating resorption when the body fluids are low in minerals. The parathyroid hormone - vitamin D complex is involved in the regulation of calcium ion

concentration and calcium balance, and oestrogen is involved in the control of the synthesis of calcium - yolk - protein complexes. These two hormones are found in vertebrates having bone remodelling skeletal processes and are likely to be factors in remodelling control. A suggested connection between gonadal hormones and bone cell activity is discussed by Villanueva et al. (1963). The gonadal output is inversely related to the number of osteoid seams, so if such a relationship exists it would be one of a suppressive action of the hormones on bone remodelling.

Over any given area of bone or in the entire skeleton, the amount of resorption and/or formation and therefore of overall level of bone mineral present can be seen as the outcome of several contributory factors. The quantity of bone being resorbed at any time depends on the numbers of new foci being introduced, the rate at which each focus is produced and the size of the focus (Sedlin et al., 1963). Within any single resorption space, its size will in turn depend on the number of osteoclastic nuclei, the rate at which these resorb bone and the functional lifetime of these nuclei (Landeros and Frost, 1964). Formation of bone depends on equivalent features of osteoblastic action. The overall product in terms of amount of bone is a result of a balance of these factors.

It is well known that a feature of increasing age is a large amount of bone loss seen from external observations of bone (see later). Age studies of bone changes from the

internal level have also been made with a view to explaining relationships between resorption and formation and the changing nature of these relationships throughout life. Microradiography provides information on the degree of mineralization of an osteon system. Amprino (1963) relates the degree of mineralization to the age of the osteon, but recognizes that there might be differing rates of mineralization throughout life giving a false impression of new bone formation. Jowsey (1960) studied a small sample of 24 of femoral cortical sections from the diaphysis. Young individuals exhibited a large number of resorption spaces and a high % of low mineralized cells indicating a high bone turnover rate. Young adults, by comparison, exhibit a remarkably low bone turnover rate, with very few of the Haversian systems showing mineralization at a level less than 75% of full mineralization. The later age groups manifest a gradual increase in the amount of resorption, especially endosteally. By 70 years of age, up to 25% of the endosteal surface was observed to be under resorption. There is very little evidence of concomitant new bone formation with few osteons of less than 75% of full mineralization. The bone shows great local variation in mineral density and porosity. The observations of plugged canals and lacunae, representing hypermineralization, are low in frequency in young adults. In old ages, there are a considerable number, particularly in the periosteal portion of the bone. Nonetheless, some elderly individuals show none of these plugged structures at all.

Similar age-related changes are recorded by Frost and his associates in their many publications. Villanueva et al., (1963) recorded the number of active osteoid seams per mm^3 of bone tissue throughout life, representing the number of remodelling foci. Their sample was from the ribs of 78 males and 62 females aged between one month and eighty-four years. The number was highest in infancy, gradually decreased to the lowest point in the mid - late thirties followed by an increase to a second peak in the seventies. No differences were explicable by sex or origin of the sample, so appeared to be purely age-mediated. Sedlin et al., (1963) concentrated on the number of resorption spaces and type. Type was classed as 1. resorbing 2. transitional and 3. starting formation. They report a similar pattern of a frequency that is high in infancy, decreasing to thirty-five followed by an increase in older ages. The completed resorption spaces (type 2) are lowest in childhood, but constant in the rest of life. The early resorptive stage decreases in relative frequency throughout life, while the final stage is highest in infancy, decreases to the late thirties, undergoes a slight increase thereafter then decreases again in the oldest ages. The overall picture, therefore, is of rapid osteoclasia in the child. The increased % of early foci in older individuals suggests the development rate of resorption foci slows with age. This is presumably from either a decrease in the efficiency or in the number of osteoclasts. The relationship in absolute numbers or the type 3 spaces to the type 2 suggests an increase in the lag phase with age between completion of

the space and initiation of matrix formation. However, such an increase in time from the beginning of resorption to the beginning of formation is not sufficient to explain the observed increase in numbers of resorption foci with age, therefore a real increase of resorption activity appears to be occurring with age. Jowsey et al. (1965) also examined the relationship between resorption and formation in a number of different bones and reported a decrease in turnover up to the thirties, then an increase in both resorption and formation in the forties and fifties, consistent with the slight increase in type 3 spaces of Sedlin et al. (1963). After fifty, formation was observed to remain constant while resorption increased by as much as a factor of two.

Sedlin (1964) reports that counts of dead osteocytes in ribs revealed that the number is lowest in infancy, increases up to the mid-thirties and then remains relatively stable. There is no evidence whether this represents a cause or effect of the remodelling process, but it does appear that a stable relationship of living to dead cells is achieved through middle and old age.

These studies of bone turnover are summarized in Table 2.1. The overall picture is of high bone turnover in children, much resorption followed immediately by formation and soon resorbed again. Turnover decreases to the thirties, both resorption and formation very low in activity. A lag is thought to develop between resorption and

Table 2.1. Age-related Changes in Internal Observations of Bone Turnover.

Age Group	Bone Activity
Children	HIGH AND RAPID TURNOVER: Large no. of resorption-spaces High % of low mineralized osteons High no. of osteoid seams High no. type 1 resorption spaces High no. type 3 resorption spaces Low no. type 2 resorption spaces Low no. dead osteocytes
Young Adults (to 35 - 40)	VERY LOW TURNOVER: High % of high mineralized osteons Low no. of osteoid seams Low no. of type 1 resorption spaces Low no. of type 3 resorption spaces Higher no. of dead osteocytes INCREASE OF LAG BETWEEN RESORPTION AND FORMATION: Increase in type 2 resorption spaces
Middle Adults (40 - 50)	INCREASE IN TURNOVER: Increase in no. of osteoid seams Increase in type 3 resorption spaces Increase of both formation and resorption
Old Adults	INCREASE RESORPTION OVER FORMATION: Increase of resorption (endosteally) High% of high mineralized osteons Resorption increase by x2 Formation constant SLOWING OF FORMATION: smaller daily increments of bone deposition

formation. A slight increase is observed in both resorption and formation, but whereas resorption continues to increase in activity, particularly at the endosteal surface, formation does not, so a real increase in resorption is seen and therefore bone loss after forty-five.

Frost (1963) used tetracycline labelling to measure the amount of new bone formed per day from the mean daily decrease in the diameter of the Haversian canals of actively forming osteons. He found that the mean values of the diameters were relatively constant throughout a fifty-five year span studied in the ribs. The mean canal diameter in a mature osteon (one completely formed) was 46μ and at the commencement of formation is 192μ. The bones of the appendicular skeleton were uniformly about 25% larger. Although the size of a mature canal is constant through life, the time taken for it to form differs with age. The amount of new bone formed per day was 1·57μ at age 7·5, thereby needing 42 days to reach completion. By 43 the amount of bone laid down per day had decreased by 40% to 0·93μ, requiring 79 days for complete formation. This finding corresponds to the report of a delay between resorption and formation, further indication that the whole turnover process of resorption and formation takes longer with increasing age.

Finally as regards turnover patterns, Kelin and Frost (1964) plotted the circumference measures of osteoid seams against the frequency with which each was found for each

decade up to eighty. They found no differences between the curves found for each of the eight decades. This suggests that little age change occurs in properties of the osteoblast in composite, that is in the actual formation process. This is suggested also by the constant size of a fully formed osteon canal throughout life described above. Changes in the bone cell system related to age are proposed to be located in the population of osteoprogenitor cells, rather than in the active bone forming cells.

Different bones exhibit certain absolute differences. Jowsey et al. (1965) note the greater turnover in the anterior iliac crest compared to the posterior crest, the femoral mid-shaft and neck, ribs and vertebrae. The rib differs from the others by not exhibiting the noticeable increase in resorption after 50. Barer and Jowsey (1967) report instead a relatively constant relation between formation and resorption, but one where resorption is constantly higher by about 1% from 20 - 80 years of age. Frost (1963) found the mean size of osteons to be 25% larger in the appendicular skeleton compared to the ribs.

Jowsey (1960) compared different parts of the same femoral cross-section in a study of degree of mineralization. The cross-section was divided first into quadrants, then each quadrant into thirds. Thirteen cases examined for all parts with no significant differences between the thirds or the quadrants. The section was also divided into thirds around parallel to the perimeter

giving a periosteal/middle/endosteal division. Differences are noted between these three with resorption being particularly high in the endosteal region of older subjects and the periosteal region having a higher level of hypermineralization.

Although a number of the studies discussed describe the sample in terms of number of males and females no discussion is made of sex differences, by contrast to those of external bone change. Villanueva et al. (1963) alone state that no sex differences were present. It is uncertain whether the absence of discussion of sex differences in other papers implies there are none or that this has not been investigated.

Bone remodelling can be affected by a number of disease processes. Rickets involves the presence of wide borders of uncalcified matrix. This represents the opposite extreme to hypermineralization mentioned earlier and occurs in the same sites, around osteocytes and canaliculi implying these are significant sites for the process of mineralization given the necessary nutrients of calcium and phosphorous (Jowsey, 1964). In Paget's disease, bone turnover is very high (Jowsey et al., 1965). Formation of bone is decreased in Cushing's disease and after the administration of corticosteroids, but the resorption levels undergo a threefold increase so are still the important factor in bone loss (Jowsey et al., 1965). Landeros and Frost (1964) examine the remodelling of bone in sufferers from diabetes

mellitus. The average size of a diabetic osteon is normal, but the rate of closure as it forms is longer than normal. They calculate the slower rate of formation is 2·8 x the normal rate at any age. The disease appears to depress the rate of bone formation. Sedlin et al. (1963) selecting a normal sample excluded cases on the following grounds, recognizing their possible influence on bone structure changes: chronic renal, hepatic, pulmonary, endocrine or febrile disease, congestive heart failure, disorder in calcium or phosphorous metabolism, bed-ridden one week or more before sampling, or recipients of radiotherapy, hormonal or cytotoxic agents. The best documented pathology involving bone loss is osteoporosis. Jowsey et al. (1965) record a greater level of resorption in osteoporotic than in normal aged individuals, whereas formation remains equal in both groups (Jowsey, 1964). Further discussions of factors involved in osteoporosis will be made at the end of the next section on external remodelling.

2.3 Internal Remodelling for Aging Skeletons.

Changes are seen to occur systematically with age in the processes of internal remodelling. However, the age-related changes are not constantly in the same direction with increasing age. On the contrary, the change in turnover rate decreases with age to the mid-thirties but then increases for a short time before the final stage of imbalance between the two components with a vastly greater increase in resorption. Methods developed on samples of

predominantly older individuals will therefore tend to be based on one end of the age-related pattern of change only. None of the specific measures used in the age assessment methods are discussed here with the exception of mean canal diameter, but it is predicted that since the overall processes of remodelling show a decrease/increase pattern, many of the measures of structures may also do the same in relation to age.

The mean osteon canal diameter used by Samson (1983), is discussed to some extent as a part of bone turnover rates. The formation of the complete osteon takes longer in old individuals than young, so it might be supposed that the mean canal diameter will be found to increase with age. This would only be the case if the number of newly forming foci were constant. This is seen not to be the case, a reduction in formation occurring with age to the mid-thirties, followed by a slight increase whereafter it remains constant. The proportion of osteons of almost full mineralization is high in all adult ages. It might equally well be expected, therefore, that the mean canal diameter will decrease from adolescence/young adulthood to old adulthood. Here again the predominantly old sample of Samson (1983) may give one trend, whereas over the course of a lifetime a number of different trends would be found.

Diseases of various kinds are found to affect the rate of bone turnover and may therefore distort age estimations

derived from structural measures.

No differences were found between points taken around the femoral cross-section, suggesting that it is of little importance which part of the section is used, as Samson (pers. comm.) also claims. However, it is important which of the two surfaces is used. Marked differences are found in activity at the two, resorption being particularly marked endosteally. All of the methods to be examined take the measures exclusively from the periosteal margin, thereby avoiding to some extent extreme fluctuations caused by increased resorption in cases of disease, malnutrition or osteoporosis.

None of the methods to be examined here make use of measures of mineralization, which are widely used by the medical researchers. As this involves microradiography, this would complicate the procedure and increase expenses so are presumably avoided for these reasons. Finally, none of the studies of bone turnover record any sex differences. It is not evident whether this results from there being none or from no consideration of the issue. The researchers discussed here are very thorough on most aspects and it is more likely that sex differences are not mentioned because none are evident. The use of the same methods across the sexes may therefore be justified.

2.4 External Remodelling.

The effects of external remodelling in relation to age can be seen in changes with age in the overall shape of bones. Arnold et al. (1966) described changes in the vertebral trabeculae which became thinner with age. Atkinson (1969) stated that the commencement of resorption of the transverse trabecular network occurs before growth is ended once the vertical struts of the body are formed. The vertical struts only start to be resorbed after 50. Acsadi and Nemeskeri (1970) similarly examine the heads of the femur and humerus and describe the thinning out of the trabeculae with age. Atkinson (1969) decribes an "axial" shift in the femur alignment as a result of age-related remodelling. At the head of the femur bone is resorbed medially and formed laterally, whereas at the distal end, the opposite is the case with lateral resorption and medial formation. Thus the head end shifts laterally while the distal end shifts medially. The young femur shaft is vertical but this axial shift moves it to an oblique angle in the elderly. The femur also undergoes a rotational shift leaving the increasingly porous areas in a spiral pattern through the bone. In the mandible bone formation on the outer surfaces enlarges the basal part while the upper portion undergoes resorption to remould the alveolar ridge. The resorption occurs first around the teeth and later extends to a large part of the buccal surface while the basal portion remains dense producing a marked contrast in the distribution of porosity in the mandible.

Studies of bone density from ashing and/or weighing the bone give results comparable to those of density studies by microradiography. Arnold et al. (1966) find an increase in the volume of ash in both sexes up to 20 followed by a decrease in the vertabrae. The rib showed a higher density at all ages than the vertebrae but the content decrease after early adulthood in the same manner as the vertebrae. In each individual, however, differences in ratio of vertebral:rib ash were observed, those with osteoporotic vertebrae did not necessarily also have high rib loss. The lowest ratios, i.e. the highest vertebral mineralization was noted in robust and muscular males who engaged in much physical activity. Overall no sex differences were noted. Thompson (1978) from a study of 19 parameters of external and internal bone structure finds core density measured as weight/volume the least correlated parameter with age. A significant decrease is observed in females of density and of bone mineral content measured by ^{125}I photonabsorptiometry. These two were observed as a non-significant decline in males (Thompson, 1980). Whites were compared with four groups of Eskimo data for those two parameters and no significant intragroup sex differences were found amongst the Eskimo and no significant differences amongst any of the groups. Dequeker (1972) reports no difference with age of the ash weight of bone, nor any difference in the Ca/P ratio with age, although there was an inverse correlation of calcium and phosphorous levels with porosity. Trotter et al. (1960) studied density from the weight/volume ratio in the

vertebrae and sacrum, the humerus, femur, tibia and ulna in 80 skeletons of 40 white and 40 black Americans with 20 in each sex-race group. The sample covered a range of ages. The four sex-race groups all showed a decrease with age in density with no differences in rate from one another, suggesting a parallel and uniform rate of loss. The blacks had denser bone overall than whites; the long limb bones were denser having more cortical bone than the vertebrae. The bones with the highest densities were the radius, ulna and tibia showing no significant differences from each other. Next came a group of femur, humerus and ribs. These first six bones do not differ significantly in mean density to each other in the females, but the second group differs significantly in males. In the spine, the cervical vertebrae are more dense than the rest of the spine and this is significant in males. It might have been predicted to find an increase in density from the cervical downwards of weight-bearing was an important determinant of density. That this is not so suggests density is more related to structure than to function.

A vast literature exists on changes in overall cortical thickness with age, representing as it does the most easily measurable parameter.

A great deal of work in this area has been done by Garn and his team. They have collected data from a large number of populations and analysed the results in considerable detail. An overall pattern of bone loss with

age and sex emerges from such studies and recurs in the literature repeatedly (Dequeker, 1972, 1975; Jowsey et al., 1965; Virtama and Helelä, 1969) regardless of population or socioeconomic status (Garn, 1973). At all ages males have thicker cortices than females. At birth cortical thickness is increasing, but at some point during the first year undergoes a period of bone loss. By the first year, however, this is reversed and cortical thickness increases again. The period of infantile loss may last 3 - 6 months. It is due to a slowing in the rate of both periosteal gain and endosteal loss, with periosteal gain slowing faster than the endosteal activity. From age one through to adolescence there is a steady increase in cortical thickness, this increase being greater in boys than in girls. At adolescence a growth spurt in cortical thickness is detected in both sexes, but unlike most growth parameters which have larger and longer adolescent spurts in the male, the cortical thickness growth spurt is more marked in the female, where both periosteal and endosteal apposition occurs. The increase in rate of gain in males is not as obvious but probably carries on over a longer period . The female therefore has a growth spurt starting earlier than males and more pronounced, whereas males have a longer cumulative gain of cortical bone, so that the cortical thickness in males is always larger and by the thirties, considerably larger. Gain in cortical thickness continues in early adulthood in both sexes until the thirties. At this time the rate of endosteal resorption overtakes that of periosteal apposition and loss of cortical bone starts

to occur. The decrease appears to begin quite slowly in both sexes and builds up to a maximum rate of loss in the fifties, declining after that. There is a sex difference in the cortical loss, females losing both relatively and absolutely more bone. The sex difference in rate of loss is at its greatest in the forties (Garn, 1970).

Garn measured cortical thickness as total diameter minus the medullary cavity diameter on the 2nd metacarpal. This allowed him to further analyse the data for specific changes at the two surfaces, periosteal and endosteal. Growth at the periosteal surface shows a similar pattern in childhood to axial growth. There is a period of rapid growth in the first six months postnatally, with a steady increase thereafter until the adolescent growth spurt which occurs at 10 - 12 in girls up until 14 and in males at 14 lasting up to 18. The male growth spurt is therefore about two years longer than that of the females. The gain in males is also larger at this time and increases the degree of sexual dimorphism. After adolescence, periosteal gain continues linearly at a slow rate in both sexes. Although the gain relative to the amount already present is greater in females than males over this post-adolescence phase, the male cortex is always absolutely larger in size. The sex differences in the periosteal diameter is 4% larger in males than females between 1 - 12 years of age, which denotes a wider degree of sexual dimorphism than in stature at this age. In adolescence, the difference rises to 15%, almost twice the sexual dimorphism for long

bone lengths. From 20 - 70, the diameter values are 19% higher in males, again still a large difference compared to stature. The periosteal surface therefore follows childhood growth patterns and has a continuing slight increase throughout adulthood.

The endosteal surface is more varied in its activity. Similar to periosteal growth in childhood, the medullary cavity size increases rapidly in the first year in both sexes, then slows to a steady rate in the juvenile phase. This continues so for males well into the late teens, whereas the females show an earlier change. The steady increase in medullary cavity size halts at the adolescent period and reverses as endosteal apposition occurs. Although this occurs about four years earlier in females (14), than males (18), it is still quite late compared to other growth spurts (10 - 12 in females). A further sex difference is the amount of endosteal apposition which is far greater in females than males. This apposition can continue through to age 30. So during adolescence males are gaining at the periosteal surface whereas females gain at the endosteal surface. By 40 apposition halts at the endosteal surface and resorption commences. The first ten years see only slight bone loss. The resorption peaks at 45 - 55 and then continues, but at a slower rate. The amount of bone loss is both absolutely and relatively far greater in females (Garn, 1970).

This pattern of overall gain in cortical thickness and later loss caused by the balance of activities at the two surfaces may hold for all circumstances but variations exist in the amounts of bone involved, the exact median ages of onset and the extent of the sex differences. Examination of some of these variations can give clues to the mechanisms controlling the bone gains and losses. Interest is focussed on these mechanisms by the clinical state of osteoporosis, seemingly an extreme form of bone loss which it is hoped to reverse or slow by understanding the mechanisms involved in both normal and abnormal bone loss. Allbright first suggested that osteoporosis was merely an extreme result of the normal processes, defining the state as too little bone compared to the average for any age, but what there is, is normal. Analyses of bone composition in osteoporosis seem to support the definition (Dequeker, 1975).

Factors thought to be important in the control of bone gain/loss cover a wide range, and suggest that the amount of bone is the end product of numerous factors acting along many different pathways.

Mechanical factors are thought to play some role, although the evidence is not very strong. Study of which bones are affected particularly by bone dynamics of growth find the pattern affects all bones, cancellous, cortical, weight-bearing and non-weight-bearing (Garn, 1970). Indeed, most studies on living samples use the metacarpal as this

is easily X-rayed, has a simple tubular structure, shows the changes well, but is not a weight-bearing bone. However, bone loss is certainly increased in inactivity states. Whether a corresponding increase in cortical thickness is found in over-activity states is as yet unknown, but thought to be a possibility (Garn, 1970). An interesting observation is made as regards bone loss in relation to stature. A negative correlation is reported for bone loss between 45 - 65 with stature in the metacarpal bone (Garn and Hull, 1966). The researchers suggest the explanation may lie in the dynamics of growth and later resorption, in that taller individuals may represent the result of a longer growth phase, or having different targets for the growth activity. If this is so then equally well they could have different targets or time schedules for post-adolescent 'growth' activities. Bone loss with age obviously affects the strength of the bone and its ability to resist demands upon it as evidenced by the increase in fracture rates with age (Dequeker, 1975; Garn, 1973). As expected females show a far greater likelihood of fracturing with age. By age 90 the cumulative risk of fracture in a cervical spine or femur neck has risen to 23% in females and only 9% in males. The fracture rate is 4 - 10 times higher in females (Garn, 1973). The most commonly fractured bones are the distal forearm, the vertebral bodies, the humerus neck and the femur neck, are all areas where the bone is predominantly cancellous and therefore less dense to begin with (Dequeker, 1975).

Genetic differences are shown by population differences where other factors, such as nutritional states, are not noticeably different. A study of the Chinese and Japanese compared to an American series showed the Asians as having less compact bone at all ages. American-born children of far eastern ancestry showed this as well as the native cases, and children of mixed marriages had intermediate measurements. There was no evidence of nutritional differences and the intermediate measures of the mixed ancestry group added support to a genetic explanation (Garn, 1964). In a study of femur neck fractures, a far lower incidence amongst negro Americans was observed than was predicted from the proportions of negro/white hospital admissions (Garn, 1973). A similar study in Singapore found femur neck fractures to be more common amongst the Indian community and lowest amongst the Malays. The Chinese appeared to be intermediate. A study of African tribesmen of the Bantu observed a lower femur rate compared to Europeans (Garn, 1973). A different study on the Bantu recognizes the low fracture rate compared to Europeans and also measures cortical thickness. Children have lower levels of cortical thickness than European children of equivalent ages, but have a longer growth phase which counteracts this effect. Adults are found to have no differences in cortical thickness to Europeans. The apparent additional strength of the bone may therefore be due to density rather than absolute thickness (Walker et al., 1970). However, Dequeker (1972) found negroes lost less bone than whites with age.

Studies of familial patterns in growth generally, show the best correlations are between sisters, implying a genetic involvement by the X chromosome. A greater intra-girl correlation was also found in growth measures compared to intra-boy correlations (Garn and Rohmann, 1966). As regards cortical thickness specifically, again sister-sister correlations are the strongest family relationships found (a correlation coefficient of 0·37). Monozygotic twins show considerable similarities, greater than dizygotic (Garn, 1970; Amtmann, 1971). Some genetic disorders affect the amount of cortex, as in medullary stenosis where the medullary cavity is decreased in size, or some forms of osteogenesis imperfecta where the cavity is increased in size (Garn, 1970). Chromosomal abnormalities also display reduced cortical thickness, particularly in trisomy G (Down's syndrome), XO, XXY and XXXY variations, the reduction being greater than can be explained by overall size reduction of the skeleton (Garn, 1970).

Cortical thickness is affected in a number of the clinical complaints and a brief summary is given by Garn (1970). Thinner cortex is found in blood dysplasias, protein-calorie malnutrition, hyperparathyroidism, renal tubular acidosis, kidney dialysis after gastrectomy, in malabsorption states and inactivity particularly long-term juvenile inactivity such as hydrocephaly or spherocytosis. Decreases in cortical thickness are recorded at high altitudes from hypogravic effects, which appear to affect activity at both surfaces in children,

but mainly the periosteal surface in adults. Reductions of cortical thickness brought about in severe nutritional conditions can be relieved to some extent by the administration of the deficient product. For example, loss through renal glycogenesis can be staunched by glucose feeding. The effects of vitamin D therapy on cortical size in D-deficiency or vitamin D-deficiency rickets is unknown and awaits study.

An increase in cortical thickness is found in adrenogenital syndrome, sexual precocities of any kind, Holt-Oram syndrome and Paget' disease (Garn, 1970).

The two main theories of mechanisms of bone loss concern nutritional agents and hormonal factors. The two, of course, are by no means exclusive of one another, but much controversy rages over the relative importance of each.

A large number of nutritional/metabolic disorders can cause a decrease in cortical thickness as demonstrated by the list of disorders given by Garn (1970). The hypothesis has been made that alterations in absorption efficiency concomitant with age and/or cumulative effects of reduced intake, may be responsible for both the normal age-related bone loss and the exaggerated loss characteristic of osteoporosis.

Most attention has been focussed on calcium levels, Ca/p ratios and protein or calorie intakes. General studies

of growth in response to marked nutritional stresses suggest that the system is capable of responding appropriately to short periods of scarcity, such as dry seasons, by slowing growth with a subsequent pick-up period such that no permanent damage is caused, except possibly some reduction in stature, particularly in males thus reducing sexual dimorphism. However, stature reductions are difficult to assess relative to a norm in a population where the established norms based on Americans are not relevant.

Garn (1973) found that although a moderately low protein diet could lead to a smaller skeleton size, the proportion of bone loss with age is still the same as individuals on a high protein diet. In severe protein-calorie malnutrition, by contrast, permanent retardation of development, physical and psychological is caused. Both sexes are equally affected by retardation in this case, the stress going beyond the capacity of the system to adjust to it. The periosteal apposition of childhood is retarded or even halted and in addition resorption occurs or is accelerated. Although marked cortex reduction occurs in all of the protein-calorie malnutrition complexes, the most severe effects are found in the marasmic forms, due to a lack of both protein and calorie intake compared to kwashiorkor where protein is deficient but calorie intake may be adequate. Some children with marasmus or mixed marasmus-kwashiorkor states may have no more bone at 4 to 6 years than would be expected in a one year old (Garn, 1964,

1970). Continued resorption occurs even after treatment, during the so-called recovery period (Garn, 1970) implying a time lag in the responses and mechanisms that control resorption.

Hypotheses concerning calcium intake postulate that a cumulative inadequate intake of calcium would lead to bone loss through both inadequate mineral supply for bone formation and compensatory resorptive action to provide the necessary minerals for other parts of the system. It has been possible to induce bone loss in rabbits fed low calcium diets but the evidence in humans does not so far support the theory. There is no evidence that groups having low calcium intake below 400 - 300 mg/day show any significant bone loss, nor that groups consuming in excess of 1,300 - 1,500 mg/day have protection against bone loss in the later decades of life. This was illustrated by comparison of a Guatemalan sample with samples from Panama and the Caribbean. The Guatemalans used yellow maize, stone-ground meal and the preparation of food by soaking in lime-water, all of which give a high calcium intake by comparison to the Panamanian and Caribbean groups which had a very low intake, as low as 200 mg/day. No differences were observed between the groups' patterns of age-related bone loss. Similar results have been found for the phosphorus intake and the Ca/P ratio. Cereal based subsistences tend to have a high level of phosphorus in the diet relative to calcium, whereas dairy pastoralists have a greater level of calcium relative to phosphorus and again no differences are recorded in bone

losses (Garn, 1973). Other evidence, possibly suggestive against the intake theory, although not directly related, is that the composition of the bone itself does not change with either normal aging or osteoporosis as regards the calcium and phosphorus content (Dequeker, 1975). Treatments of increased intakes of calcium have not proved to be useful in themselves against bone loss, but only indirectly as possibly stimulating a hormonal reaction (Dequeker, 1975). Patients who suffer from lactase deficiency and thus avoid dairy products, often seem to suffer from osteoporosis, and yet negro populations rarely show signs of osteoporosis whereas lactase deficiency is common (Dequeker, 1975).

Intake of trace elements has been noted as possibly significant. High intakes of magnesium observed in Iranian populations neither protect nor exacerbate bone loss. However, certain groups along the Indian Ocean and around Lake Rudolf have been found who possibly have no adult bone loss, or very little. All of these groups are associated with high levels of dietary fluoride. Fluoride appears to reduce bone loss by reducing solubility and increasing amounts of bone by altering the size and nature of the bone crystals (Dequeker, 1975; Garn, 1973).

Rather than examining simply intake levels, particularly of the critical elements of bone, equal attention should be given to absorption, excretion and rates of resorption from the bone (Jowsey et al., 1965). It has already been shown above that severe malnutrition not only

halts formation but causes resorption of bone elements. Lactation is a common candidate for promoting accelerated resorption of calcium. In fact the net result of pregnancy followed by lactation is a normal rate of bone loss, as bone is formed endosteally during pregnancy (Garn, 1970). Urinary levels of calcium, phosphorus and total hydroxyproline excreted show a negative correlation with age in both sexes. This occurs without any associated reduction of calcium or phosphorus intake and thus implies a reduction in intestinal absorption of the elements. As hydroxyproline levels are probably determined by the skeletal collagen, the reduction reflects the reduction of skeletal mass with age rather than being directly involved in producing it. The diurnal pattern of mineral excretion shows a high day : night ratio in young adults, a relationship which is reversed in older age groups. The daytime reduction is probably related to a fall in absorption, possibly to reduced intake and/or to reduced renal excretion (Dequeker, 1975). Secondary hypercalciurea can be provoked by dietary factors such as increased salt intake or carbohydrate. A possible pathway is proposed by Dequeker (1975) involving calcium deficiency in the body provoked by such dietary factors causing a decrease in the serum calcium level, increased parathyroid activity and increased bone resorption. The example is given, in support of the network, of negroes who have a low level of osteoporosis and a low salt intake (Dequeker, 1975).

Levels of calcium, phosphorus and alkaline phosphatase in the blood are also examined by Dequeker (1975). The serum content of calcium and alkaline phosphatase shows a lower level in females relative to males under fifty and the reverse after fifty. It is possible that the serum calcium level may be related to the serum protein level. The sex difference and age-related reversal for serum protein remains unexplained, as does the serum alkaline phosphatase level, although alkaline phosphatase activity is thought to reflect osteoblastic activity in some unknown manner. It may also be related to size in young adults, although there is no supporting evidence, or to physical activity. The age associated increases in the two substances, most marked in the female, has also been attributed to age-related changes in liver function (Dequeker, 1975). However, the patterns of growth in childhood, the sex differences of adolescent growth, the difference in adult loss between the sexes and the above mentioned differences in levels of minerals and compounds are suggestive of hormone-induced changes.

The post-menopausal theory has long been popular to explain the more dramatic loss in females in later life. The sister-sister sibling correlation has suggested an X-linked factor, and the apposition patterns of growth in adolescence and early adulthood points to hormone-specific responses at the two surfaces. The periosteal surface experiences adolescent growth disproportionately in males to females while at the endosteal surface the reverse is true.

Garn (1970) speculates that the periosteal surface is controlled specifically by testosterone and the endosteal by oestrogen. Another indication of the role of oestrogen in promoting bone apposition endosteally is observation of this activity in pregnancy, increasing cortical thickness. Lactation would normally reduce cortical thickness to its normal age-specific level but pregnancy without prolonged lactation can lead to permanent gain in cortical thickness. Females in their sixties who have had a number of live births without extensive lactation show greater cortical thickness than others of the same age (Garn, 1970).

The hypothesis, therefore, is that the greater bone loss in females is a post-menopausal feature brought about by a reduction in oestrogen levels which appear to inhibit resorption and even stimulate apposition endosteally. Testosterone decrease in males merely slows formation periosteally rather than allowing increased resorption. Garn (1970) compares women who had undergone ovariectomy early in life (34+) with those experiencing natural menopause at the average age around the fifties, and found the former group to suffer from greater and faster bone loss. Increases of serum levels of calcium and alkaline phosphatase, of serum phosphatase and urinary excretion of calcium and total hydroxyproline can be reduced by oestrogen treatment (Dequeker, 1975). At the same time, bone resorption was slowed. Oestrogen therapy is a common treatment for individuals suffering from

osteoporosis.

Determination of the exact age of onset becomes critical in assessing the hypothesis. Most researchers on modern samples record the age of onset of bone loss as post-menopausal, giving rise to the theory in the first place. However, Garn (1970) with a large sample size, was able to detect loss of bone earlier than the menopausal age, although the loss was only slight at first. In the next section, archaeological studies of cortical thickness are reviewed and it is seen that bone loss begins as early as the mid-twenties in females of some early groups. This may be an artefact of problems in determining age at death, but the authors themselves regard the finding as evidence against the post-menopausal case (Dewey et al., 1969a, 1969b).

It is concluded that the post-menopausal reductions in oestrogen are obviously an important factor towards bone loss in females, but do not represent the whole explanation, since loss occurs in both sexes and pre-menopausally. The role of oestrogen is probably part of a far more complex cycle of factors and interrelationships.

Another potentially important hormone is parathyroid hormone. Levels of parathyroid hormone appear related to serum calcium levels and oestrogen has been demonstrated to have an inhibitory effect on parathyroid activity. It is proposed that parathyroid hormone therefore may be directly responsible for stimulating bone resorption. Dequeker (1975)

emphasises the role of parathyroid hormone, incorporating dietary factors into the cycle as explained earlier. Jowsey et al. (1965) also stress the importance of parathyroid hormone in bone loss from internal remodelling.

Finally, calcitonin is involved in bone turnover and can reduce resorption in young adults and in the extreme case of Paget's disease. However, treatment with calcitonin of post-menopausal osteoporotic women did not halt bone loss and even worsened it in some cases. Probably the effect of hypocalcaemia, produced by calcitonin activity, promoted parathyroid hormone secretion and subsequent bone resorption (Dequeker, 1975). This kind of close interrelationship of hormones, mineral levels and bone cell activity illustrates the difficulties of identifying roles or even the inappropriateness of such an approach. Even greater are the problems of treatment to arrest excessive bone loss since formation and resorption act as a unit together and form part of a complex system where any interference with one factor will have repurcussions in the others, e.g. slowing down resorption rates will invariably lead to a slowing down of formation rate also (Dequeker, 1975).

2.5 Archaeological Studies of Cortical Thickness.

There have been two main groups of archaeological populations studied for age-related changes in cortical thickness:- First, the Nubian populations on which extensive skeletal analysis has been performed by Armelagos

and his colleagues; secondly, a number of American collections, namely the Terry Collection (Bricksen, 1982) the University of Utah, Great Basin Skeletal Collection (Van Gerven, 1973) and the Campbell Site, Prescott Co. Missouri (Carlson et al.1976).

The Nubian material consists of three groupings: Meroitic, X-group and Christian. Cortical thickness has been studied by direct measurement from cross-sections. Six measurements were taken at equally spaced points around the section and the mean cortical thickness calculated. The three populations do not exhibit any significant differences in genetic traits of the dentition, nor in the age-related loss of cortical thickness, so the three populations can be treated as one group for the purpose of analysis. This gives a larger sample size of 83 males and 120 females. The sample is classed into four age categories : 16 - 21, 22 - 31, 32 - 41, 42 - 50+. There is marked difference in the age-related changes of the two sexes. The loss of cortical bone in males with age is not significant at $p < 0{\cdot}05$ whereas the loss for females is significant at $p < 0{\cdot}01$. Males do not lose bone until the last age category, that is over forty, whereas the loss of bone in females begins from twenty-two onwards. The total absolute loss of 0·124 cm of cortex in females is three times the male loss of 0·042 cm. The overall male loss represents 13·75% of the bone and the female loss is 23·39%. The greatest loss in females is found in the thirties, comparable to modern populations. The overall loss of bone in a modern female

population is given as 40% and the authors suggest that the slighter loss in the nubian females is evidence of a shorter life expectancy (Armelagos et al., 1972; Dewey et al., 1969a, 1969b).

Further analysis is undertaken by examining the effect of normalization on the cortical thickness measurement. It is recognized that a relationship exists between femur length and robusticity, therefore probably with cortical thickness, and that a great range of femur length will be found in a single population. It is considered therefore that the size of the individual as expressed by the femur length should be taken into account in order both to compare populations of different statures and to compare age groups within a population to better effect. The mean cortical thickness measurement is divided by the femur length and multiplied by a 100, thus expressing the cortical thickness as a % of the femur length. A comparison of results using normalized and non-normalized data shows a number of relationships are heightened to statistical significance with the measurements normalized. Particularly important was the result that the normalized cortical thickness loss with age was significantly different between the three nubian groups, whereas the non-normalized was not. This suggests that the variation of femur length between the populations is greater than the cortical thickness variation (Dewey et al., 1969a).

The main interest of the analyses was to compare archaeological samples with modern and to try to shed light on the etiology of bone loss. The results were comparable to modern samples except that bone loss appeared to begin far earlier in the nubian females. It is possible that this could be an artefact of incorrect age assessment. However, assuming this is not the case, a gradual progressive decrease of bone with an early age of onset would not be consistent with the post-menopausal hypothesis. The authors feel the bone loss is more likely to reflect insufficient calcium intake coupled with extensive lactation which accentuates the effect of low calcium levels by drawing on the skeleton's store of calcium for milk production (Armelagos et al., 1972; Dewey et al., 1969a, 1969b).

Sections were taken from the femora of 23 females and 20 males from the late Mississippian site of Campbell, Prescott Co., Missouri, for a similar examination of archaeological bone loss by Van Gerven and Armelagos (1970). The same six measurements around the section were made plus an additional one on the linea aspera. Again, the adults were placed in age categories of 22 - 31, 32 - 41, 42 - 50+. The younger 16 - 21 category of the nubian analysis was not included. Similar to the Nubian results, an observed overall loss of 11·3% of bone amongst males was not significant at $p < 0{\cdot}05$, whereas the larger loss in females of 29·9% of bone was significant. The values of cortical thickness were not normalized. The differences in the patterns of cortical thickness loss between the sexes is

significant at $p<0.01$. Loss of bone was thought to begin in the 22 - 31 age group in females, but without the younger category for comparison this could only be a non-metric observation of bone porosity. The greatest loss of bone occurred in the oldest age category in both sexes. The results were comparable to the nubian ones, again showing a lower overall loss than modern samples, suggestive of fewer elderly survivors (Van Gerven and Armelagos, 1970).

An extension of these studies was made by Van Gerven (1973) to include the tibia and humerus and to measure area as well as cortical thickness. The sample comes from the University of Utah, Great Basin Skeleton Collection and numbered 23 females and 20 males. Age could not be assessed beyond fifty; the youngest individual included was aged at nineteen. In males, the tibia showed the most steady loss of bone with age, although none were significant. In area, the tibia did show a significant decrease at $p<0.05$. Amongst females all three bones showed significant loss with age of both thickness and area at $p<0.01$. In both sexes, the humerus undergoes the least amount of bone loss, in males the tibia loses most and in the females the femur loses most. The corresponding measures of cortical thickness and cortical area were correlated and found significant at $p<0.05$ in all cases. Similarly, comparison of loss in the different bones show a significant correlation between them all with $p<0.05$. This indicates a certain uniformity in the long bones in cortical loss, further supported by a stepwise regression analysis. This

demonstrated that sampling of multiple sites provided very little further information than one site alone (Van Gerven, 1973).

Carlson et al. (1976) investigated cortical thickness, cortical area and diaphyseal diameter in the femur at different points on the diaphysis from 21 females and 19 males from the Campbell Site and were grouped by age as 20 - 40 or 41 - 55+, then further divided into 20 - 30, 31 - 40, 41 - 55+ for detailed analysis. Five cross-sections were made along the length of the shaft and eight measurements around each section were taken to get the mean cortical thickness. Diaphyseal diameter was measured as the mean of the anterior-posterior and medial-lateral measures of diaphysis width. The two sexes showed different patterns of loss along the diaphysis from one another. In females the greatest reduction of cortical thickness with age occurred at the second cross-section (proximally), whereas this had the least decrease in males, the greatest loss being distally at the fifth cross-section. The loss of cortical thickness is not accompanied by a correspondingly high loss of cortical area. In females, the second section loses the least amount of area and the fifth section in males only loses a moderate amount although more than elsewhere in the shaft. The fifth section is also the site of greatest area loss in females. Increases with age of the diameter were not significant among females although there was a trend towards an increase. Males did show significant increases in diameter distally at the fourth and fifth

sections. This is interesting since, in spite of this periosteal gain, loss of cortical thickness and cortical area are greatest at this point indicating a dynamic site of remodelling activity. Overall, therefore, females become thin in bone proximally and males distally. The explanation for these sex differences is not evident, but the results add to the growing body of evidence for different bone dynamics between the sexes from whatever causes.

Finally, Ericksen (1982) has utilized the Terry Collection of known age, sex and ethnicity to follow up similar studies with a larger sample. 458 femora were used and radiographed from the posterior-anterior angle and from the medial-lateral view at eleven levels spaced at distances of ¼ of the femur head diameter. The first of these was drawn through the lower border of the greater trochanter. Only six of the levels were reviewed in the article published. The ages of the sample ranged from 20 - 98. The levels between the two trochanters showed less change than those subtrochanteric, interpreted as evidence that remodelling activity in the higher levels occurs in cancellous bone rather than the cortical bone. In the lower levels, loss was observed to occur first in the anterior-posterior quadrants. In males, no loss at all was recorded in the medial or lateral quadrants. Females did show considerable thinning but not until their fifties. Ericksen suggests bone loss may be retarded in the medial and lateral quadrants through some mechanical stress-related maintenance factor (Ericksen, 1982). The negro

Americans in the collection lose bone earlier than the whites, although elsewhere in the literature it is stated that African negroes show less bone loss with age and rarely suffer from osteoporosis (discussed earlier). The later onset of bone loss amongst the modern Americans compared to the archaeological samples leads Ericksen to postulate that cortical loss in the femur may have been accelerated in the past, relative to modern samples (Ericksen, 1982), but does not discuss what possible mechanism this may affect.

2.6 External Remodelling for Aging Skeletons.

Age-related changes in the gross morphology of size may prove useful for aging purposes. To date, the only method using this scale of observation is the assessment of trabeculae distributions in the proximal epiphyses of the humerus and femur by Acsadi and Nemeskeri (1970). Density was measured by Thompson (1978) but does not feature amongst the five best parameters for age assessment in the femur with the sexes together.

Cortical thickness is seen to change predictably with age, but, similarly to the internal level of bone activity, cortical thickness does not alter in a linear fashion with age. First, it shows an increase up to the mid-thirties and only in the forties does a decrease in thickness occur. Thompson (1978) makes use of cortical thickness as the second of his five parameters. Since his sample was heavily biassed towards older individuals it is feared that the

inclusion of cortical thickness is an artefact of his sample. Thompson (1978) does, however, say his aim is to devise a method for aging the older individuals in the population. It may be that these have to be identified first by other criteria before Thompson's methods can be used.

Samson explored the potential of using cortical thickness as a parameter but eventually abandoned it as not reliable (pers. comm.).

Overall, sex differences exist in cortical thickness at all ages in both absolute size and in rates of gain and loss. Normalization procedures could deal with the absolute size differences possibly, but the rates of change have to be recognized. Use of the cortical thickness measure should involve separate methods of conversion for the sexes. Thompson (1978) does provide separate equations, but the ones where sexes are together do not show a very much greater level of resolution. This seems to be surprising since cortical thickness is a parameter in all but one of the equations.

Overall, from the literature review on age-related changes in cortical bone structure, there is no reason to expect measures of either internal or external parameters to increase linearly with age, as neither the turnover of bone internally nor the external growth and loss pattern alters uniformly in one direction with age. It is predicted, therefore, that only low correlations will be

found between age estimates from bone structure and other estimates. Analysis of each measure by dental age may help evaluate the possible usefulness of bone structure measures.

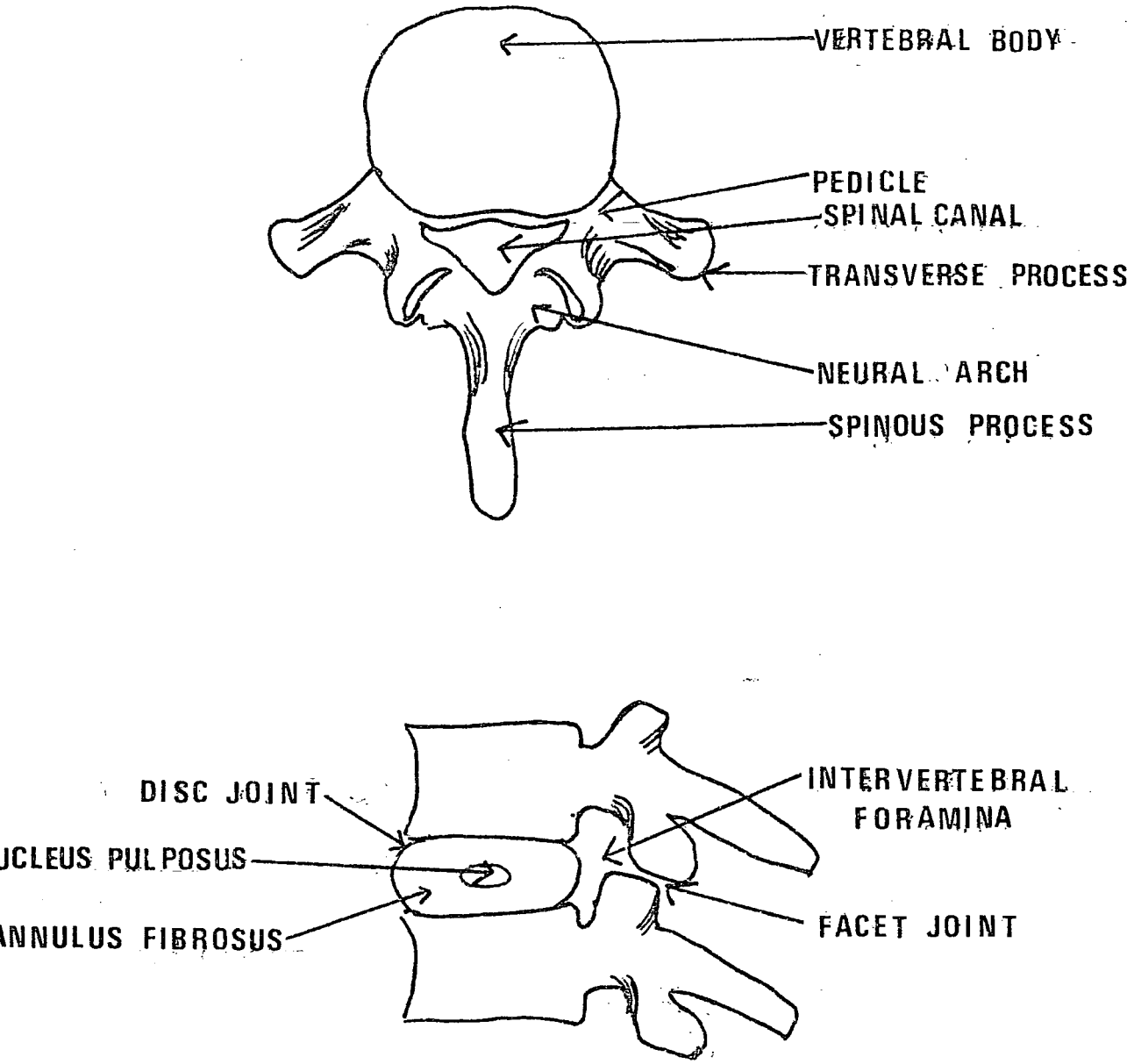
Chapter 3. ANATOMY OF THE VERTEBRAL COLUMN AND DEGENERATIVE CHANGES.

In addition to the examination of age-related changes in bone structure of the femur, a study will be made of the incidence and severity of degenerative joint disease in the spine. This chapter will describe the anatomy of the spine, how to identify the osteological elements and the changes of the tissues in degenerative joint disease.

3.1.1 Anatomy: Vertebrae.

The human vertebral column is composed of twenty-four bone elements called vertebrae which alternate with the cartilaginous intervertebral discs. The sacrum has five fused vertebrae and the coccyx four or five vestigial fused elements. The vertebrae are classified by region into three groups; the cervical region, numbering seven vertebrae, the thoracic region with twelve vertebrae and the lumbar region of five vertebrae. All the vertebrae have the same basic design of a cylindrical body joined by a bridge or pedicle on each side to the neural arch which together enclose the spinal canal through which the spinal cord runs. From the back of the neural arch projects a spinous process, and from each side there projects a transverse process (see Fig. 3.1).

FIG. 3·1 VERTEBRAL ANATOMY



On each side of the vertebra by the projections of the transverse processes are two pairs of articular facets superiorly and inferiorly placed, which form a joint with the corresponding facets of the adjacent vertebrae. The intervertebral disc lies between the bodies of adjacent vertebrae forming another joint. The facet joints (zygapophyseal joints), pedicles and disc joint enclose a space called the intervertebral foramen through which pass the segmental nerves (see Fig. 3.1).

The vertebral bodies are linked by two longitudinal ligaments running anteriorly and posteriorly from the sacrum to the basi-occiput. Both are firmly attached to the intervertebral disc and adjacent bone margins, but less so to the rest of the vertebral body.

The total number of presacral vertebrae although usually twenty-four, can show variation relatively commonly on either side of this norm. There is clear indication of some form of genetic control in this from family studies. In addition, a number of studies comparing different racial groups summarized by Allbrook (1955) and Bornstein and Petersen (1966). Sex differences also play a part with a tendency for males to show an increased number and females a decrease (Bornstein and Petersen, 1966; De Beer Kaufman, 1974, 1977).

The cervical vertebrae seem to be numerically stable (Sager, 1969). Instances of six vertebrae are found once by

Allbrook (1955) from 206 spines, and twice by Ledouble (1912) from 1420. Lanier (1939) reports a case of eight cervical vertebrae. Willis (1929) finds the usual seven in all 1471 of his sample.

An additional thoracic vertebra seems to be fairly common. Allbrook (1955) reports an incidence of 4·6% from an East African sample. A reduction to eleven is rarer; Allbrook (1955) finds only one case, which is associated with six lumbars giving the same overall number of twenty-four.

The lumbar spine is the most variable portion showing an increase of 8·4% in Allbrook's sample. He finds a decrease in 2·4% of cases, which was always associated with an extra sacral segment, most probably representing the fifth lumbar sacralized.

The occurrence of six segments in the sacrum may reflect incorporation of the first coccygeal element, or, as mentioned above, the last lumbar vertebra. In some instances, a real extra sacral segment may occur. Sacralization describes the incorporation of one of the other spinal structures. Lumbarization describes the separation of the first sacral segment from the rest of the structure. Maurice-Williams (1981) reports the occurrence of sacralization as 3% in the present day British population. Lumbarization is found in 1% of the population. Allbrook (1955) finds sacralization in 11% of the East African sample.

This broke down into 6·5% out of 185 spines where the fifth lumbar is sacralized and 66% of fifteen spines with six lumbars showing sacralization of the extra sixth vertebra. Kellgren and Lawrence (1958) in contrast to the other studies found only one case of sacralization in the extensive survey of the Leigh population in South Lancashire.

3.1.2 Anatomy: The Joints.

The joints of the body are classified into three main types: fibrous, cartilaginous or synovial (Gardner, 1972).

In a fibrous joint, also called a synarthrosis, the bone elements are joined by fibrous tissue and very little movement, if any at all, occurs at the joint. Fibrous joints are of two kinds, sutures and syndesmoses. The joint of the tooth in its socket is sometimes included as a third kind, termed a gomphosis.

In cartilaginous joints the bone elements are joined by either hyaline cartilage or fibrocartilage. The joints joined by hyaline cartilage are also called synchondroses. Such joints are characteristic of the immature skeleton where bones are joined to their epiphyses by hyaline cartilage while growth is in progress, but most hyaline cartilage joints are replaced by bone by adulthood. Cartilaginous joints where the bone elements are joined by fibrocartilage are called amphiarthroses. The fibrocartilage is usually separated from the bones by a layer of hyaline cartilage.

The intervertebral discs of the spine are amphiarthrodial joints; the pubic symphysis is another example.

The vertebral bodies are joined together by intervertebral discs numbering twenty-three in all from the second cervical to the first sacral segment, forming a series of amphiarthrodial or slightly movable joints. The discs make up about twenty per cent of the spine's total length in the adult; the size of each of the discs corresponds to the size of the vertebral bodies it joins. The shape of the disc can influence the curvature of the spine. In the cervical spine the discs are wedge-shaped contributing to the presence of the usual cervical lordotic curve. The thoracic discs do not really contribute to the usual kyphotic curve although they are a little more prominent posteriorly than anteriorly. The lumbar discs play a very important role in the lumbar lordotic curve being considerably heavier anteriorly especially the disc of the lumbo-sacral joint.

The disc has three components: the cartilage endplates, the annulus fibrosus and the nucleus pulposus.

The cartilage endplates cover the surfaces of the vertebral body within the bony ring epiphyses, are about 1mm thick made of hyaline cartilage and are thickest centrally, merging with the annulus fibrosus on the periphery. As the disc is avascular in the adult, the cartilage endplates permit fluids and nutritive substances from the blood vessels within the vertebral bodies to perfuse through to

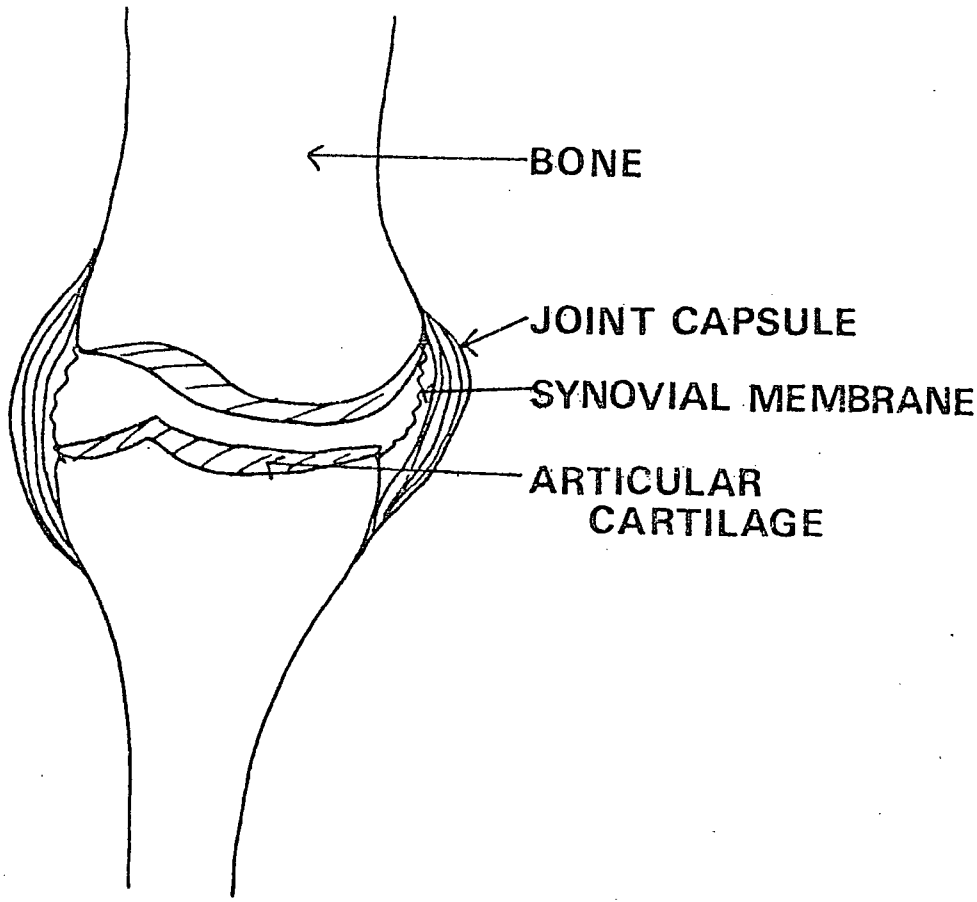
the substances of the disc itself.

The annulus fibrosus is a ring of superimposed concentric layers of collagen fibres, attached to the adjacent bone by Sharpey's fibres. The layers run in at angles to each other and run in a spiral fashion between adjacent vertebral bodies.

This encircles the nucleus pulposus, a gelatinous hydrophilic tissue consisting of randomly arranged collagen fibres and a few elastic fibres in a matrix of water held in protein-polysaccharide complexes. The nucleus pulposus comprises about fifteen percent of the total disc volume and is positioned slightly behind the central part of the disc. This means the annulus fibrosus is thinner posteriorly and therefore weakest at this part.

The majority of the joints of the body, including the facet joints of the spine, are synovial joints or diarthroses. In a synovial joint the bone elements are linked by an envelope of capsular cartilage which encloses the whole joint forming a capsule. The articular surfaces of the bones are covered in a layer of articular cartilage. This in turn is lined on the inside by synovial membrane. The space enclosed by the synovial membrane is called the synovial cavity. The synovial membrane surface elaborates synovial fluid which is a thick viscous liquid. Lastly, ligaments stretch across the joint linking the bone elements and providing strength and stability to the joint (see Fig.3.2).

FIG. 3·2 SYNOVIAL JOINT ANATOMY



The facet joints form the hinges of the spinal column's three-pillar support structure. The synovial joints of the body are designed to resist the forces upon them and still allow the maximum necessary degree of freedom of mobility. The angles at which the facets meet very much influence the kind of mobility possible at that joint. The cervical spine is the most mobile portion of the spine freely enabling flexion, extension and lateral flexion. Some rotation is possible too, particularly at the atlantoaxial joint. Movement in the thoracic spine is limited by the rib cage, although flexion, extension and lateral bending are all possible as well as rotation in the mid-thoracic region. The lumbar region has very limited rotation (about five degrees only). Flexion and extension are the main movements (Maurice-Williams, 1981).

3.1.3 Anatomy: The Articular Cartilage.

The articular cartilage of the joint is the most important structural element in the synovial joint as regards development of degenerative changes in the joint.

The cells of the hyaline articular cartilage are called chondrocytes and are estimated as comprising about five percent of the tissue, although this figure varies enormously with age, being far greater in the growth period.

The inter-cellular substance breaks down into:

collagen 23[illegible]

mucopolysaccharides 20%

non-collagenous protein 23% (Sokoloff, 1969).

Articular cartilage has a very high water content, estimated at around seventy percent of the whole tissue during life. The mucopolysaccharides are 35 - 40% chondroitin sulphate. These rough estimates, made for synovial joints in general, of the proportions of the different components are found to vary enormously between individuals, with age, by anatomical location between and within joints.

The articular cartilage is arranged in a three-layer system, each layer of which demonstrates a different composition and structure. Articular cartilage is seen as a dynamic tissue undergoing constant degeneration, proliferation and remodelling throughout life similar to those remodelling processes in bone tissue (Johnson, 1962).

The outer, superficial layer of articular cartilage is thin with collagen fibres orientated tangentially, forming long transverse arcs. The arrangement is designed to absorb shocks as well as to maintain the integrity of the surface. The cells are small and flattened along a long axis parallel to the joint surfaces. This superficial layer undergoes constant shedding and replacement from the second transitional layer. This is characterised by a high water content with larger, more rounded chondrocytes arranged in columns whose axes are at right angles to the joint surfaces. This layer is a broad zone of hyaline cartilage and is the

principle shock absorbing portion. The third layer or deep layer has large chondrocytes with many of these adult cells often dead surrounded by intercellular substance. Calcification also occurs in this layer. It acts as a barrier to the percolation of materials from the cartilage to the underlying subchondral bone. This is also the part which acts to anchor the cartilage to the bone. The deep layer is where enchondral growth and consequent remodelling of the bone and cartilage is taking place (Jurmain, 1975).

The combination in the intercellular matrix of collagen and ground substance acts as a two-phase material. Collagen is highly resistent to tension but not compression. The ground substance by contrast, as a result of its high water content, is highly incompressible, but has no tensile strength. The mucopolysaccharides add toughness and resilience to the tissue and the ratio of these to collagen varies by joint and joint areas, showing a higher proportion in joints and joint areas subject to greatest pressures.

The overall thickness of the articular cartilage varies between individuals, from joint to joint, surface to surface, again mainly in response to functional demands. It has been demonstrated (Säaf, 1950) that vigorous exercise can increase the thickness of the articular cartilage by 25 - 35%. Jurmain (1975) lists as factors influencing cartilage thickness:

1. greater in larger joints than smaller
2. greater in joints or parts of joints subject to

functional pressure (especially true of joints of the lower limb)

3. increase with friction, scissor action of cartilage and lateral displacement.
4. less thick in smoothly fitting joints.
5. thickness tends to decrease with non-use.

There is an upper limit on the thickness of the articular cartilage set by the means by which cartilage is nourished. Articular cartilage has no nerve or blood supply therefore the acquisition of the nutrients from the synovia, the vascular ring of the synovial membrane and the blood vessels of the subchondral bone. The process of diffusion therefore sets a limit of the potential dimensions of the cartilage.

Very little is understood of the metabolism of articular cartilage as yet. Studies are performed by measuring rates of uptake of various labelled isotopes by the cells and subsequent appearance in intercellular substances. Collagen in connective tissue, as is true for collagen elsewhere in the body, is a static material, relatively inert metabolically showing very little turnover or degradation or synthesis once net synthesis has ceased. It does appear to have an active capacity for repair and replacement, however, as evidenced by repair in wounded tissue. The inert collagen probably provides a stabilizing influence in the chemical structure of the connective tissues.

By contrast, the mucopolysaccharides have very rapid

turnover rates e.g. half life measurements for hyaluronic acid and chondroitin suphate have been estimated at respectively 2 - 4 days and 7 - 10 days.

The rate of synthesis of ground substance exceeds that necessary purely in compensating for attritional loss, indeed a cathepsin has been identified, thought to be specific for the degradation of the protein part of protein-polysaccharide complexes. This upholds Johnson's (1962) view of connective tissue as a dynamic tissue with an internal remodelling system.

Greater understanding of the nature and turnover rates of the tissue components would almost certainly shed much light on both the aging and pathological processes in connective tissue. Identification of the influences controlling and regulating these processes and possible disruption factors are of primary importance for etiological considerations of joint diseases (Mankin, 1968; Castor, 1972).

A number of possible controlling hormones have been proposed by various experimenters. However, since such experimentation is almost always on animal tissues, caution must be shown in applying the results to the human system as effects of hormones may well be species-specific. This is an area where there is still much to be learned of the fundamental roles and relationships (Castor, 1972).

Mucopolysaccharide synthesis has been shown to be stimulated by the administration of androgen, oestrogen, thyrotropin and somatotropin whereas synthesis of mucopolysaccharides can be suppressed by thyroid hormone. Somatotropin has also been observed to stimulate collagen formation. The most widely studied groups of suppressive hormones are the adrenal glucocorticoids. The action of these have been shown to suppress the turnover of the suphated mucopolysaccharides, suppress both the oxidative and glycolytic metabolism of rheumatoid synovium, reduce the hexosamine to collagen ratio (Sobel et al., 1958) and to reduce the cellularity and dermal thickness of skin (Castor and Baker, 1950). Hydrocortisone has been shown to affect the activities of connective tissue cells including accelerating mitosis, suppressing collagen deposition and depressing the specific rate of hyaluronic formation (Castor and Miurden, 1964; Castor and Fries, 1961). Vitamins could also have an important role but again information on their specific influences is minimal. Those having possible effects noted so far include ascorbic acid - important for collagen fibre formation; vitamin A - an excess can increase osteoclastic action in bone (Selye, 1958) and trace elements (metals) - particularly a copper-deficiency can affect both quantity and quality of connective tissue components. Experimentation with chemicals or drugs has also identified blocking or stimulating substances (e.g. BAPN from sweet pea and CTAP) and could lead to further insights into the metabolism and regulatory processes (Castor, 1972).

3.1.4 Anatomy: Joint Lubrication.

The functions of the joint components can be seen as aiming to achieve two specific goals. First, to provide strength and elasticity for resisting the forces and stresses imposed upon the joint and secondly, to provide an efficient lubrication system to minimize friction in the joint. A number of theories of joint lubrication have been taken from the fields of engineering and applied to the biological joint. The complexities of movements of joints and the range of different kinds of stresses they will be subjected to, demands various means of lubrication to cope with the different circumstances. No one theory alone, therefore, covers all the features of the synovial joint system, rather it would seem several means of lubrication are operating together to achieve the same ends of protecting the more or less irreplaceable bearing surfaces of the articular cartilage from coming into contact and of minimizing friction. Both of these goals are achieved with great success; the coefficient of friction in synovial joints is less than that of ice sliding on ice (Gardner, 1972).

The synovial joints can be further classified by the shape of the articulating surfaces of the bones. The facet joints of the spine are "plane" joints, characterized by articulating surfaces which are usually curved and the amount of curvature on the two components of a joint is unequal; the convex surface having a greater degree of curvature than the concave. This incongruence of the articulating

surfaces causes a wedge-shaped gap to exist between the two bones which is of importance for lubrication mechanisms.

Movement in the joint causes the synovial fluid to move around the joint. As the fluid flows through the narrower part of the wedge-shaped gap, its incompressible nature causes an increase in both its rate of circulation and its pressure. The pressure is great enough to keep the moving surfaces apart. The synovial fluid acts as a non-newtonian fluid in that its viscosity decreases with increased velocity of movement. This is thought to aid circulation of the fluid around the joint. Conversely, the increase of viscosity with lower velocities of movement is thought to help reduce friction. These features fit a model of lubrication called hydrodynamic lubrication requiring incongruous bearing surfaces, a viscous lubricant and a relative speed of movement.

Hydrodynamic lubrication is not suitable for dealing with slowly moving, heavily loaded joints with reciprocating movements. It seems also that under certain conditions, the viscosity of the lubricating fluid is of no importance and the main factor is the nature of the sliding surfaces. Under these circumstances a mechanism called boundary lubrication is thought to operate. The moving surfaces are separated by a thin layer of lubrication of only a few molecules thickness which adheres to or is incorporated into the articular surfaces. The coefficient of friction in this case is independant of the velocity of movement.

Engineering bearing surfaces in lubrication systems are smooth and it was assumed this would be the case for the articular surfaces of the cartilage. However, studies have shown the surfaces to be far rougher than those in any mechanical joint. The roughnesses on the surface of the articular cartilage or undulations fall into two types: so-called secondary undulations of 200 - 500μm diameter on which are superimposed so-called tertiary ovoid hollows of about 20-50μm diameter. The observation of such unexpected surface roughness stimulated the creation of another model of lubrication called boosted lubrication.

Synovial fluid becomes trapped as pools in the undulations where under pressure of load bearing the synovial fluid undergoes enrichment by losing water content and other substances of low molecular weight through diffusion, leaving a gel form of synovial fluid with a high concentration of hyaluronic acid-protein complex, the substance responsible for synovial fluid's stickiness and viscosity.

A last mechaniam to be considered of that of hydrostatic lubrication, a form of lubrication where fluid is pumped into the joint under pressure to keep the moving surfaces of the joint apart. A specialized form of this is thought to operate in synovial joints, called 'weeping' lubrication. A property of articular cartilage is its ability to absorb synovial fluid which then oozes out or 'weeps' under pressure providing a continuous film of fluid. This is seen as a supplementary means of lubrication to hydrodynamic

lubrication and also to boundary lubrication in special conditions.

It is seen then what a vital role the synovial fluid plays in joint lubrication. Synovial fluid represents a fluid ground substance, containing a single mucopolysaccharide-hyaluronic acid synthesized by synoviocytes. Hyaluronic acid is responsible for the characteristic viscosity and stickiness. The hyaluronic acid is normally bound to a protein-hyaluronate-protein which is also an important factor in lubrication. Synovial fluid is also important in carrying nourishment to the articular cartilage. Movement of the joint for circulating the synovial fluid is essential both for nutrient flow and for hydrodynamic lubrication. Too little exercise of the joints has been observed as equally damaging as too much (Gardner, 1950; 1972; Gardner and Longmore, 1974; Walker et al., 1968).

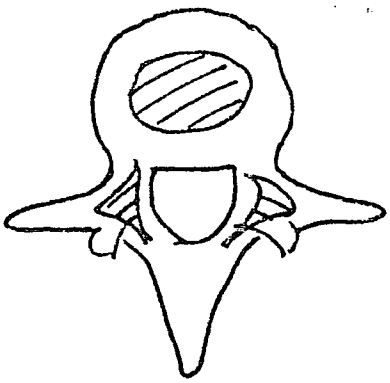
3.2 Development.

The first structure having a stiffening function in the embryo is the notochord which provides a framework around which the true vertebral column is formed, initially as a cartilaginous vertebral column which subsequently undergoes ossification. As the cartilaginous vertebral bodies enlarge they compress the notochord into the dense intervertebral discs where the notochord cells degenerate as part of the development of the nucleus pulposus.

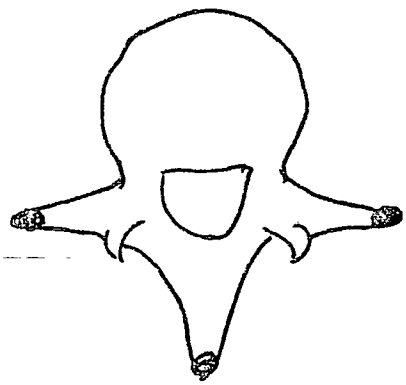
The majority of the vertebrae have three main centres of ossification which appear around the eighth week: one for the body and two for each side of the neural arch (see Fig. 3.3). There are also three secondary centres which appear around sixteen years of age on the tips of the transverse and spinous processes (see Fig. 3.3). Also around sixteen years of age, the ring epiphyses of the upper and lower surfaces of the vertebral bodies ossify and fuse (see Fig. 3.3). The rami of the arches unite first in the lumbar vertebrae during the first year of life followed by the thoracic and cervical. The arch first fuses to the body, at the so-called neurocentralsynchondroses in the cervical vertebrae in the third year of life and lastly in the lumbars in the sixth year of life (see Fig 3.3). The exceptions are the first and second cervical vertebrae - the atlas and the axis. The atlas has three centres of ossification, one for each lateral position appearing in the seventh week and one appearing later at the end of the first year to the anterior arch (see Fig. 3.3). The axis has five main centres and two secondary centres: the body and the neural arch are the same as the other vertebrae, except that the centre of ossification for the body appears later in the fourth month. The dens, which is derived from the body of the atlas, has two laterally placed ossification centres appearing in the sixth month which unite into one structure at birth without the tip, for which a separate ossification centre appears at two years of life. The union of the body, dens and tip occurs just before adolescence (see Fig. 3.3).

FIG. 3·3 OSSIFICATION OF THE VERTEBRAE

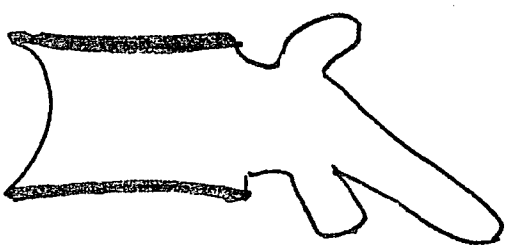
PRIMARY OSSIFICATION CENTRES



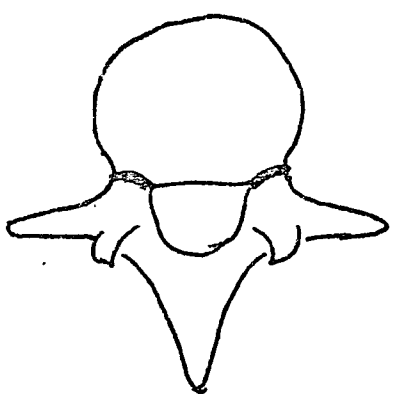
SECONDARY CENTRES



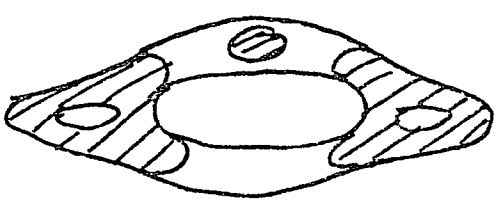
RING EPIPHYSES



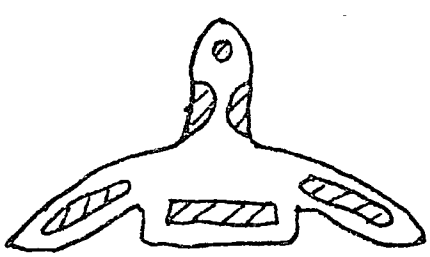
NEUROCENTRAL SYNCHONDROSES



OSSIFICATION CENTRES OF THE ATLAS



OSSIFICATION CENTRES OF THE AXIS



The sacral segments are separated from each other in infancy by intervertebral fibrocartilages. The lowermost two unite around eighteen years of age and union proceeds gradually upwards. The epiphyses for the superior and inferior aspects of the vertebral bodies fuse around sixteen years of age; the epiphyses on the lateral aspects of the sacrum fuse between eighteen and twenty years of age (Epstein, 1976).

3.3 Functions of the Spine.

The structures of an antatomical feature such as the spine have to be seen as a result of the functional demands placed on it to bring about adaptation from its evolutionary precursor.

The history of the spine started with early fishes where the demand for a skeleton of some kind for locomotion, plus the necessity of a streamlined shape, resulted in the development of an internal longitudinal structure for muscle attachments. In the evolution of land animals the spine changed from being solely concerned with locomotion to a dual function in locomotion and support. This dual function placed demands on the spine's design to cope with both mobility and load-bearing, often at the same time so that flexibility had to be balanced by the need for strength and stability. The final demand placed on the design of the human spine arose from the transition from quadrupedalism to bipedalism which necessitates the shift of the centre of

gravity of the trunk to be over the hindlimbs. Other bipeds achieve this by balancing the forward thrust of the trunk by a tail or by bent knees and hips. However, human ape ancestry led the human development of bipedalism towards the unique straight-kneed, tailless form (Evans, 1982).

The spine is particularly subject to compressive loads, bearing as it does the whole weight of the body and it is resistance to compression that has been of primary importance in the development of strength. The design of the intervertebral disc maintains the segmental structure allowing mobility but also can support large compressive loads by converting unidirectional loads into stresses acting in all directions. The property of the nucleus pulposus' strong affinity for water and subsequent high water content gives the nucleus pulposus an intrinsic tension even in the absence of stresses operating and increases the disc's resistance to any forces. The force is distributed outwards from the nucleus pulposus to the surrouding annulus fibrosus and cartilage endplates. The strong intermolecular links formed between the collagen molecules in the annulus fibrosus makes the collagen virtually inextensible and the greater the tension on the annulus fibrosus the tighter the fibres pull together and in this way resist the increased pressure from the nucleus pulposus.

The cartilage endplates are structurally weak in resisting forces but act to distribute forces equally over the surface of the vertebral body. Under heavy compressive

forces the cartilage endplates are the first structures to give way.

The cancellous bone of the vertebral bodies is arranged in trabeculae along lines of compressive force. There are also two fans of trabeculae from the surfaces of the vertebral body into the articular processes. Through the length of the whole spine the vertebral bodies and articular facet joints form a three pillar structure of support.

The intervertebral disc is less good at resisting torsion and bending forces so mobility is limited to restrict the occurrence of such forces. The projections from the neural arches i.e. transverse processes, spinous processes and articular processes restrict mobility between vertebrae, although this varies in different parts of the spine, as described earlier. In particular the articular joints seem to prevent excessive torsion and indeed seem to resist torsion the more the compressive load is increased. Damage or degeneration of the articular joints therefore reduces the limits on torsion and increases the risk of disc degeneration. The disc's own resistence to torsion depends on the annulus fibrosus as the gel of the nucleus pulposus can provide no resistence to torsion, so damage to the annulus fibrosus can play a crucial role in the degeneration of the disc.

The disc, although linking the vertebral bodies, actually tends to push them apart due to the outward force exerted by the nucleus pulposus. The overall intrinsic

stability of the spine is provided by the ligaments running the length of the spine binding the bodies together. These ligaments are under tension and so pull the bodies towards one another, counteracting the opposite force of the disc.

The S-shaped curvature is a very distinctive feature of the human spine. The thoracic kyphosis is a trait of all mammals and is related to the respiratory cage. The cervical lordosis serves to counteract the forward weight of the head to maintain the centre of gravity over the hips. The lumbar lordosis, not seen in any animal other than humans, partially counteracts the thoracic kyphosis, but more importantly appears to be related to attaining the last few degrees of upright stance with straight knees since the lordosis is not present in humans when tilting slightly forwards (Evans, 1982).

Kapandji (1974) has argued an additional role for the curvature of the spine in weight-bearing. Drawing on engineering principles he argues a curved column can resist axial compression at about ten times greater force than a hypothetical straight column.

The vertebral bodies are linked by two longitudinal ligaments running anteriorly and posteriorly from the sacrum to the basi-occipit. Both are firmly attached to the intervertebral disc and adjacent bone margins, but less so to the rest of the vertebral body.

3.4 Identification of Bone Elements.

Identifying the position in the spine of a vertebra is necessary in archaeological samples. The three groups of vertebrae each have different morphology from one another and so can be easily distinguished. The identification of the specific position within the group can also be done with relative ease if the spine is more or less complete. However, without other segments of the group preserved identification can be more difficult. This is particularly true of the thoracic vertebrae.

The first two cervical vertebrae are very distinctive and easily identified. The first, called the atlas, has lost its vertebral body to the second, called the axis, where it is fused onto the axis' own vertebral body in the form of an upright pivot, called the odontoid process or dens, around which the axis turns, forming an additional joint to the facet joints. The other cervical vertebrae are distinguished by quadrilateral shaped bodies, a triangular shaped spinal canal and backwards and upwards facing superior facets. The cervicals have two particularly characteristic features. The spinous processes are forked or bifid at the ends. The transverse process forms the back wall of a foramen, the foramen transversarium, which carries the vertebral arteries and veins. The rest of the bone surround of the foramen transversarium is the homologue of a rib and gives rise to anterior and posterior tubercles. The sixth and seventh cervicals can be distinguished from the other cervical

vertebrae. In the sixth cervical the anterior tubercle is very prominent and is called the carotid tubercle. In the seventh cervical the spinous process is very prominent and ends in a tubercle. The transverse processes of the seventh cervical are also more prominent than the others and the foramen transversarium is smaller as the vertebral arteries and veins are not carried beyond the sixth cervical.

The thoracic bodies are larger than the cervical, increase in size progressively down the column and change from quadrilateral in shape to more rounded. The transverse processes point slightly backwards and end in blunt rounded tips, the superior facets face backwards and slightly upwards and laterally up to the tenth thoracic. The spinal canal shape changes from oval through circular to a rounded triangular shape from the first thoracic through to the twelfth. The spinous processes of the upper thoracic spine are heavy and are angled sharply downwards becoming increasingly vertical down the column. Particularly characteristic of the thoracic vertebrae are the presence of articular facets on the bodies and on the tips of the transverse processes for articulation with the rib heads and rib tubercles. The first, ninth, tenth, eleventh and twelfth differ from the others and can be identified from the others. The first thoracic bears similar resemblances to the cervical vertebrae in having a quadrilateral shaped body and relatively long transverse processes. In the thoracic vertebrae from the first through to the eighth, the articular facets for the ribs are positioned between vertebrae so take the form of a demi-facet

on the superior and inferior edges. In the ninth thoracic this demi-facet only occurs on the superior edge. In the tenth through to the twelfth only one complete facet occurs, the transverse processes are short and have no articular face for the rib tubercle. The patterns of the superior facet joint changes between the tenth and twelfth thoracics usually at the eleventh thoracic to the lumbar pattern being concave and facing backwards and medially.

The lumbar vertebrae are distinctive in their size, being much bulkier than the thoracics, wider from side-to-side than from back-to-front with heavy spinous processes which are quadrangular and lie horizontal. The spinal canal is larger than in the thoracics but not so large as that of the cervicals, and is triangular in shape. The superior facets are concave and face medially and somewhat backwards. The transverse processes vary, being shorter in the first, second and fourth than in the third, and are variable in the fifth. In the first three lumbars the transverse processes originate from the junction of the pedicles and the neural arch, whereas in the fourth and fifth lumbars it originates from the pedicle itself. The fifth lumbar is usually markedly wedge-shaped, the front being much deeper than the back.

3.5 Introduction to Osteoarthrosis and Osteophytosis.

Osteoarthrosis is the commonest form of the arthritic diseases in modern populations, and most probably was also

so in the past. The recognition of osteoarthrosis as a separate disease from rheumatoid arthritis was achieved by Adams in 1859. As with most of the arthritic diseases, there are numerous synonyms for osteoarthrosis in the medical literature. Garrod introduced the term osteoarthritis in 1890, which has been the most commonly used term until recently. Osteoarthritis implies as inflammatory process and is therefore an incorrect description of this disease which is non-inflammatory. Osteoarthrosis is used here (Nuki, 1980). Also popularly used in current literature is Degenerative Joint Disease, a term pioneered by Bennett and Bauer (1937). Osteoarthrosis denotes a non-inflammatory and progressive disorder of the joints causing degeneration and loss of articular cartilage, with associated proliferation of the articular tissues at the margins and base of the joints. The disease is localized to any given joint or even part of a joint rather than a systematically expressed disease. The sequence of changes in the various articular tissues is very little understood and indeed it seems most unlikely that a definite description of the relative timing of changes will ever be possible. Osteoarthrosis appears to cover a wide range of patterns of changes in articular tissues all bringing about the same overall result of degeneration and proliferation (Sokoloff, 1980). Osteoarthrosis is further divided into primary and secondary osteoarthrosis. Osteoarthrosis is associated with mechanical demands on the joint, a wear and tear process that is progressively cumulative in its effects over the years. Secondary osteoarthrosis is found where injury, trauma or

some indirect factor predisposes the joint to abnormal mechanical strain. Primary osteoarthrosis would appear to occur spontaneously with no very obvious predisposing factor. In discussions of etiological factors of course, any factor which causes an increase in mechanical demand or a decrease in the joints resistance can be an important influence which leaves the range of causative factors very wide (Radin et al., 1980).

There is a very strong correlation with age both in frequency in a population and the severity of the disease. Debate persists as to whether the normal changes of senescence include the changes of osteoarthrosis or whether osteoarthrosis is to be interpreted as something abnormal at any age. Bennett et al. (1942) suggested that the changes in the articular tissues of osteoarthrosis were merely more advanced forms of normal senescent changes. Therefore by a certain age a limited amount of osteoarthrosis could be viewed as a normal condition (Bohatirchuk, 1955, 1957). At what point a distinction is made between a normal amount of osteoarthrosis and a pathological state is almost undefinable. Stecher (1961) by contrast states that the changes in the articular tissues of osteoarthrosis include the normal age-related changes, but also involve in addition congestion of the synovial membrane, fibrosis and an increased blood supply. Most recent authors treat osteoarthrosis as a pathological condition, regardless of how frequent it may be in most populations.

Osteoarthrosis is seem as a disease originating in the soft articular tissues, particularly the articular cartilage, with bone responses as secondary developments. This view has traditionally been held by English-speaking researchers in contrast to European work which has emphasized the alterations of bone as the primary change. This latter view has been advocated recently by Johnson (1962) who stresses the dynamic quality of joint tissues which is responsible for continued remodelling of the joint throughout life. When the remodelling rate exceeds the rate of cartilage repair then osteoarthrosis develops.

Since many different patterns of joint changes are recognized to lead to an osteoarthrotic state it is possible for a resolution to be found to these apparently opposed views of pathogenesis in that both could be true and have a close relation to each other (Sokoloff, 1972).

Osteoarthrosis describes a disease of synovial joints which does not include the joints between the vertebral bodies. Again there are numerous synonyms for the comparable degenerative and proliferative changes of these joints. Here it will be termed osteophytosis. Other widely used terms are spondylosis, spondylosis deformans. Degenerative Joint Disease can cover both types of joint as a catch-all phrase. Although the vertebral body joints involve a different form of connective structure the general features of osteophytosis are the same as in osteoarthrosis.

3.6 Degenerative Changes in the Soft Tissue.

As already stated it is impossible to distinguish a sequence of changes in the cartilage and although many authors have done so, reading the different papers together gives a picture of inconsistent results. The following are the main changes noted in the appearance of the cartilage.

The cartilage is seen to undergo focal areas of softening where the cartilage matrix appears to swell and manifests an increased affinity for hematoxylin, which is taken up by collagen. There appears an associated increase of chondrocyte number adjacent to the softened areas and a decrease in number of chondrocytes within the actual area itself. The cartilage splits along the planes of the collagen fibres with a loss of intercellular substance. When this dehiscence goes no further than the tangential layer of the surface, it is termed flaking. Once the disruption and greater loss of intercellular substance extends to the deeper radial layer, it is called fibrillation. Clusters of chondrocytes have been reported at the edge of the minute fissures in the cartilage, seen either teleologically as a response to the dehiscence in a reparative attempt, or mechanistically, as resulting from increased access to the nutritive synovial fluid (Sokoloff, 1972). The fibrillated cartilage has lost the smooth, shiny appearance of normal cartilage and the surface appears irregular (Stecher, 1961). The fibrillated cartilage becomes eroded and thinning of the joint space is a common roentgenological

finding (Jaffe, 1972). On the other hand, proliferation of chrondrocytes can cause thickening of cartilage in other parts of the joint (Stecher, 1961). The areas affected by erosion and thinning are thought to be the areas of pressure in the joint, while the non-pressured areas experience proliferation and thickening (Stecher, 1961). However, Bullough et al. (1973) have reported the earliest erosion occurring not in the main pressure areas, so the relationship between strains on the joints and responses are not direct and straightforward as at first thought. The changes of fibrillation also occur as a senescent process.

In addition there are changes reported which appear to be specific to osteoarthrotic cartilage. Normal cartilage does not show alteration in cell counts once adulthood is achieved whereas evidence of chondrocyte death has been identified at submicroscopic level (Meachim and Collins, 1962). Stecher (1961) claims congestion of the synovial membrane and fibrosis of the capsule are other changes not seen in normal aging tissue. Lloyd-Roberts (1953) has studied the matter causing congestion in the joint capsule and finds it to be cartilage and bone debris, thought to have been eroded from fibrillation and the further extensive damage which reaches the bone. Work of the hip has demonstrated a proliferation of blood vessels penetrating into the cartilage from the subchondral marrow (Jaffe, 1972), which is not observed in normal cartilage. Stecher (1961) sees the invasion of capillaries and larger blood vessels as part of a process in the non-pressure areas which starts with a

proliferation of cartilage which in turn degenerates and is invaded by these blood vessels. Remodelling processes occurring in the subchondral bone cause a shift and reduplication of the cartilage/bone border, as bone replaces degenerated cartilage (Stecher, 1961; Jaffe, 1972). Hollander and Horvath (1953) report that the intra-articular temperature is increased in osteoarthrotic knees compared to normal ones. The histologically observed results of cartilage change of dehiscence, fibrillation, erosion etc. have been reported many times and are widely recognized now, only the interrelationships are less well understood.

Researchers have turned to studying the biochemical alterations of cartilage for further insights. Much work in this area comes from Mankin and his colleagues and it is from their publications mainly that this summary is drawn (Mankin and Baron, 1965; Mankin and Laing, 1967; Mankin, 1968; Mankin and Lippiello, 1970; Mankin et al., 1971; Mankin, 1974). These are changes found specifically in the biochemical structure of osteoarthrotic cartilage not just aging cartilage. The matrix of the cartilage undergoes a reduction in the proterglycan content with osteoarthrosis (Mankin and Lippiello, 1970; Maroudas et al., 1973). The proteoglycan molecules supply most of the charge of cartilage and it is thought the ionic charges bind water in the joint, a concomitant decrease in water content would be expected with the proteoglycan decrease. However, there is no evidence for this and there is some evidence for an increase in the water content in

osteoarthrotic cartilage (Ehrlich and Mankin, 1980). It has been proposed that this may result from water becoming trapped in fissures in the osteoarthrotic cartilage and thus giving a false impression of the bound water content, or that in the absence of proteoglycans, water binds to the collagen molecules (Ehrlich and Mankin, 1980).

Actual production of proteoglycan has been demonstrated by a number of researchers to increase in osteoarthrosis. The increase in production is proportional to the severity of the disease up to a certain degree, at which point production drops sharply. This is seen as a point of failure by Ehrlich and Mankin (1980) and this concept recurs in their observations on biochemical alterations.

The type of glycosaminoglycans produced changes in osteoarthrotic matrix in that keratan sulphate decreases relatively and the ratio of chondroitin-4-sulphate: chondroitin-6-sulphate increases. This finding seems controversial, other workers not having obtained this result. Mankin (1975) reported no increase in production of chondroitin-4-sulphate so some preferential degradation of keratan sulphate and/or chondroitin-6-sulphate must be operating if the changes reported are confirmed in further researches.

There has to be an increase in the degradation of proteoglycans if both a decrease overall is recorded together with an increase in production. A number of

degradative enzymes have been found to increase their activity in affected tissues. again in proportion to the disease's severity. The fact that the degradative activity is initiated from those focal areas of cartilage affected (Ehrlich et al., 1973), while the production is also enhanced, presumably in a reparative response, involves a balance between the two processes which many factors could disrupt.

The collagen production in the cartilage has been shown to increase along with collagenase activity. Again this increase is proportional to the disease's severity up to a point of failure.

Similarly with cells, a proliferation occurs particularly in the early stages of the disease, increasing with severity to the point of failure. So the biochemical changes in osteoarthrotic cartilage involve on one hand reparative action with an increase in proteoglycan and collagen production and cell proliferation balanced by degradative activity of enzymes and collagenase. There seems to be a point of failure where the disease has progressed to a certain severity and reparative activity falls off. The mechanism by which these reparative and degradative activities are triggered on and off is unknown, or at best, speculative. The precise effect of these changes on the functional integrity of the joint and its resistance to stress are equally unclarified.

The synovial membrane is the least affected of all the

joint tissues, but in severe states of osterarthrosis will become congested and somewhat villous. Jaffe (1972) lists possible alterations that may be seen in the membrane:

a. Islands of cartilage proliferation

b. Small pedunculated fatty and fibrous polyps containing cartilage, which may become partly ossified.

c. Villi, which have become necrotic through being caught and compressed between bones.

d. Large, bulbous, fatty villi.

The membrane may come to incorporate debris from the articular surfaces of bone and cartilage, which can cause irriation and proliferation. The joint capsule may become distorted or even slightly thickened (Jaffe, 1972).

There has been some suggestion of alterations in the synovial fluid, causing disruption of the lubrication processes. Diseases involving disruption of the fluid such as haemophilia or gout, are often accompanied by the onset of degenerative changes (Jurmain, 1975). However, Sokoloff (1972) concludes that there is no satisfactory evidence for a role by defective lubrication in the pathogenesis of osteoarthrosis.

3.7 Degenerative Changes in the Hard Tissue.

The complimentary changes in the bone tissue of the joints are of five kinds:

a. osteophyte formation

b. sclerosis of bone under the damaged cartilage

c. pitting and eburnation

d. formation of subchondral cysts

e. shifting and reduplication of the bone/cartilage border.

Bone growth in osteoarthrosis can occur at two sites in the joint. Exophytic growths, or osteophytes, are seen at the margins of the joint and deposition of new bone takes place in the marrow cavity subjacent to the cartilage. Osteophytes are the most characteristic osteological sign of osteoarthrosis. However, although always present in the disease, they are not a specific diagnostic criterion, if found alone. Reports of osteophytic growth in old but non-osteoarthrotic joints such as the humerus and the femur have been published (Jaffe, 1972). Osteophytes can follow one of two patterns of growth, either as protuberances into the joint spaces or as ossifications of the capsular ligamentous attachments (Sokoloff, 1972). In both cases the direction of growth follows the lines of mechanical force exerted on the joint, taking the path of least resistance by following the contour of the joint outwards. The osteophytes are usually found to have formed continuously with the spongy trabeculae of the adjacent bone area. They, themselves, are composed of spongy trabeculae and fatty intertrabecular marrow with a layer of bone continuous with the joint surface (Jaffe, 1972). The bone of the osteophyte is normally fragile and covered by a fibrous cartilage and possibly synovial fibrous tissue (Sokoloff, 1980). The osteophyte will itself become involved in the osteoarthrotic process, its cartilage fibrillated, abraded and the

osteophytic bone exposed.

A favourite explanation for the presence of osteophytes is a teleological, mechanical one. The osteophyte provides an increase of the bone articular surface over which loads can be spread relieving the pressure from the overstressed joint (Radin et al., 1980). However, the bony outgrowths can cause impediments to the mobility of the joints, impairing function (Sokoloff, 1969, 1980). The origins of the osteophytes, or the triggers to their growth are speculative. The most popular idea is that the osteophytes arise from overgrowths out from the subchondral bone. Vascularization of the cartilage from the subchondral marrow increases in osteoarthrosis (Stecher, 1961) and the area on the periphery of the joint are particularly well-nourished, with the presence of adjacent synovial vessels. Cartilage around this region becomes calcified, forming new enchondral bone as part of the remodelling process. Collins (cited in Jurmain, 1975) sees this overgrowth of bone, stimulated by increased vascularization, as part of a process where cartilage has first been worn away from the bone surfaces. However, a problem with the Collins view and the view of Radin et al. (1980) where osteophytes are a direct response to mechanical stresses, is that osteophytes form in regions of the joint of low stress and which do not transmit weight (Harrison et al., 1953). Stecher (1961) presents a model of a process whereby cartilage proliferation occurs in non-pressured areas and subsequently degenerates from inadequate nourishment. This inadequacy arises from the cartilage

becoming too thick for diffusion to operate in the absence of sufficient action and movement in the non-pressure areas. Increased vascularization, ossification of the degenerating cartilage and osteophyte formation are then the result. Johnson (1962), as will be seen below, sees joint remodelling and imbalances therein as the primary initiating factor in osteoarthrosis. This view then sees the osteophyte as resulting from some upset of equilibrium between formation and resorption, similar to the cartilage changes already noted. The exact roles of the relationship of such a disequilibrium to function and distribution of stresses is still obscure. Osteophyte formation is, therefore, best seen for the present as a single response to many different patterns of preceding events (Sokoloff, 1969).

As the articular cartilage is worn away, the subchondral bone becomes exposed and will rub against the opposite articular bone surface. Bone growth in response to this friction is manifested by sclerosis of the bone. This is deposition of new bone on the existing trabeculae giving a hard, dense appearance to the bone surface. Continued rubbing gives the bone a polished appearance reminiscent of ivory and thus termed eburnated. With eburnation the surface layer will show necrosis of osteocytes, presumed through heat generated locally from friction (Sokoloff, 1980). More extensive eburnation. with grooves forming and minute fractures, is accompanied by more extensive bone necrosis. This situation is the common state of severe osteoarthrosis and the necrosis of the bone is most probably the proximal

cause of much of the observed deformity even if not the true ultimate cause of the osteoarthrotic changes.

In spite of the hard, ivory appearance of sclerotic bone, it can be quite weak, mainly due to formation of cysts below the articular surface. Such areas represent degeneration of marrow and trabeculae and new bone is formed encircling the spaces, These cystic areas have been shown to be deficient in blood vessels and most often positioned with the apex at the eburnated articular surface. These cysts sometimes contain fluid and it has been proposed by Landells (1953) that the cysts are formed by the intrusion of synovial fluid, or at any rate of pressure coming from the articular cavity through defects in the articular cortex into the bone marrow. The pressure is dissipated radially to the surrounding bone marrow compressing blood vessels in the region and leading to the degeneration of blood supply, marrow and trabeculae locally.

Finally, throughout life remodelling takes place in both bone and cartilage which can affect the contour of the joint. In skeletal growth, both cartilage and the bony epiphyses contribute to enlargement through endochondral ossification of calcified cartilage. The border between the calcified and non-calcified cartilage is known as the tide-mark in histological preparations. Old tissues characteristically show the presence of several parallel tidemark lines witnessing to a progression of calcification and new bone formation through remodelling. Although not

per se an osteoarthrotic change emphasis has been laid by Johnson (1962) on this process as the initial process to be disrupted in the development of osteoarthrotic changes, thereby interpreting the hard tissue changes as the alterations of primary importance rather than those in the cartilage.

3.8 Disc Joint Changes in Osteophytosis.

As noted earlier, Sokoloff (1972) while accepting a terminological distinction between the degenerative joint diseases of the synovial and disc joints, sees no reason for this necessarily to involve a difference in the pathological processes. It is common to find a strong correlation between the degeneration of the two types of joint in the spine. The changes occurring in the respective tissues of the two kinds of joint are remarkably similar. The fibrillation of the cartilage endplates, by which the disc is attached to the vertebral bodies, is indistinguishable from that of synovial articular cartilage. Similarly, with degradation of the intervertebral disc, the bone changes involve eburnation and osteophyte formation.

The formation of osteophytes is thought to occur in response to the mechanical stimulus of the protrusion of the annulus fibrosus against the ligaments on the anterior margin. Posteriorly placed osteophytes can occur and, although are far less common, are of greater clinical importance since they protrude into the spinal canal. It is possible to find advanced degeneration of the disc without

concomitant osteophyte formation and also the converse where osteophytes are well formed with no evidence of obvious degeneration (Jaffe, 1972). It would seem therefore, that the formation of osteophytes is not a direct response to degeneration and forward protrusion of the disc, but more directly from the pull on the ligaments under pressure. This can occur in physical labour without degeneration of the disc first (Jaffe, 1972).

3.9 Using Degeneration in the Spine for Aging Purposes.

The identification of the osteological units of the spine is fairly simple, certainly at least into the three regional groups.

The identification of degenerative joint disease is not so simple as it is thought that the initial changes occur in the soft tissues. This, of course, is not available in archaeological samples. The most distinctive bone change is the development of osteophytes. This may not, by itself, represent degenerative joint disease, but is a concomitant development of age. Therefore, although it may not be strictly true to refer to osteophytic growths as degenerative joint disease, their presence in non-degenerative states is equally related to age so will serve the purposes required here. Students of palaeopathology may wish to be more precise in their diagnostic criteria. The more severe cases will manifest sclerosis and eburnation which are easily recognized.

Chapter 4. ETIOLOGY AND AGE RELATIONSHIPS OF DEGENERATIVE JOINT DISEASE.

There is a multitude of publications on research into etiological factors which it would be quite impossible to cover fully. Therefore, only a very few papers have been selected for discussion here to provide an illustration of the main issues under debate. Two books have been of particular help as introductions to the subject and the questions researchers are tackling. These are:
Sokoloff, L. (1969) The Biology of Degenerative Joint Disease.
and
Nuki, G. (ed.) (1980) The Aetiopathogenesis of Osteoarthrosis.
In addition the theses by Jurmain (1975) and Chapman (1968) have been useful guidelines for an anthropological perspective.

The review of etiological factors is divided into sections defined by the nature of the research: comparative studies, experimental induction, natural secondary occurrence, population studies of the primary form and archaeological evidence of the disease. Specific studies of the spine are then considered, followed by a review of the reports of relationship of the disease to age and sex, first, in general and secondly, in the vertebral column. This very last part is, of course, the one of most direct relevance to the aims of

the thesis.

The range of different results to have come from research into the possible determinants of degenerative joint disease point to one conclusion only, that degenerative joint disease is the final common outcome of many different etiological factors, processes or pathways. Radin et al. (1980) do not mince their words over the matter : (p. 84) "anyone who attempts to suggest that osteoarthrosis is caused by a single factor, be it congenital, developmental, vascular or traumatic, is a fanatic and should be treated as such." The function of the joint depends on a balance maintained between the forces exerted on the joint, and the joint's ability to withstand these forces. Any factor which either increases the mechanical demands on the joint, or decreases the joint's resistance, to a point where there is an imbalance between demand and the resistance will be a potential etiological factor. Many such factors have been identified through experiments and epidemiological studies, and in any incidence of degenerative joint disease, a number of etiological agents are probably involved. It is necessary to explain the different distribution of incidence within a population, between populations, between different joints of the body or even between different parts of the same joint and the level of severity observed. Difficulties in finding one etiological agent to explain all these observations again suggests an involvement of multiple factors.

There appear to be four kinds of degenerative joint disease which may have varying importance of etiological determinants to one another. The four are:

Osteoarthrosis - of synovial joints

Osteophytosis - of intervertebral joints

Generalized osteoarthrosis - multiple joint involvement

Heberden's Nodes - involvement of digits.

It has even been suggested that osteoarthrosis of each joint should be treated as a separate disease, that is as osteoarthrosis of the hip, of the shoulder etc. (Stecher, 1961). There is, of course, some validity in the idea that each joint has its own peculiar structure and specific demands upon it and will therefore be more or less susceptible to the various agents. For this reason, the structure of particular interest here, the spine, is treated separately at the end of the review, albeit at the expense of some repetition. Nonetheless, in the overall review here, only the four forms listed above will be referred to specifically. The four types also overlap considerably in the relationship of factors which will be implicit when all four are treated together under the general term of degenerative joint disease.

Jurmain (1975) groups the possible factors in etiology into two broad categoris as follows:

systemic factors - age, sex, metabolic, hereditary, vascular, endocrine, disturbed nutrition.

operating factors (mechanical/functional) - trauma (acute), occupational (chronic trauma), increased weight-bearing from obesity.

Systemic factors alone seem unlikely to explain the occurrence of the disease which is localized, often to one joint or part of a joint. Mechanical demands, on the other hand, do not always explain distributions within a population or localization in non-weight bearing joints, such as the hands which are frequently involved. A complex interaction exists between the systemic and mechanical influences representing the internal and external environments of the joint.

As mentioned above, the material is grouped here by the type of study rather than by etiological factor as in Jurmain (1975). This is thought to better emphasize the multiple involvement of factors, rather than treating each factor in turn which draws the reader to thinking in a single factor context.

4.1 Inter-specific Comparative Studies.

Results from inter-specific comparative studies with other animals give insights into disease processes. Some diseases are species specific and cannot be compared in this way and it must always be questionned if an apparently similar non-human disease can properly be defined as the same disease as the human variant.

A report by Stecher (1958) of degenerative changes in the left knee and lower spinal bodies of an aged Gorilla, can be used as an example of the similarity of the changes to those of human degenerative joint disease. Stecher (1958)

describes the changes and states that the observed abnormalities in the gorilla skeleton can very easily be interpreted as three diseases observed in human subjects: sacralization of the fourth lumbar vertebrae to the sacrum (gorillas only have four lumbar vertebrae), osteophytosis of the second and third lumbar bodies and osteoarthrosis of the synovial joints seen in the left gorilla knee. Perforations were also present on the lumbar bodies ressembling those sometimes observed in human material and thought to be the result of a proliferation of blood vessels. Fox (1939) made a comparative inter-specific study of joint degeneration, based on 1749 animal skeletons and claimed that the identification of lesions in non-human species was not difficult , finding the examples showed strong similarities to human degenerative joint disease.

The association of degenerative joint disease with nutritional disorders has been discussed by Sokoloff (1960) in a review of animal studies. There is no evidence of degenerative joint disease connected with rickets or excess vitamin A intake in horses. Avitaminosis can lead to degenerative changes, but this may not be a direct relationship as the epiphyseal development could be affected first causing joint disruption. Investigations of copper deficiencies give contradictory results at present. The administration of copper supplements was reported to alleviate the condition in one report, whereas the evidence that copper deficiency creates defective bone formation suggests that the degenerative changes could again represent

developments secondary to joint disruption. Similar conflicting reports exist on the role of general nutrition on pedal arthritis in horses. A Swiss report claims nutrition appeared not to be at all important, whereas a paper by Greenlee (1939, cited by Sokoloff, 1960) states that dietary supplements can protect against the development of the disease. As regards toxicity, chronic selenosis has been demonstrated to cause hypertrophy and sloughing of the hooves. Fluorosis can lead to cortical hyperostosis of the spine but there is no evidence of an association with ankylosing spnodylitis.

Comparisons of non-human species, experiencing a wide range of different mechanical demands upon their joints, can provide information as regards the role of external factors on joint function. Sokoloff (1960) points out that any horse-dealer "knows" that certain types of body configuration in horses will be more prone to degenerative joint disease, depending on the kind of work to be done. Such differences are presumably genetically influenced via the body build's susceptibility to wear and tear (Sokoloff, 1960). As a general feature, hip osteoarthrosis tends to be rare in horses whereas some breeds of large dog suffer frequently. For example, German shepherd dogs can show a frequency of 67% with severe osteosrthrosis by a relatively early age (Sokoloff, 1973). Pets, especially old cats and dogs may quite often show some degree of degeneration.

It has been a common notion that wild animals tend not to show changes of osteoarthrosis and osteophytosis, but rather that the non-human species showing degenerative joint disease are either those that have been domesticated or live in captivity both of which represent conditions abnormal to the environment for which the species is functionally designed. Two broad comparative reviews refute this notion. Both samples are made up of a mixture of both wild and captive animals, and degenerative changes are observed amongst wild specimens of a species equally well as amongst the captive specimens.

Schultz (1956) has both reviewed the literature and made his own observations on non-human primate arthritides in general. He concludes that what he calls 'chronic arthritis', which appears to be equivalent to osteoarthrosis or osteophytosis, appears to be limited to the catarrhines. It is only rarely seen amongst the lower species of catarrhini (the cercopithecines), is fairly common in baboons and has the highest frequencies amongst gibbons, orangs and gorillas. Amongst this latter group, sufficient information was available to also observe a rapid increase in frequency with age. Schultz does not discuss his findings at all, but the most affected were longer-lived, larger bodied species which is in keeping with Fox's (1939) results (see below). Why only the catarrhini are affected suggests a possible genetic explanation, although the most frequently involved species mentioned by Schultz, have no equally large-bodied, long-living counterparts amongst the platyrrhini, so the apparent

isolation of the catarrhines in susceptibility is misleading.

Fox (1939) studied 1749 skeletons, a mixture of wild, captive and domesticated animals from a wide range of taxonomic groups. He found no relation of degenerative joint disease to phylogeny. The major conclusion was that amongst those species displaying degenerative joint disease, the majority were macrosomic species rather than small bodied ones. Some bulky species seemed to have escaped involvement, for example Rhinoceros and Camel, although Sokoloff (1960) reports that degenerative joint disease has been recorded in these two species by other researchers. Within the affected group, the carnivora display more osteoarthrosis in the fore-quarters and more involvement of the caudal spine in contrast to the artiodactyla where the hindlimbs were the most affected part together with the cephalad spine. Fox (1939) suggests a functional explanation for the observed differences. Jolt-shock and locomotive power may be responsible for the localization of osteoarthrosis in the carnivora forelimbs and hindlimbs of artiodactyla. Similarly, the affected portion of the spine could be related to the degree of mobility present. Where the greatest amount of stabilization of the spine is necessary for the animals' locomotion, there the osteophytosis is seen to arise most. Those animals which hang from trees rather than bear their weight, thereby experiencing tension rather than compression, do not show signs of osteoarthrosis. The primate group show the greatest incidence of osteoarthrosis in the hands and represent the group in which the hand is most evolved for mobility and multiple function.

These two studies still remain the only broad comparative studies and it is regrettable more detailed comparative work has not followed on from their initial 'censuses' in analyses of specific biomechanical stresses of locomotory activities.

4.2 Experimental Induction.

Laboratory experimentation can provide a means of exploring specific potential effects of a single controlled variable. However, the extent of stress exerted by any one variable is normally far greater in experimental situations than ever occurs in natural situations, and thus only provide suggestions of potential factors rather than specific proof. The use of experimental research can serve to eliminate possible influences.

A vast range of chemical and physical insults have been applied to animal joints in experimental situations to induce degenerative joint disease. The major outcome of such experimentation is summarized by Sokoloff (1969).

The introduction into joints of acids, microbial products and irritants, produce osteoarthrotic changes. However, this could well represent an indirect secondary effect provoked by the synovitis caused by the introduced matter. Attempts to selectively alter the matrix balance have not produced totally successful results. The introduction of papain into rabbit joints does produce degenerative changes, but again synovitis is also produced so that it is difficult

to assess the exact degree of the proteolytic action induced on the ground substance, and therefore the drug's direct responsibility for the observed lesions. Kalbhen (1980) gave intra-articular injections to the knee-joints of hens using a number of drugs, chosen for two reasons. First, a chemical was chosen, known to be a potent inhibitor of the glycolytic processes which are responsible for energy balance in chondrocytes. This chemical was sodium iodo-acetate. Secondly, a group of drugs were chosen which are used for anti-inflammatory or anti-rheumatic purposes because they inhibit the synthesis of glycosaminoglycans, collagen and other proteins. These drugs were: sodium salicylate, phenylbutazone; oxyphenbutazone, bumadizone; clofezone, flutenamic acid; niflumic acid, indomethacin; ibuprofen, salicylamide; dexamethasone-O-phosphate. Only quantitative effects on the time and intensity of the progression of osteoarthrosis was found. The most potent agent by far was the sodium iodo-acetate compound. The conclusion therefore was that any chemically induced disturbance of anabolic activities of the cartilage cells can cause the alterations of composition and function leading to degeneration. Of particular importance was inhibition of the anabolic process.

Major incisions of supporting tissues are not always sufficient to lead to osteoarthrotic changes. The procedure seems to need an influence on the joint causing necrosis of the chondrocytes such as electrolysis before degeneration follows. However, degeneration does follow major surgery that leads to a mechanical derangement of the joint in terms

of deformation of the joint contour or induction of instability. One such example of the latter is reported by Williams et al. (1982), who cut the cruciate ligament in rats and observed subsequent changes of osteroarthrosis in the knee joints.

The mechanical hypothesis of degeneration resulting from load-bearing has received much support from experimental studies. These are reviewed by Sokoloff (1969). The traditional version is that excessive forces, particularly compression or increased friction is the mechanical demand necessary to lead to degenerative joint disease. Compression forces which press the joint parts together, do cause necrosis of chondrocytes. This is seen as resulting from hindrances to normal joint movement and to the diffusion of the metabolites necessary for chondrocyte nutrition. However, studies of immobilization of joints, fixed in extension without any compressive forces, also results in necrosis of chondrocytes and the sloughing of cartilage. Presumably, this is from a similar cause, namely that without any movement diffusion of metabolites is equally inhibited as under excessive load.

Experiments of the kinds of mechanical stresses most likely to lead to degenerative joint disease suggest continual impact loading can be the most significant. Radin and Paul (1971) demonstrated the importance of impact over simple loading in a controlled experiment using the ankle joints of cows. The joints were subjected to loads just under the structural capacity for long periods. Such loads

did not lead to the wearing of the articular cartilage. When the same load was exerted combined with impact, wear followed rapidly.

It is evident, therefore, that disturbance of metabolites to the chondrocytes is an important precursor to degenerative changes of the cartilage. As regards mechanical demands, compression is important particularly combined with impact. The various joints may well respond differently to the kinds of mechanical demand depending on their primary joint function.

Genetic influences have been demonstrated to be involved in the occurrence of degenerative joint disease by a series of experiments with mice by Sokoloff. Different genetic strains of mice vary in their susceptibility to degenerative joint disease. As yet the number of genes involved is unknown but the evidence points to the susceptibility being recessive. Correlations are often observed between obese individuals and degenerative joint disease, presumed a result of the greater load-bearing in obesity. However, experiments with different strains of mice and different diets found that obesity per se did not lead to degenerative joint diseases, but rather that a high fat diet lead to both obesity and degeneration and finally that strains of mice resistant to degeneration were also resistant to obesity when fed a high fat diet (Sokoloff et al., 1960). It seems that the liver may be a mediating agent as the obesity-resistant strains were also less prone to develop a fatty liver and concomitant liver problems.

Sokoloff (1969) suggests that some form of disrupted lipid metabolism could be a factor in primary degenerative joint disease, disruption arising in the mice from a genetic origin, but liable to other causes also. Strains of mice with the genetic "scratching" disease were particularly resistant to the degenerative joint disease regardless of diet. Also, the extraction ratio from the thyroid of radioiodide relative to the serum varied between strains and showed a rough correlation with degenerative joint disease (Sokoloff, 1959). Sokoloff does not discuss these findings further but merely uses them as further illustration of a genetic factor.

There appears to be very little work on possible endocrine factors. Hulth (1969) presents a review paper of experiments with mice. Injections of ACTH have been shown to decrease the spontaneous incidence of degenerative joint disease while somatotrophin appears to increase the incidence. How these are involved in an overall framework of interacting factors, or are triggered into action by other contributory agents is not discussed. The main contribution of experimental studies, therefore, has been towards elucidation of mechanical and genetic agents.

4.3 Secondary Degenerative Joint Disease.

Secondary degenerative joint disease describes those cases where the disease is a result of disruption in joint function by a specific event such as an injury, a disease, or an anatomical abnormality acquired at birth or during the

developmental phase. The dividing line between primary and secondary forms of the disease is not always obvious. Indeed, the search for etiological factors causing joint imbalances itself implies that all degenerative joint disease is secondary to some other agent in the joint. However, the term is generally used to refer to those instances of joint disruption where the predisposing agent is very obvious and beyond question. Diseases, or injuries which cause joint disruption likely to be followed by degenerative joint disease may have factors in their own etiology or particular action which sheds light on the etiology of the primary degenerative changes.

Degenerative joint disease is known to follow the metabolic diseases of ochronosis and gout. Ochronosis involves the deposition of homogentisic acid and gout, the deposition of urates. Generalized osteoarthrosis is also associated with gout and also with alkaptonuria. Both of these diseases are known to have a genetic determinant, adding support to the genetic susceptibility theory (Lawrence, 1960). Other disorders having a genetic involvement together with associated degeneration are congenital dislocation of the hip, multiple epiphyseal dysplasia, familial coxarthrosis, Perthes' disease and slipped epiphyses (Lawrence, 1960). Disorders in the spine which may predispose to osteophytosis or degeneration in the facet joints are found too infrequently for any conclusions to be drawn (Kellgren and Lawrence, 1958). Gross level vascular disorders such as arteriosclerosis or gangrenous limbs have not shown any association with

osteoarthrosis (Jurmain, 1975).

Studies of degenerative joint disease as a result of injuries were made amongst the population of Leigh, in South Lancashire, by Kellgren, Lawrence and their colleagues. They found a significant occurrence of degeneration at the site of an earlier injury. Although this was found in both sexes, it was particularly marked in males, many of whom worked in physically demanding jobs such as mining so were more exposed to the possibility of injury. For the knees and tarsal joints, osteoarthrosis was associated with injury in about half of all male cases (Kellgren and Lawrence, 1958). Injury, therefore, represented an important etiological agent in males. A study of hip joints in young individuals by Murray (1973) estimated that half of the hips showing osteoarthrosis could be classified as cases secondary to either Perthes' disease, acetabular dysplasia or epiphysiolysis. Amongst the primary cases, Murray (1973) commented that even very slight anatomical abnormalities could lead to degenerative changes. He suggests that young individuals involved from an early age in sporting activities, could suffer minor, but significant disturbances to growth. The strains of athleticism were seen as giving rise to the later development of osteoarthrosis. An unusual study by Schneider et al. (1962) of the famous Acapulco divers of Mexico showed a number of bone changes in the cervical spine of fractures and other such traumatic damage. This was particularly marked in those individuals who dived with their arms outstretched, absorbibg the shock directly onto the

head. In spite of these injuries, little cervical degenerative joint disease was recorded, suggesting, in contrast to a number of other studies mentioned, that recurrent trauma is not an important agent. The particular nature of the injury incurred is probably emphasized here in its role as a precursor to secondary degeneration. The injuries of the Acapulco divers may have occurred without damaging the integrity of the joint structures.

Studies of secondary cases, thus suggest the importance of metabolic, genetic and mechanical factors.

4.4 Population Studies.

The greatest insights into etiological factors can come from a detailed study of the incidence and distribution of degenerative joint disease naturally occurring in a population or series of populations, that is an epidemiological survey.

There is much disagreement as to whether the areas of the joint that show the earliest signs of degeneration, are the load-bearing (Ekholm, 1956) or non-load-bearing areas (Bullough et al., 1973). Harrison et al. (1953) found that non-use of a joint was a more common cause of degeneration than over-use. Under-use would not give sufficient exercise to a joint to force the nutrient bearing synovial fluid around the joint. This supports the same conclusion discussed above from experimental studies. In addition to the

necessity of metabolites circulating the joint structures adequately, actual disruption of metabolic processes has already been suggested from other types of study. As part of the comprehensive epidemiological study of Leigh and Wensleydale, Kellgren (1961) reports finding above average serum cholesterol levels in individuals with osteoarthrosis. Hormonal influences are proposed as possibly significant in the development of osteoarthrosis, particularly Heberden's nodes, by Stecher (1955). He reports that half of the cases of Heberden's nodes in females occurred within three years of the last menstruation implying a post-menopausal relationship. However, the menopause is associated with a lowered pain threshold, thus the increase in complaints is an artefact of increased susceptibility to pain not an increase in occurrence (Copeman, 1964).

Hereditary factors have been strongly suggested by experimental studies and from cases of secondary involvement. In a comparison of Caucasians and Asians, Byers et al. (1974) noted that osteoarthrosis of the hip is rare in Asians compared to Caucasians but find the patterns of change are the same when it does occur. The Terry Collection in the U.S. is a sample of known age, sex and racial group of white and black Americans. Roche (1957) and Stewart (1947) record slightly less spinal degeneration in negro groups. On the other hand, Jurmain (1977) found a slightly higher incidence of knee osteoarthrosis in the negro group. Incidences in the major weight-bearing joints are more liable to have been affected by cultural differences between various groups

compared. Studies of the hands and Heberden's nodes have provided more concrete evidence of genetic influence. A family study of Heberden's nodes (Stecher, 1955) found a high association of nodes with family group. The results are interpreted as indications of a single gene acting dominantly in females but recessively in males. Different populations show different frequencies of Heberden's nodes, particularly in that a lower frequency is characteristic of negro populations (Bremner et al., 1968). Blumberg et al. (1961) report a lower incidence of osteoarthrosis in the hands and wrists of Eskimos and suggest that individuals with a susceptibility to osteoarthrosis would be selected against in a population living under rigorous environmental conditions such as those of the Eskimo life. A strange, and as yet totally unexplained finding was reported by Bennett and Burch (1968) in a comparison of the incidence of Heberden's nodes amongst the Pima and Blackfoot Indians. The Blackfoot follow the observed pattern elsewhere of a far higher frequency of nodes amongst females than males. The Pima, by contrast, have a greater percentage of males involved. It would appear that some other factor in the Pima is operating amongst males overriding the usual predominance, thought to be genetically controlled, in females.

Studies of multiple joint involvement or generalized osteoarthrosis reveal a greater preponderance amongst females in the studies by Kellgren, Lawrence and colleagues in Northern England. The observed differences in incidence seemed to indicate different processes of osteoarthrotic

progression between the sexes, presumably under genetic control to some extent. However, conflicting evidence again comes from the North American Indians. In a comparative study of incidences between populations of worldwide distribution, Lawrence and Sebo (1980) find the North American Indians have an advantageous resistance to generalized osteoarthrosis compared to other groups. This is particularly marked in the females who do not show the higher incidence of generalized osteoarthrosis characteristic of all other populations. Once again the extensive epidemiological survey carried out in Leigh and Wensleydale presents the most detailed information by analyzing the incidence by family group. The frequency of osteoarthrosis in four or more joints is higher amongst relatives than amongst the general population. This finding could not be accounted for by age relationship nor by occupation. Finally, this survey in Northern England also found an association between osteoarthrosis and rheumatoid arthritis, a disease known to have a genetic component.

Evidence of mechanical factors comes from the pattern of joint involvement in populations. It would be expected that weight-bearing joints should be the most affected. To a certain extent this is seen to be the case but some joints remain universally affected without any obvious mechanical explanation. The study of Leigh and Wensleydale, one of the few studies where a large number of joints were examined, showed great involvement of the fingers which are not weight-bearing joints. However, they may be thought to experience low levels of continual stress from their mobility

and involvement in almost any activity. Other joints greatly affected are those of the lower half of the body, that is the big toe, the knees, the other toe joints, the lower spine and the hips, all of which are subject to weight-bearing. Willis (1924) studied the relationship between body build and the occurrence of degenerative joint disease in the spine. He found that stockier, heavily-boned individuals showed a higher incidence of involvement presumably through greater weight-bearing. Obese individuals are considered to increase the load carried by the weight-bearing joints. The Leigh sample shows a significantly greater involvement of osteoarthrosis in obese individuals of both sexes. This was particularly noticeable in the big toe and the knee. Obese males also showed an increase of degeneration over non-obese in the lumbar spine and the distal interphalangeal finger joints. This last set of affected joints is surprising as they are not weight-bearing. Sokoloff's experiments with mice must be recalled here, where obesity per se was not the causative agent of the exaggerated degeneration, but rather the high fat diet seemed responsible for both obesity and joint degeneration. Obese males in Leigh are also reported to show a greater incidence of multiple joint involvement, which might add support to a systemic effect of a high fat diet (Kellgren, 1961).

The strongest evidence for mechanical involvement in the etiology of degenerative joint disease is the comparison of incidence between different occupational groups with reference to stresses experienced by each. Wickstrom (1983)

compares the frequency of osteoarthrosis of the knees between concrete reinforcement workers and maintenance painters. The concrete reinforcement workers experience greater loads on their knees and minor injuries and similarly manifest more symptons of osteoarthrosis in their knees than the maintenance painters. Roemmich (1962) uses figures from the U.S. social security data on the number of working days lost through various diseases and compares the relative incidences of degenerative joint disease and rheumatoid arthritis in different occupational groups. White collar workers experience twice as much rheumatoid arthritis as degenerative joint disease, skilled and semi-skilled workers have both types equally whereas labourers show one-and-a-half times as much degenerative joint disease as rheumatoid arthritis. In Leigh, Kellgren and Lawrence (1958) report more degenerative joint disease in both miners and cotton-workers compared to the rest of the population. However, different joints were affected. Miners experienced greater osteoarthrosis in their knees and disc degeneration in the lumbar spine. They also showed a trend towards a greater involvement of the hips and metacarpophalangeal joints, although this is not significant. In cotton-workers the small joints of the hands and the cervical spine showed greater involvement. The numbers of the sample in this group were small and only the greater involvement of the first carpometacarpal was significant. Kellgren et al. (1953) also report that tradesmen showed less osteoarthrosis than the general population. Caplan et al. (1966) studied the lumbar spine in a sample from Russelton, Penn. U.S., comparing

occupational groups in terms of strains on the back, heaviness of work and the duration of work. They report increased osteophyte formation with the duration of heavy work, but not necessarily disc narrowing. By contrast, injuries can cause disc narrowing but not necessarily osteophyte formation. The facet joints showed no significant association in involvement with injury, disability or duration of heavy work. They were not seen to be involved at all unless disc narrowing was also present and may represent a secondary form arising from local mechanical stresses generated by degeneration of the disc joints. Lawrence (1969) demonstrated that disc degeneration in the lumbar spine was especially related to heavy lifting and trauma to the back. No association was found with working in stooping positions unless associated with lifting. Bremner et al. (1968) studied a Jamaican population for comparison with the U.K. samples. Some differences in joint involvement were noted. The Jamaicans experienced greater involvement of the cervical spine and of the knees. The proffered explanation calls upon mechanical factors from the different activities indulged in. The Jamaicans carry heavy loads on the head which explains the high cervical involvement from compression, whereas the high values in the knees are seen as resulting from jarring as the footpaths are very rough. This corresponds to the experimental findings of Radin and Paul (1971), that impact loading is particularly important. A similar finding is reported by Sokoloff (1969) in reviewing data from Germany on the use of pneumatic drills. Individuals using equipment with a high frequency vibration suffered little degenerative

change, as opposed to those using machines with a low frequency vibration. The shock from these drills is absorbed in the elbows and shoulders where increased involvement is seen of degenerative joint disease.

Studies of the localization of affected areas within joints give conflicting results as regards the weight-bearing versus the non-weight-bearing involvement. Ekholm (1956) claims the preponderance of osteoarthrosis in the knee occurs in the fibular portion where the greatest load is usually borne. By contrast, Harrison et al. (1953) and Bullough et al. (1973) both report that the important area for the onset of the degenerative process is the non-weight bearing portion. As mentioned in earlier discussions this is seen as degenerating from a result of insufficient exercise needed to pump nutrients around the tissues. It does indeed seem that both too little and too much stress are detrimental.

Within the spinal column both Willis (1924) and Shore (1934a,1934b)draw upon mechanical factors to explain the distribution of osteoarthrosis and osteophytosis in the column. Both suggest degeneration occurs in response to maximum stresses at points of curvature, and as secondary effects in response to degeneration elsewhere in the spine. These will be discussed in far greater detail in the later section on distributions within the vertebral column.

It can be seen that a number of factors contribute to degenerative joint disease. Diet may influence susceptibility

Table 4.1

Etiological Factor	Source of evidence	Author
High fat diet	Experimental - rats	Sokoloff (1960b)
Hormonal	Experimental - injections to rats	Hulth (1969)
Disrupted metabolism	Experimental - injections to rats	Sokoloff (1969)
	injections to hens	Kalbhen (1980)
	secondary to metabolic disorders	Lawrence (1960)
Genetic	experimental - rats	Sokoloff (1960b)
	secondary with genetic disorders	Lawrence (1960)
	Heberdens nodes	Stecher (1961)
	Generalized Osteoarthrosis	Kellgren, Lawrence et al.
	Population differences	Bremner et al. (1965) Blumberg et al. (1961)
Injury	Experimental	Williams et al. (1982)
	secondary	Murray (1973) Kellgren and Lawrence (1958)
Disuse	experimental population; pattern of joints and within joints	Sokoloff (1969) Harrison et al. (1953) Bullough et al. (1973)
Compression	Inter-specific comparative	Schultz (1956) Fox (1939)
	Weight-bearing: body build	Willis (1924)
	obesity	Kellgren (1961)
	occupation	Wickstrom (1983) Kellgren and Lawrence (1958)
Impact	experimental	Radin and Paul (1971)
	population activity differences	Bremner et al. (1968)
	occupation	Sokoloff (1969)

if high in fat content. Some evidence of hormonal influences are given but these appear not to be very prominent. Vascular changes can be seen, but these appear to represent part of the degenerative process rather than a causative agent. Genetic predisposition is an important agent, particularly in Heberden's nodes and possibly in generalized osteoarthrosis. Injuries and anatomical abnormalities can be precursors to degeneration of the integrity of the joint is disturbed. Similarly, metabolic disorders can lead to degeneration if the metabolic processes of the joint itself are affected. The most important single factor is seen to be mechanical stresses on the joints. Compression is important, particularly if acting in repeated impact. Under-use can be equally important. Both compression and under-use inhibit the free circulation of metabolites for the continuing nutrition of the joint tissues, causing necrosis and subsequent degeneration. The studies are summarized in Table 4.1.

4.5 History of Degenerative Joint Disease and Archaeological Studies.

Lesions comparable to those of degenerative joint disease in modern humans have been recorded from the mesozoic dinosaurs through to Neanderthal man. The extensive studies of Moodie (1880) on examples in the American museums provide detailed descriptions of observed pathological lesions. Notable is the section of the tail of *Diplodocus longus* which shows osteophytic growths in the area where it touches and drags on the ground. Similar lesions were also observed in

the tail of Cetiosaurus, an English dinosaur. Parts of the spine, other than the resting part of the tail, have also shown osteophytic involvement. Moodie (1880) reports a case of cervical involvement in Camarasaurus, Diplodocus and Tyrannosaurus. Possible osteoarthrotic lipping is reported, together with other abnormalities, in the foot bones of the mosasaur, Platecarpus from Kansas. This multiple involvement of the foot bones, with abnormal bone form generally, suggests a case of secondary degenerative joint disease in conjunction with another disorder (Moodie, 1880).

Degenerative joint disease is thereafter found widely. Moodie (1880) reports cases in the Eocene mammals, Limnocyon and Pantolambda, in a miocene crocodile, Tomistoma dowsonii, from Egypt, a pliocene camel from Nebraska and a pleistocene sabre-toothed tiger, Smilodon. Other cases from the pleistocene are well-documented by other authors such as Virchow, Von Walther, Esper and Cuvier. (see Snorrason(1942) and Wells (1964)). These involve cave bears, cave lions, cave hyaenas, reindeer, and sabre-toothed tigers. Most of the reports involve cases of osteophytosis, although the pleistocene mammals are reported to have some involvement of the extremities. The cases of modern animals affected have already been discussed under the section on inter-specific comparative studies. It is recognized that degenerative joint disease is not exclusive to humans and seems to have existed as long as have vertebrate joints.

The human history of degenerative joint disease could

possibly aid towards the illumination of etiological factors. Populations living under circumstances very different from present day often present a far more homogeneous group in terms of dietary and activity habits and provide a greater range of natural situations from which to study the occurrence of degenerative joint disease. The majority of such samples are small in number and have only limited background information on cultural practises, at best only gross details of diet and subsistence patterns. Patterns of degenerative joint disease are more often studied by physical anthropologists as a source of information on the activity habits of the skeletal collection. In such studies, the etiological agents are simplified to a mechanical one. This is not done in ignorance of other possible agents but highlights the general acceptance that wear and tear is likely to prove the most useful single factor explaining variations in population incidences of the disease.

The infamous reconstruction of Neanderthal man's gait by Boule, based on the La-Chapelle-aux-Saints specimen, illustrates the presence of degenerative joint disease in Neanderthals. The re-examination by Straus and Cave (1957) make clear the severe nature of the disease in this specimen particularly in the spine. The affected portion was the joints between the fifth cervical vertebra and the third thoracic. The lower lumbar region was also affected to a lesser extent. Other Neanderthal specimens show similar involvement of both osteoarthrosis and osteophytosis. Wells (1964) points out the common involvement of the temporo-

mandibular joint, for example in Krapina, La Ferrassie, La Quina and La Chappelle-aux-Saints. He suggests this represents evidence that the Neanderthals ate a tough, perhaps uncooked diet of roots and nuts, thus putting the jaws to hard use.

Remains from Egypt have been well studied, particularly by Ruffer (1918). He studied mummies from pre-dynastic through to the start of the Christian era, a six-thousand year span. The prevalence of osteophytosis was particularly noticeable through all periods. Fifty percent showed some involvement after the age of 25 and older individuals would display severe cases, often even fused. Wells (1964) reports that the most involved region of the spine was the thoracic spine, whereas Bourke (1971) in a study of 132 individuals from Hou (dated 1500B.C.) observed two peaks of severe involvement. Osteophytosis was most marked in the lumbar region with a second peak in the cervical region. The thoracic region did not feature prominently contrasting strongly with Wells' (1964) report. A possible explanation of extensive osteophytosis in the cervical region could have resulted from the custom of carrying loads on the head (MacAdam, pers. comm.)

Other examples of outstanding involvement of particular joints are reviewed by Wells (1964) and Snorrason (1942). Macedonian remains of soldiers, presumed followers of Alexander and Ptolemy I, show abundant degenerative joint disease in the foot, a site not afflicted in the Egyptian samples from Egypt. A suggestion has been that this resulted

from heavy marching in clumsy footwear carrying heavy equipment over rough terrain. Skeletons of early Patagonians, who were great horseriders show noticeable involvement of the shoulders and elbows, supposed to arise from the use of the bola, a hunting weapon that hurls stones. The rotational movements necessary to use this on horseback concentrates the strain on the joints of the upper limbs. West and South African remains have shown lipping of the small wrist bones and the base of the thumb, particularly in the right hand. The use of hoe and mattock for preparing the soil which has baked hard under the sun, causes a jarring or concussion in the wrist region. New Caledonians, eating a hard, coarse diet, developed much degenerative joint disease of the temporomandibular joint.

Wells (1964) states that the incidence of degenerative joint disease is lower in populations where the overall stress of life has eased. Work by Angel (1979), on a series of Turkish and Byzantian populations, was not able to record an evident decline in the incidence of degenerative joint disease from ancient through to modern times. Wells (1964), however, observes a decrease in incidence in Ancient Greece from the level in the Neolithic and Bronze age. Farming practices had been basic and provided merely a subsistence crop up to the later Bronze age. Around this time, irrigation and crop rotation was introduced yielding a higher output, an improvement in nutrition, increases in stature, longevity and populations size and a decrease in degenerative joint disease. The view of Wells (1964) that it is a decrease in

the overall stress of life that has occurred causing the decrease in incidence and severity of degenerative joint disease, is not supported generally. Rather, it is the specific stresses, particularly the mechanical ones, leading to a degeneration which have declined. This may often accompany other decreases of stresses but is not an automatic partner. In a study of remains throughout the 1st to 17th centuries in Bulgaria, the frequency of involvement of the spine remained relatively constant except for the period betweeen the 15th and 17th centuries where the frequency increased (Goranov et al., 1983). The authors depict this period as one of great inequalities where the majority were forced to undertake hard physical labour and slavery was common. This presents a picture of increased degeneration with increased physical activity rather than a lack of technological skills.

Two detailed studies of spinal degenerative joint disease have been carried out on the late mediaeval population from Aebelholt, Denmark (Sager, 1969; Ingelmark et al., 1959). Specific details will be reviewed in the sections on the spine and on spine and age-related changes. Ingelmark et al. (1959) note a curious association between dental caries and degenerative joint disease in the spine. The relationship had previously been reported amongst Peruvians by Moodie (in Wells, 1964). The explanation for an association between infectious lesions of the teeth and non-infectious lesions of the spine is not at all understood. As seen already, the etiology of degenerative joint disease is imperfectly understood and very many factors are involved.

Although the field of palaeopathology is expanding with the prospect of many publications in the near future, at present the majority of reports on British material consists of the pioneering work of Wells on Anglo-Saxon remains. One earlier publication is a description of a case of osteoarthrosis of the hip in a Bronze Age skeleton (Pitt-Rivers, 1965). However, Wells states that on the whole the Bronze Age peoples showed little improvement by contrast to the Anglo-Saxons. Wells (1963) examined remains from the 6th to 11th centuries and found that most adults over twenty-five would display some degeneration. The most commonly affected region was the spine. Commonest sites amongst the limb bones were the elbow, knee and foot. Wells (1965) contrasts the subsistence activities of the Anglo-Saxons with those of the earlier Bronze Age peoples. The Bronze Age inhabitants were mainly pastoralists, able to spend most of the time accompanying the herds at a leisurely pace. Agriculturalists, often immigrants having to break new land or clear woodland are involved in much heavier work. The Anglo-Saxons are also thought to have been taller and more robust in build than their Bronze Age predecessors (Wells, 1965). The combined factors of heavy agricultural labour and larger size would place far greater mechanical demands upon the Anglo-Saxon joints than those of the Bronze Age. This is further borne out by the observed higher incidence amongst Anglo-Saxon males than females, presumably as a result of division of labour (Wells, 1972).

A more recent publication comes from Rogers et al.

(1981) who have studied a mixed collection of skeletons from Saxon, Romano-British and Mediaeval sites. They found a particularly high frequency of osteoarthrosis in the shoulder of Anglo-Saxons, a joint not commonly affected in modern samples. They suggest the possibility of a different disease process amongst Anglo-Saxons, involving excessive bone growth at the joints in general. Mediaeval skeletons also had frequent involvement of the spine compared to the Anglo- Saxons and common involvement of the hip and shoulder but less than the Anglo-Saxons.

A greater number of publications are already in print from American investigations of palaeopathology.

A comparative study of three American groups was made by Jurmain (1975) on the Terry Collection, a modern 20th century sample of white and black Americans, of both sexes and known age, plus two archaeological samples. These were a group of protohistoric Alaskan Eskimos and the 12th century Pecos Pueblo Indian Collection. The four large joints were graded for presence of osteoarthrosis, that is the knees, hips, elbow and shoulder. The Eskimo group were found to have noticeably more osteoarthrosis of both knee and elbow than the other populations. The Pecos Pueblo Indians had the least knee involvement but had more elbow involvement than the modern sample (1977, 1980). All populations showed more involvement of the right knee over the left. The patellar of the knee showed changes that were more age-related than the femur or tibia portions of the knee joints, especially in the

archaeological samples. Jurmain discusses the observed differences in terms of known activity habits, particularly in explaining the high incidences in the Eskimo population. The vigorous lifestyle necessary to survive in the harsh conditions of the Artic has involved a number of cultural activities unique to Eskimos. Sled driving, ice prods, boat rowing, harpooning and lancing are all given as examples of activities stressing the elbow. Crouching positions put a great deal of stress on the patellar portion of the knee joint, and the figures implying less stress on the patella in archaeological samples is explained in the Eskimos at least by their habit of bending at the waist rather than crouching. They still, however, experience a much higher level of knee involvement overall, again presumably related to activities such as sled driving and kayak/canoe use.

Stewart (1947) compares almost the same populations for spinal degenerative joint disease. Variations between the three groups are reported for both in the pattern of involvement in the spine. The Eskimos, by contrast to the major joints have very little osteophytosis except in the lumbar area, but they do exhibit much more involvement of the facets peaking in the mid-cervical and lower thoracic regions. The Pueblo Indians have a little more osteophytosis on all regions than the Eskimos, but few facets are involved peaking in the upper thoracic region. The whites have the most osteophytosis, peaking in the lower cervical, followed in order by the lumbar and lower thoracic regions. Facet involvment is very little and falls mainly in the mid-cervical

and upper thoracic regions. The Eskimos, therefore appear to have the greatest involvement of the synovial joints amongst the three groups but the lowest level of osteophytosis . Indeed the relationship of osteophytosis to osteoarthrosis seems to be an inverse one from Stewart's descriptions. Stewart acknowledges that the different patterns of involvement are most probably due to cultural differences, but he does also point out that there are racial variations in the anatomical structures of the spine which could affect mobility. Roche (1957) also has examined the Terry Collection for spinal degenerative joint disease and compares black and white American subpopulations. He finds in both there is more osteophytosis than osteoarthrosis, the females show more involvement generally than males, but if broken down into age categories the males will catch up and overtake females in the older age groups. In all age groups and both sexes whites have a higher incidence than blacks.

Studies of early American Indian groups have looked at patterns of joint involvement in a similar fashion to Jurmain (1977). Pickering (1979) has studied skeletons from the Ledders Site of the late Woodland period, where the subsistence pattern was hunting and gathering. Females showed an earlier age of onset, but males had a higher incidence later with more severe forms. The females suffered in the elbow and thumb, whereas the males suffered more from the wrist. Drawing analogies with the contemporary !Kung groups, Pickering observes that females will enter the labour force earlier than males, thus explaining the earlier onset age.

The division of labour between the sexes of hunting males and gathering females could explain the different patterns of involvement between the two. Carrying burdens and the use of tools such as digging sticks would lead to greater mechanical stresses on elbows whereas hunting tools put more stress on the wrist action. Martin et al. (1979) have studied osteoarthrosis in the long bones of the Dickson Mounds population, while Clark and Delmond (1979) studied the lumbar spine. In the lumbar spine, more males were affected than females, but no further details of subgroups by time or age is given. The long bones studied divide the sample into two groups, although three cultural horizons have been identified. The first group, late woodland-transitional dated 1050 - 1200 A.D., subsist by hunting and gathering mainly with a little farming in later periods. The second group, middle Mississippian dated 1200 - 1300 A.D. had a predominantly maize diet and lived a more sedentary life in denser groups. Both groups had a similar age of onset around 30 - 35 and have similar incidences of involvement in subsequent age groups. However, in the patterns of joints involved the middle Mississippian group have a far greater involvement of the wrist suggested as a result of the use of tools in agriculture and maize preparation. An interesting additional point was the observed association between multiple joint involvement and infectious diseases with more severe cases of both also associated. They recognize the potential here for interesting further research into the interplay and possible cultural factors involved.

The difficulties of a comparative study between archaeological populations is illustrated by Chapman's studies of osteophytosis (1968,1965,1972). She examines the presence and severity of osteophytosis in fifteen skeletal collections from a range of geographical sites in the Americas, covering a span of time periods and having different subsistence activities. First, most of the samples are so small in number, statistical analyses are not possible and a purely descriptive comparison has to be made. Secondly, the range of variables involved in this ambitious undertaking are so many, as Chapman herself points out, with precise information on many of them lacking, that it is impossible to decipher any observed population differences in terms of possible agents. Indeed, no consistent patterns emerge presumably because where one subsistence activity group may experience greater mechanical stress, a genetic susceptibility amongst others, or climatic or dietary influences etc. would cloud the emerging pattern. The lack of background information on the necessary details makes a broad comparison of this nature of very low information value. Chapman has produced two papers (1965, 1972) where she takes just a few of the populations for comparison. This is more useful for some of the variables can be eliminated. Chapman (1972) compares the Mexican groups, based at high altitudes and covering hunter/gatherer and agriculture subsistence patterns. Both groups show an early age of onset compared to the North American group from the middle Mississippian site of Dickson Mounds, to which she compares them. They also show a greater degree of involvement than the Dickson Mounds

group. A Mexican group from a lower altitude is added in for comparison and shows similar high frequencies. Amongst the Mexican groups therefore, subsistence activity does not appear to be causing noticeably different patterns, nor altitude. The other paper (Chapman, 1965) compares two Arctic Eskimo groups with a Midwestern group from Modoc shelter. In all groups males have more severe involvement than females by the older age groups. However, the two Eskimo groups have later ages of onset than the Midwestern group. The Eskimos have a high fat and protein intake compared to the Midwesterners which from Sokoloff's experiments with rats, may have lead to an expectation of a higher suseeptibility to osteophytosis, yet here they show a lower incidence. The possiblity of important genetic influences being in operation is discussed by Chapman (1965). Chapman's recording of lower osteophytic incidence amongst Eskimos is consistent with the reports of the same by Stewart (1947). However, he found a high incidence in the synovial facet joints, and Jurmain (1975) reported a high incidence in the large synovial joints. So it does appear that the two kinds of joints may undergo different processes of degenerative change.

Overall, the work on archaeological samples shows useful discussions of etiology can be provoked from observations if good sample sizes are available and the number of environmental variables limited in comparative studies. Use of degenerative joint disease incidences has also been useful to shed light on lifestyles of prehistorical populations where very little is known.

4.6 Studies of Degenerative Joint Disease in the Spine.

It has been proposed that degenerative joint disease of the spine is a disease particularly besetting humans as a result of their upright posture (Thieme, 1950). As discussed in the preceding section, degenerative joint disease has been observed in skeletons from dinosaurs to modern humans. A great majority of the observations on the now extinct dinosaurs and mammals involved the joints of the spine. Bohatirchuk (1955, 1957) compares age-related changes of degenerative joint disease in humans and dogs and finds that dogs undergo similar degenerative changes to humans in the spine. He comments that the case for a particularly human susceptibility cannot be maintained. The studied dogs showed a peak of incidence in the lumbar spine, similar to a modern human population, in spite of very different mechanical demands on the columns of the two species. The inter-specific comparative study of Fox (1939) also discussed the involvement of the spine in many non-human species. There appears, therefore, to be no case for spinal degenerative joint disease to be seen as a specific scourge of humans.

Mechanical factors are commonly seen as largely responsible for patterns of distribution of degenerative joint disease through the spine. Willis (1924), only examined the lumbar disc joints in 625 skeletons from the Harmann Museum and records the highest incidence of osteophytosis in the lower lumbar segments. These are thought

to be under the greatest strain from the curvature of the spine. Increased osteophytosis is also found at these sites if the lumbar curve is exaggerated. Clark and Delmond (1979) similarly studied osteophytosis in the lumbar spine in relation to the degree of wedging of the vertebral units. They examined ninety skeletons from the Dickson Mound sample. Wedging represents the difference between the anterior and posterior heights of the vertebral bodies (P - A). A positive wedge (P>A) is a measure of kyphosis and a negative wedge (P<A), a measure of lordosis. Clark and Delmond (1979) find males have a greater degree of kyphosis than females in the lumbar spine and that this increases with age in both sexes but relatively more so in males. The fourth lumbar vertebra is a transitional point between the kyphotic curve of the upper lumbar region and the lordotic one of the fifth lumbar. As a point of contraflexure, therefore, the fourth lumbar is expected to be subject to greater stresses than elsewhere in the region and the authors do find the greatest amount of osteophytosis on this segment amongst males (only males were examined for the presence of osteophytes.). Lindblom (1951) compares a hundred prehistoric Swedish skeletons with two thousand present day Swedes, again studying only osteophytosis of the lumbar region. Whereas the prehistoric population shows the highest frequency in the mid-lumbar area at L3, the modern population peaked at L5. Neither of these two fit with Clark and Delmond's (1979) predictions or own findings. However, none of the other studies on the spine have studied the association of degeneration with measurements. It is possible that different

points of contraflexure may exist. The occurrence of the greatest incidence on the fifth lumbar suggests that stresses on the lordotic curve are greater in the modern sample than on the kypholic or point of the contraflexure. The study of Caplan et al. (1966) of lumbar osteophytosis amongst modern miners, also indicates that L5 is the most commonly affected. It seems that a shift in stresses has occurred from earlier populations to modern such that maximum lumbar involvement has moved down the spine from L3/L4 to L5.

In studies of the whole spinal column, Bourke (1971) reports that amongst ancient Egyptians osteophytosis is both most common and most severe in the lumbar region, but he is, unfortunately, no more specific than this. Nathan (1962) has made a very detailed study of osteophytosis in the spinal column of four hundred modern specimens, mainly from the Todd Collection comprising both black and white Americans. The most affected joint is the tenth thoracic, but peaks in the other two regions are seen also, at the fifth cervical and the fourth lumbar joints. This pattern of three peaks, one in each region is found in all studies of the total column for both osteophytosis and osteoarthrosis. Nathan. like Clark and Delmond (1979) and Willis (1924), relates the pattern to the curvatures of the spine. The basic stresses of the spine's curve and associated lipping can therefore be regarded as the normal pattern of osteophytosis. Variation in the relative incidences and severity of the three regional peaks will occur with cultural activities, but deviations from a three peak pattern would demand detailed

study and explanation of an unusual or pathological process.

Three studies exist on distribution throughout the spine of degenerative joint disease of both facet and disc joints. Stewart (1947) examined 177 Eskimo, 83 Pueblo and 104 white Americans from the Terry Collection. The distributions are only generally given but serve to compare the populations and involvement of the two kinds of joint. The Eskimo show maximum osteophytosis in the lumbar region, whereas white Americans show the most in the lower cervical region, then in the lumbar and last in the lower thoracic portion. The Pueblo do not have a greater prevalence in any one region. By contrast osteoarthrosis shows a different distribution of maximum points. In Eskimos the maximum region is the mid-cervical, as it is also in the white Americans, but the Pueblo show the most involvement in the upper thoracic region. This region figures as the second most affected portion in the white spine, but in Eskimos the lower thoracic is more commonly involved. The lumbar region is not particularly affected compared to the other regions in any of the groups. Detailed studies have been made by Shore (1934a, 1934b) on 126 skeletons from various sources and Ingelmark et al. (1959) on 211 skeletons from the late-mediaeval site of Aebelholt. Shore (1934a) reports three outcrops of osteophytosis in the spinal column with maximum and minimum points in each region. The lumbar region shows the greatest involvement, then the thoracic and lastly the cervical. The cervical outcrop has a peak incidence of osteophytosis at C4 - 5 with a minimum point at the cervical-

thoracic junction. The thoracic outcrop peaks fairly low down the region at T8 - 10 followed by a region of decrease at the thoracic-lumbar junction. Finally, the lumbar outcrop peaks at L2 - 4 and again falls off at the lumbar-sacral junction. In the facet joint a similar pattern of three regional peaks is found but which shows differences in detail from the disc joints. The lumbar region again is the most involved with a peak at L2 - 3. The thoracic outcrop is slightly irregular with two peaks and a small decrease between them at C7 - T1 and T4 - 5. This denotes a higher maximum point than that of the thoracic outcrop of osteophytosis. Indeed, the minimum point of facet involvement between the thoracic and lumbar outcrops is at T8 - 9, the same point as the maximum osteophyte thoracic involvement. Lastly, the cervical outcrop peaks at C3 - 4 with a minimum point at C6 - 7. The atlanto-axial joint shows the least involvement of the entire spine. Shore's (1934a) distribution pattern of osteophytosis corresponds well with that of Nathan (1962) and similarly adopts a bio-mechanical explanation for the outcrops. Osteophytosis therefore is seen as occurring most prominently at the points of contraflexure of the spinal curves with the minimum points found at the tops and bottoms of the various curves. (Nathan, 1962; Shore, 1934a; Clark and Delmond, 1979). Shore (1934a) assumes the lumbar region is the region affected first since it is the site of greatest involvement for both facet and disc joints. Osteoarthrosis of lumbar facet joints would accentuate the lordotic curve of the lumbar spine and thereby throw the thoracic facet joints back upon the lumbar spine such that

they become incorporated with the lumbar curve. Shore (1934b) proposes this lumbarization process as an explanation for the greater spread of the lumbar outcrop beyond the original lumbar curve. The thoracic outcrop of osteoarthrosis is seen as a result of action of upper limbs and movement from respiration. The cervical outcrops of both facets and discs result from the stresses of the lordotic curve, the same principle as explains the lumbar involvement. Such bio-mechanical analyses seem the most fruitful line of speculation to follow. Unfortunately, Shore does not discuss the distribution patterns of the two kinds of joint together. The exaggeration of lordosis which explains the spread of the lumbar outcrop of osteoarthrosis does not appear to affect the disc joints in a similar manner. The breathing actions of the upper thoracic region of the body are presumed not to affect the disc joints but do affect the facet joints. No explanation is offered for these differences. Ingelmark et al. (1959) records an even more striking difference between the facet and disc joints. The peaks of osteophytosis and related low zones correspond reasonably well with Shore (1934a) and Nathan (1962) except for the cervical outcrop which peaks somewhat lower at C6 - 7. However, the Danish facet joints show no lumbar outcrop at all, but drop right away in incidence after the thoracic peak at T4 - 5. This is thought to be most unusual and demands some discussion. Unfortunately, none is given (Ingelmark et al., 1959). Discussion is made, instead, on the relationship between the two kinds of joint. The authors feel the evidence suggests that the degeneration in each joint type proceeds independently of one another.

Stewart (1947) also comments on the lack of relationship in involvement of facets and disc joints. It could be equally suggested that the two do show a relationship, but an inverse one, recalling the correspondence of maximum disc and minimum facet involvement of Shore (1934a, 1934b) in the thoracic outcrop.

Roche (1957) using the Terry Collection of both black and white Americans reports an overall incidence of osteophytosis which is higher than that of osteoarthrosis in the spine. This was the case for both sexes and both racial groups. Only 3% of the spines displayed osteoarthrosis without osteophytosis, whereas 25% had osteophytosis without osteoarthrosis. This 25%, however, did have less severe cases of osteophytosis than those individuals showing osteoarthrotic lesions as well. Osteophytosis appeared in earlier age groups than osteoarthrosis. Roche (1957) proposes a causal relationship between the two such that osteophytosis is a pre-requisite for osteoarthrosis. Goranov et al. (1983), studying Bulgarian remains, also report a greater incidence of osteophytosis than osteoarthrosis but do not detail the relative ages of onset. Ingelmark et al. (1959) find that osteoarthrosis shows the earlier age of onset, an equal level of involvement of both joints in adulthood and a greater incidence of osteophytosis in the older individuals. Ingelmark et al. (1959) therefore present results which support other researchers' conclusions that osteophytosis is more prevalent overall, but negates Roches' (1957) hypothesis that osteophytosis necessarily precedes

osteoarthrosis.

The localization of osteophytes on the superior or inferior margins of a joint has been examined by Nathan (1962) and Ingelmark et al. (1959). Terminology in examining the papers has to be read carefully as the superior and inferior surfaces referred to by Nathan (1962) apply to those of the osteological unit, that is the vertebral body, whereas the superior and inferior surfaces of Ingelmark et al. (1959) refer to the anatomical unit, that is the joint and are therefore the opposite of Nathan's. Here the usage of Ingelmark et al. is followed in referring to the joint, not the bone unit.

Ingelmark et al. (1959) find a preponderance of involvement in the cervical and upper thoracic regions of the superior surface. No particular differences are found in the mid-thoracic and lumbar regions display a greater involvement of the inferior surface. Nathan (1962) finds a more specific region by region change. The cervical region has greater superior surface involvement up to C6 - 7 after which the inferior joint surface predominates. The superior surface again displays the greater incidence between T2 - 3 to T10 - 11 after which the inferior emerges more strongly. In the lumbar region L1 - 2 has a superior dominance and L3 - 5 shows greater inferior osteophytes. Ingelmark et al. (1959) interpret their simpler pattern of distribution of surface localization as related to the curvature and alterations caused by degenerative joint disease. In osteophytosis the

cervical curve is altered, particularly in the lower cervical spine, from a lordotic one to a kyphotic curve making a continuum with the kyphotic curve of the upper thoracic spine. The lower thoracic similarly becomes incorporated into the lordotic curve of the lumbar spine. This is the same alteration of curvature from degenerative joint disease proposed as an explanation by Shore (1934b) for facet joints distributions. Thus, Ingelmark et al. (1959) claim that the superior joint surface is affected in a kyphotic curve and the lower surface in a lordotic one. Nathan's (1962) explanation is equally related to the curvature but is not related to the curve's direction. He claims the pattern of surface localizations follows the peaks of curvature; above the peaks or points of contraflexure, the superior joint surface predominates whereas below it the inferior joint surfaces are more involved. This explanation does not presume that some degree of osteophytosis will have occurred before the localization pattern emerges.

Nathan (1962) also examines the presence of asymmetry in osteophytosis of the spine. It is common to find a predominance of osteophytosis on the right hand side of the thoracic spine. Nathan (1962) records this from T5, peaking at T10, whereafter the left hand side osteophytes begin to increase in frequency until the asymmetry disappears in the lumbar region. It has been proposed that this reflects the predominance of right-handedness. Nathan (1962) points out that as the proportion of left-handed individuals is around 10% in the population, then the incidence of those with left

hand side asymmetry of osteophytes should also be around 10%, but this is not at all observed. Culver and Pirson (1956) also point out that X-rays of individuals known to be left-handed still show the predominance of osteophytosis on the right hand side. This really negates the handedness hypothesis. A more plausible explanation is offered by Culver and Pirson (1956) who make the observation that the absence of osteophytes corresponds to the section of the spine where the aorta is in apposition to the left hand side of the vertebral column. They suggest the presence of the pulsating aorta somehow inhibits bone growth along this portion of the spine. Nathan (1962) similarly describes the aorta as placed exactly where there was the greatest asymmetry of osteophytosis, from a small sample of dissection-room cadavers. Finally, Nathan (1962) cites a case presented by Shapiro and Batt where the aorta descended abnormally on the right hand side and osteophytosis occurred predominantly on the left.

A summary of maximum and minimum peaks of incidence of degenerative joint disease, localization by joint surface and side asymmetry is given in Tables 4.2 and 4.3. The explanations for the specific patterns of distribution of the disease within the spine itself all draw upon biomechanical explanations. Population differences are found which are presumed to reflect the different activities in which different groups are engaged.

Table 4.2 Summary of Research on Distribution through the Spine of Degenerative Joint Disease.

Researcher	Sample	Peak of Incidence
A. Lumbar: Osteophytosis		
Willis (1924)	625, Haman Museum	"where lumbar curve most marked"
Clark and Delmond (1979)	90, Dickson Mounds	L4
Lindblom (1951)	100, prehistoric Swedes	L3/4
	2000, modern Swedes	L5
Caplan et al. (1966)	178, U.S. miners	L5
B. Entire spine osteophytosis		
Nathan (1962)	400, Todd Collection	C5 - 6; T10; L4
Bourke (1971)	132+, Egyptians	lumbar region
Stewart (1947)	177, Eskimo	lumbar
	83, Pueblo	(no region especially greater)
	104, Terry Collection	lower cervical/ lumbar/ lower thoracic
Shore (1934a)	126, mixed	C4 - 5; T8 - 10; L2 - 4
Ingelmark et al. (1959)	211, Aebelholt, danish	C6 - 7; T8 - 9; L2 - 3
C. Entire spine: osteoarthrosis		
Stewart (1947)	177, Eskimo	Mid-cervical / lower thoracic
	83, Pueblo	Upper thoracic
	104, Terry Collection	Mid-cervical/ upper thoracic
Shore (1934b)	126, mixed	C3 - 4; C7 - T1; T4 - 5; L2 - 3
Ingelmark et al. (1959)	211, Aebelholt	C3 - 4; T4 - 5

Table 4.3 Summary of Surface Localization and Asymmetry of Osteophytosis.

Researcher	Sample	Result
A. Surface localization		
Ingelmark et al. (1959)	211, Aebelholt	Cervical and Upper Thoracic = superior predominant Mid-thoracic = both surfaces same Lower thoracic and lumbar = inferior predominant
Nathan (1962)	400, Todd Collection	Cervical: C1 to C6 superior predominant C7 to T2 inferior predominant Thoracic: T2 - T10 superior predominant T10 - T12 inferior predominant Lumbar: L1 - L2 superior predominant L2 - L5 inferior predominant
B. Side Asymmetry		
Nathan (1962)	400, Todd Collection	Right predominates: T5 - L1

4.7 Incidence of Degenerative Joint Disease by Age and Sex

It has been shown in the review of etiological factors that wear and tear from mechanical demand emerges recurrently as a highly significant agent. The one other equally important single factor in the incidence of the disease is age, hence the name degenerative joint disease. It has already been pointed out in the previous chapter that certain changes occur in the tissues and bone of the joint concomitantly with age which may reduce the resistance of the joint structures to stresses. In addition, insults to the joint from wear and tear accumulate with age increasing both the likelihood and severity of degenerative joint disease. Since the relationship of the disease to age and its possible use as a means of establishing age at death from skeletal remains is the theme of the thesis, the review of incidence of degenerative joint disease with age and sex at population level is presented here as a separate section from the other results of population studies. A large number of survey studies, involving adequate sample sizes for breaking down into age and sex groups for analyses, have been made on the hands and feet. As mentioned earlier, Heberden's nodes represents a particular form of degenerative joint disease which appears to have a stronger genetic determinant than the other forms, particularly affecting the incidence by sex. The results, therefore, are not necessarily indicative of trends in degenerative joint disease in general. The particular aspects of age-related change which are of importance are the rate of increase in incidence, the

progression of severity, the number of joints affected and sex differences observed.

Stecher (1940) examined 6913 subjects of both sexes of negro and white racial groups. The incidence of Heberden's nodes increased with age in all subgroups but the rate of increase varied being fastest amongst white females and lowest amongst the black females with the males of both racial groups in between. The vast survey of non-institutionalized civilians in America involving 6672 individuals between 18 and 79 is analyzed by Gordon (1968) and Roberts and Burch (1966). The overall figures for both sexes depicts an increase in incidence from 4% in the 18 - 24 age group up to 85% in the 75 - 79 age group. The two sexes analyzed separately show different rates of increase. The males have a higher incidence than females up to 45, by as much as two times. However, the females catch up and show a higher incidence than males after the age of 55. Tzonchev (1968) examined a large number of joints in a Bulgarian series and also reports a greater incidence in females over males by the oldest age group. The study of the Pima and Blackfoot Indians shows a higher age-specific rate of involvement than the U.S. sample, but have differing incidences by sex. The Blackfoot also show a higher incidence in females by the oldest age group in keeping with other studies, but the Pima curiously show the reverse trend (Bennett and Burch, 1968). The reversal of the usual distributions by sex in this group remains a mystery. Eskimo populations, studied by Blumberg et al. (1961) display a lower incidence of osteoarthrosis of the hands and wrist at

all ages compared to the U.S. sample. There was a slight tendency for females to show an earlier age of onset of Heberden's node, but the sample size was too small for statistical analysis. The females also showed a higher incidence than males in the oldest age group.

The progression of severity of osteoarthrosis of hands and feet with age was examined in the large U.S. sample (Roberts and Burch, 1966). Under 45 nearly all cases were mild. After 45 the frequency of moderate and severe cases increased until they were as common as mild cases by 75. Mild cases reach a frequency peak in males at 65 - 74 whereas the female incidence peaks about ten years earlier at 55 - 64. Females show a higher incidence of moderate and severe forms of osteoarthrosis, a sex difference which is particularly marked after 45. Laine (1962), working with Finnish country populations, found a slightly higher incidence of moderate and severe cases amongst females of all ages. A further detailed breakdown by age is not provided.

Finally, the U.S. survey of osteoarthrosis of the hands and feet found an increase of multiple joint involvement with age in both sexes (Roberts and Burch, 1966).

The broad studies of Kellgren, Lawrence and their co-workers have provided vast amounts of detail on incidence, severity and multiple joint involvement over many different joints, not just the hands and feet. The two populations of Leigh, in South Lancashire and Wensleydale display age-

related increases in incidence that are more or less identical to one another. Leigh had an incidence of degenerative joint disease of 8% in males and 7% in females in the 15 - 24 age group, which increased to 96% in males and 99% in females by 65+. Wensleydale had incidences in the 15 - 24 age group of 10% and 8% for males and females respectively, which increase up to 99% in males and 96% in females by 65+ (Lawrence, 1963). The age-specific rate is slightly higher than that of the U.S. sample figures for hand and foot involvement. The proportion of individuals showing some involvement of degenerative joint disease was not significantly different between the sexes (Kellgren and Lawrence, 1958). Bremner et al. (1968) studied 536 subjects from a Jamaican population. The prevalence of osteoarthrosis was found to be similar to the U.K. samples.

The incidence of severity grades 3 - 4 of Kellgren and Lawrence were found to be slightly more prevalent in males up to 55 and in females thereafter (Lawrence et al., 1966). The Jamaican population showed a slightly lower level of severity than the U.K. sample, although this was not significant (Bremner et al., 1968).

The number of joints affected in the Northern England samples increases with age and females are far more frequently afflicted by multiple site involvement than the males (Lawrence et al., 1966). A summary of the findings from present day studies are presented in Table 4.4.

Table 4.4 Summary of Incidence, Severity and Multiple Joint Involvement in Modern Day Populations with Age.

Researcher	Sample	Result
A. Hands and Feet		
Incidence:		
Stecher (1940)	6913, U.S. black and white	white females > black / white males > black females
Roberts and Burch (1966)	6672, U.S.	males > females until 45 females > males 55+
Tzonchev (1968)	Bulgarians	females > males oldest age group
Bennett and Burch (1968)	N. American Indians	Indians rate > U.S. Blackfoot females > males oldest age group Pima males > females oldest age group
Blumberg et al. (1961)	Eskimo	Eskimo < U.S. females onset before males females > males oldest age group
Severity:		
Roberts and Burch (1966)	6672, U.S.	all mild up to 45 moderate/ severe increase 45+ moderate/ severe frequency = mild 75
Laine (1962)	Finnish	female moderate/ severe > males 45+ females > males
Multiple:		
Roberts and Burch (1966)	6672, U.S.	increase with age.

Table 4.4 contd.

Researcher	Sample	Result
B. All joints		
Incidence:		
Kellgren and Lawrence	Northern England	Increase with age > U.S. above no sex difference
Bremner et al. (1968)	Jamaica	similar to U.K.
Severity:		
Kellgren and Lawrence	Northern England	Grade 3 - 4: males > females to 55 females > males 55+
Bremner et al. (1968)	Jamaica	severity < U.K.
Multiple joints:		
Kellgren and Lawrence	Northern England	Increase with age females > males

Age-related figures from archaeological samples are difficult to obtain since few collections are numerically large nor adequately preserved for statistical analysis by age and sex.

Pickering (1979) compares the incidence of osteoarthrosis between the sexes from the Ledders site, of the late Woodland period. Females show an earlier age of onset than males but males have a higher incidence in the later age decades. Martin et al. (1979) compare two populations, temporally distinguished, from Dickson Mounds. Osteoarthrosis appears in both groups in the 30 - 35 age range and increases with age until the 45 - 50 age group by which time all individuals show some involvement. There is no difference in incidence between the two groups. Sex differences are not considered. Jurmain (1977, 1978, 1980) compares samples from four American populations for incidence of osteoarthrosis of the large joints. The increase in involvement of the hip and shoulder show a closer relationship to age than the elbow and knee (Jurmain, 1980). The elbow and knee are viewed as more affected by mechanical demands over all ages. The four populations are compared for the incidence of osteoarthrosis of the knee. The Eskimo have the earliest age of onset and within the Eskimo, the female age of onset is earlier than males. However, over all ages the males show a higher incidence than female. The collection of Indian remains from Pecos Pueblo shows the lowest levels of incidence of the four populations. Amongst the two racial groups of the Terry Collection, blacks show a slightly higher incidence than

whites. These observations are exactly the opposite of the relative incidences between North American populations in Heberden's nodes, where the Indians had the highest incidence, blacks less than whites and the Eskimos the least involvement of all.

The severity of osteoarthrotic involvement is found to show a higher frequency of severe grades in males at the Ledders site, the opposite to modern trends (Pickering et al., 1979). The frequencies of more severe forms of osteoarthrosis is not found to be different between the two cultural horizons from Dickson Mounds, studied by Martin et al. (1979). The Eskimo population studied by Jurmain (1977) shows a higher frequency of severe cases amongst males.

The prevalence of females amongst sufferers of multiple joint involvement seen in modern populations is not duplicated in the archaeological samples of Jurmain (1980). Differences seen between modern and archaeological groups, particularly in the patterns of involvement by sex, may result from greater division of labour in the past populations. It may more probably reflect the lower mean age at death, since the greater involvement of females in modern samples of incidence, severity and multiple joint involvement tends to be noticeable after 55. The results of archaeological studies are summarized in Table 4.5.

A number of studies specifically on the spine have been published. Lawrence (1969) examined disc degeneration as part

Table 4.5 Summary of Incidence, Severity and Multiple Joint Involvement in Archaeological Populations with Age.

Researcher	Sample	Result
Incidence:		
Pickering et al. (1979)	Ledders Site	Females earlier onset than males Male > female
Martin et al. (1979)	Dickson Mounds	two populations incidence same age of onset same
Jurmain (1977, 1978, 1980)	Eskimo Pecos Pueblo Black American White American	hip, shoulder more age-related than knee, elbow Eskimo earlier than others Pueblo least Black > white Eskimo females onset before males Eskimo males > females
Severity:		
Pickering (1979)	Ledders Site	Males > females
Martin et al. (1979)	Dickson Mounds	No difference between the two populations
Jurmain (1977)	Eskimo	males > females
Multiple:		
Jurmain (1980)	Eskimo Pecos Pueblo Terry Collection	no difference in sex

of the Northern England study, plus samples from Rhondda and Watford. The cervical and lumbar spines were X-rayed in individuals over 35. Both regions of the spine showed a higher incidence of disc degeneration amongst males than females. The females show the maximum level of incidence later in life than males (Kellgren et al., 1953). Nathan (1962) studying osteophytosis in the Todd Collection found no significant differences between the sexes or between the black and white sub-groups. By 30 - 39, all individuals in the population had some evidence of osteophytosis. Roche (1957) examining the Terry Collection reports a higher incidence of both osteophytosis and osteoarthrosis in females compared to males. This is the opposite of the relative incidences of osteophytosis in the sample of Lawrence et al. (1969).

Caplan et al. (1966) studying the lumbar joints of miners finds the frequency of moderate and severe osteophytes increases with age, whereas the associated disc narrowing did not. The sample of Lawrence (1969) showed a higher frequency of grades of severity 3 - 4 in the lumbar and cervical discs amongst males. However, this difference occurred largely after 55, the relative incidences of the more severe grades being equal up until 55. There is no comparable sex difference in the lower grades of severity, so the evidence is that males simply develop a more severe form of osteophytosis with age. Nathan (1962) observes no sex differences in the incidence of more severe grades, nor any differences between black and white sub-groups. The

frequency of grades 3 - 4 of osteophytosis increased with age and by 70 - 79 all individuals had at least one joint with grade 3 - 4.

Multiple joint involvement of osteophytosis was found to be more frequent amongst males in the U.K. sample of Lawrence (1969). This sex difference was observed at all ages. These results of modern sample age and sex relationships are summarized in Table 4.6.

Patterns of age-relationahip of degenerative joint disease of the spine in archaeological populations can be compared with modern samples. Sager (1969) examines a modern Danish population and the Aebelholt skeletons for osteoarthrosis and osteophytosis of the cervical spine. He finds significant increases of incidence with age for the facet and disc joints in both modern and mediaeval samples. The incidence of osteophytosis reached 86% in the 70+ age group in the modern sample and 74% in the oldest age group of the mediaeval sample, using four adult age groups. Facet joints showed a lower incidence of 53·8% in the 70+ age group of the modern sample and 49% in the oldest mediaeval age group. Males showed a higher incidence than females in both kinds of joint and in both populations. Discs showed a higher incidence than facets at all ages, but the relative increase with age was the same for both in the modern sample. In the mediaeval sample, the rate of increase of osteophytosis was greater than osteoarthrosis with age. The modern sample had a higher incidence of disc involvement than the mediaeval,

Table 4.6 Summary of Incidence, Severity and Multiple Joint Involvement in Modern Samples in the Spine with Age.

Researcher	Sample	Result
Incidence:		
Lawrenc et al. (1969)	U.K.	osteophytosis males > females
Nathan (1962)	Todd Collection	osteophytosis no sex differences no racial differences 30 - 39: 100% involvement
Roche (1957)	Terry Collection	osteophytosis and osteoarthrosis females > males
Severity:		
Caplan et al. (1966)	U.S. miners	osteophytosis, increases with age disc narrowing, does not
Lawrence et al. (1969)	U.K.	osteophytosis males > females
Nathan (1962)	Todd Collection	osteophytosis no sex differences no racial differences 70 - 79: 100% grades 3 - 4
Multiple:		
Lawrence et al. (1969)	U.K.	osteophytosis males > females

whereas the reverse was the case for the facet joints. Ingelmark et al. (1959) studied the same mediaeval sample, but examined the entire vertebral column, using the same age categories and scoring system as Sager (1969). The incidence in the disc joints increases from none in the first age group to 40% in the next, 82·8% in the third and 89·5% in the oldest. No sex differences were observed in contrast to Sager's (1969) study of the cervical region alone. The facet joints showed a similar increase from a very low incidence in the first age group to 44% in the second, 71% in the third and 91·9% in the oldest group. Again no sex differences were noted. There is no significant difference in incidence between the disc and facet joints found at any age. Stewart (1958) combines the males from the Terry Collection with the male sample from the Korean War to study incidence of osteophytosis and finds an age-related increase in frequency.

In severity grading, Sager (1969) finds a static trend until 60, after which it increased dramatically in the cervical disc joints of the modern sample. The facet joints showed a gradual increase of severity up to 70 followed by an accelerated increase. The mediaeval sample was not so precisely aged and simply showed a gradual increase in severity with age group for both facet and disc joints. Sex differences in severity were not discussed. The mediaeval sample had a higher frequency of severe cases of both osteophytosis and osteoarthrosis than the modern one. Ingelmark et al. (1959) find an increase in severity with age over the entire spine but which is not significant for

both sets of joints. Stewart (1958) does find a significant increase of severity of osteophytosis with age, but says severe lipping only becomes pronounced after 50. Stewart (1957) claims the very few more severe cases of osteophytosis appearing before 30 represent the result of back injuries rather than some form of premature aging. Stewart (1958) and McKern and Stewart (1957) stress the variation in each age group of the degrees of severity encountered.

The number of joints involved in osteophytosis in the Aebelholt sample increased significantly with age from 7·8% of the column involved on average in the second age group, 37% in the third and 54·2% in the oldest group. Again Ingelmark et al. (1959) found no sex differences. The facet joints showed the same increase of number involved with age, again with no sex difference. The second age group had 6·5% of the joints affected, a figure which increases to 17% in the third age group and to 30% in the oldest group. Stewart (1957) remarks that an increase in the number of joints affected in osteophytosis in his sample in association with increasing age is very noticeable (McKern and Stewart, 1957). The findings from the archaeological samples, the modern Danish sample and Stewart's combined sample are presented in Table 4.7.

It seems, therefore, that the occurrence of degenerative joint disease in the spine may show a different pattern between the sexes than other joints of the body. Where a sex difference is found, in both modern and archaeological

Table 4.7 Summary of Incidence, Severity and Multiple Joint Involvement in Archaeological Studies in the Spine with Age.

Researcher	Sample	Result
Incidence:		
Sager (1969)	Aebelholt Modern danish (cervical spine)	osteophytosis and osteoarthrosis males > females disc > facets modern relative rate increase same mediaeval disc rate > facet modern osteophytosis > mediaeval modern osteoarthrosis < mediaeval
Ingelmark et al.(1959)	Aebelholt (all spine)	no sex differences
Stewart (1958)	Males, Terry Collection and Korean war	osteophytosis significant increase with age
Severity:		
Sager (1969)	Aebelholt Modern danish (cervical spine)	Modern disc static to 60 then increase Modern facet gradual to 70 then increase Mediaeval disc and facet gradual increase Mediaeval disc and facet > modern
Ingelmark et al. (1959)	Aebelholt (all spine)	Increase with age not significant
Stewart (1958)	males, Terry Collection and Korean war	Increase osteophytosis with age severe, 50+ pre-30 severe = injury Range of variation in each age group
Multiple:		
Ingelmark et al. (1959)	Aebelholt	Disc and facet significant increase with age. No sex differences
Stewart (1958)	males,Terry Collection and Korean war	increased osteophytosis with age

samples, the males display greater incidence, severity and multiple involvement of osteophytosis. Only Roche (1957) reports the opposite. Fewer studies have been made on the facet joints, but hint at the same trend (Sager, 1969). Again Roche (1957) records a higher female incidence than male.

4.8 The Use of Degenerative Joint Disease in the Spine for Aging Purposes.

The incidence of degenerative joint disease in both kinds of joint shows an increase in a population with age. However, at any time there is almost always found to be some individuals without any involvement. Simply finding an increase in incidence does not provide the anthropologist with a means of establishing the age of any single individual in the population.

Examination of severity suggests this also increases with age. Usually four grades of severity are used but again, in any age category a wide range of cases of severity will be found. Severity seems to particularly increase with age after about 55 in modern samples. Since many archaeological samples are thought to have a lower mean age at death, very few will necessarily display marked severity.

The vertebral column is composed of many individual joints. The number of joints affected increases with age and a measure of the % of the column involved gives a wider measure than simple severity. The two measures could be

considered together to incorporate both extent and severity of involvement.

The two kinds of joint both show age-related degeneration but studies of distribution and relationships between the two have suggested that degeneration procedes independantly in the two sets of joints. A consideration of both types of joint together could therefore prove profitable.

Lastly, sex differences have been demonstrated to occur. The disc joints definitely show a tendency for males to have greater levels of incidence in the population, and of extent and severity over females. It is not clear if this is also the case in the facet joint. Other synovial joints of the body show a higher incidence in females, with greater severity and extent of involvement, but usually only particularly marked after 55. Sex differences in grades and scores have to be borne in mind when the conversion from observed degeneration to an age estimate is made.

PART II

ORIGINAL RESEARCH

Chapter 5. MATERIALS AND METHODS.

5.1 Background of the Sample.

The skeletal collection used in this study comes from the Romano-British cemetary outside Dorchester (Dorset) at Poundbury. The cemetery was excavated over a twelve year period between 1966 - 1978 by Christopher Green. Over a thousand burials have been exhumed over a two acre area, although the total cemetery is thought to cover five acres with a possible total of three to four thousand burials. Dates have been obtained from both carbon tests and from coins found with burials. The main part of the cemetery was in use in the later part of the Roman occupation of Britain, in the 3rd and 4th centuries. However, some areas do represent both earlier and later burials, beginning as early as the first century and continuing in use into the post-Roman period (Green, 1966, 1968, 1972, 1973, 1974, 1975, 1976). The middle-third century saw an apparent decline in prosperity, thought due to high inflation rates and unrest in Gaul and Germany. An eventual separation of Gaul, Spain and Britain from Rome occurred in 259 under Postumus, followed by Victorinus and Tetricus, lasting until 274 when they were recovered by Aurelian. Todd (1981), however, views this traditional interpretation of events as unsubstantiated and does not feel the case for economic decline is proven.

Nonetheless, over the course of the subsequent century, dissatisfaction with the administration from Rome and its inadequate protection of civilian Romano-Britain is perhaps reflected by the number of insurrections in the Western provinces during the 4th century (Todd, 1981). For example, the naval commander, Carausius, rebelled, was condemned to death and used the navy to seize power in Britain. Todd feels Britain fell to his attack surprisingly easily suggesting other forces at work against Rome than simply Carausius' military skills. The very real threat posed to Britain by invasions of the Picts is demonstrated by the fact that in 306, Constantius then emperor, himself came to lead the counter-attack, which was then continued in a number of campaigns by his successor, Constantine (Todd, 1981). The threat from Germanic tribes may have begun even as early as the second century and by the end of the third century, a line of shore forts had been constructed in defence along the Southeastern coast from the Wash round to Southampton (Alcock, 1971). As threats to Rome herself grew at the end of the 4th century, troops were increasingly withdrawn from the more distant provinces such as Britain, allowing an escalation in raiding from Britain's enemies. Between 406 - 7, Britain elevated three would-be usurpers to the emperor, symptomatic again of dissatisfaction with the central administration and a hope of a better deal from their 'own man' (Alcock, 1971). By 410, it is thought that Britain rebelled against Rome's supremacy and removed herself from Rome's jurisdiction rather than the opposite of Rome pulling out. Honorius' decree from Rome, that the British should look to their own defences, was

a way of saving face in Rome. This active role by the British in the split is recorded quite categorically by the Roman historian Zosimus (Todd, 1981; Alcock, 1971). Romano-British culture and presumably the trade-links would have continued well on into the start of the so-called Dark Ages in the 5th century, only gradually becoming effaced by the incoming Saxons. Germanus of Auxerre in his visit to Britain in 429 records meeting civic leaders at York who were obviously still able to afford fine garments and a high life-style (Todd, 1981).

The county of Dorset was originally ruled by the tribe known as the Durotriges. The area appeared to be centred on two main forts at Maiden Castle and Hod Hill at the time of the Roman invasion, lead in this area by Vespasian. The Romans would appear to have based themselves at Dorchester, called Durnovaria, where there was a small Celtic hillfort of Poundbury but which does not appear to have been a significant centre prior to the advent of the Romans (Wacher, 1974). Little is known of Dorchester and its inhabitants but by the 3rd century the process of Romanisation would be over and the Romano-British synthesis become a way of life (Johnson, 1980). Dorchester is known to have been the home of one of the major Romano-British schools of mosaics (Johnson, 1980; Johnson, 1982) suggesting the town was predominantly a civilian centre rather than a military base at this time. The region seems to have spawned another centre at Ilchester, called Lindinis, which although not apparently so important in fact is situated in far richer

agricultural land (Rivet, 1964) and has a far greater cluster of villas around it, indicative of both wealth and political power (Rivet, 1964; Wacher, 1974). So the exact role of Dorchester in the area appears ambivalent perhaps being the earlier centre of power with a gradual shift to Ilchester as the 4th century progressed.

Very little is known of the composition of the population in Romano-British times, of their lifestyles and customs, or of their health. The cemetery at Poundbury shows a number of features unlike other late Romano-British cemeteries of the area, but rather having ressemblances to typical Christian cemeteries. The majority of the burials are in wooden coffins, orientated with the head to the West. A number of the burials have been packed in gypsum, particularly the lead-lined coffins, a Christian custom and probably indicative of wealth (Toller, 1977). One of the lead coffins bears an inscription IN DNE, expanded to read In Nomine (tuo) Domine. Very few grave goods were found with the orientated burials again typical of Christian rites. Mosaics found in the vicinity at the villas of Frampton and Hinton St. Mary show motifs thought to be Christian, and the Frampton mosaics at least are believed to be the work of the Dorchester school (Johnson, 1980; Johnson, 1982). This would suggest a flourishing Christian community in the area during the 4th century. This may prove to be a commonplace feature of the regional capitals of the South-east by the late Roman period, as indeed is thought to be the case on the continent (Green, 1977).

It is assumed in a civilian market town, such as Dorchester, that the number of immigrant settlers was probably never very significant and certainly any would have been absorbed into the community by the 3rd and 4th centuries. The city leaders and administrators would be indigenous dignitaries rather than imposed rulers. One of the lead burials from the cemetery had hair surviving in a pigtail, possibly a sign of the retention of Celtic styles amongst the wealthier classes (Green, 1977). Alternatively, it could represent an adherent of one of the pagan religions, such as Mithraism, many of which were thought to undergo popular revival (Johnson, 1980). However, since the coffin was oriented in custom with Christian customs the former suggestion is preferred.

Almost nothing is known of Romano-British health and in this respect analyses of the Poundbury collection should provide some unique information. The skeletons are currently housed at the British Museum of Natural History, in the care of Theya Molleson, who has supervised the collection of basic data by Dr. Patty Stuart-Macadam and Robert Kruszynski and is now carrying out analyses of these data with Suzanne Gautier. In addition numerous detailed studies have been or are being made on specific areas.

There is no evidence for the prevalence of rickets and the fracture rate seems fairly low. There could be some evidence of cemetery site differences in the latter but the uneven sample sizes of the different areas makes the observation inconclusive (Molleson, pers. comm.). An appraisal

of the use of stature estimates was made from the Poundbury collection by McClintock (1984), assessing the most appropriate formula for this sample by comparison of Poundbury limb bone proportions to the reference samples. Her conclusion was that the Dupertius and Hadden formula was the best suited for stature estimation in the Poundbury sample.

A general study of the incidence of the arthritides compared to modern day levels by Thould and Thould (1983) found a high incidence of osteoarthritis especially in the spine and a similar prevalence of vertebral ankylosing hyperostosis and rheumatoid arthritis. The latter claim was surprising as rheumatoid arthritis had always been considered as a modern disease. However, in response to the publication Rogers and Dieppe (1983), who have examined a wide range of Saxon and Romano-British remains, question the diagnosis of rheumatoid arthritis as described and feel it is better interpreted as psoriatic arthropathy or Reiters disease. Thould and Thould (1983) record no incidence of gout from visual observation, although the analysis of pathologies through radiography carried out at the Natural History Museum by Dr. John Price has indicated the presence of gout (Molleson pers. comm.). As Rogers and Dieppe (1983) emphasise in their communication, the criteria for diagnosis in palaeopathology are far from adequately defined.

The large sample size has allowed a number of studies to be made, similar to the present one, where, although the

control information is not known i.e. in the present case age, the number and completeness of the remains, allow sufficient comparison to be made to draw strongly argued conclusions. For example, those skeletons able to be sexed with a high degree of confidence from the pelvis can then be used to assess the accuracy of other measured and observed sexing criteria. In the Poundbury sample the tooth diameters proved to be clearly sexually dimorphic in adults and since a bimodal distribution is also found in juvenils, a strong case is presented for sexing those juveniles (Molleson, pers. comm.).

Whittaker et al. (unpubl. m.s.) studied the dental health of the sample and reported that Poundbury did not suffer from excessive tooth loss ante-mortem, and that the rate of tooth loss correlated well with age. Patterns of former shape of the condyles of the temporomandibular joint were also observed to change with age and a suggestion is proffered that a relationship exists between tooth loss and condylar change. No relationship was found between attrition and condylar shape.

A study of cribra orbitalia from Poundbury and modern radiographs has elucidated the relationship of the observed pitting to the various anaemias. The pitting most probably represents the occurrence of childhood anaemias (Stuart-MacAdam, 1982).

It is known that food preparation techniques in Roman

times could have caused high lead levels in the food, from water piping and utensils. Reports of high lead levels in Roman times have caused much debate and controversy over the years, even giving rise to a suggestion that lead poisoning could have been an important contributory factor to the fall of the Empire. Studies of lead levels at Poundbury have been made from bone, teeth and soil. Samples of lead levels from burials in lead coffins show an extremely high level indicating post-mortem uptake by the skeleton from the coffin. The importance of taking lead samples from the post-mortem environment is thus emphasized (Waldron, 1981). The soil levels at Poundbury are not high, and do not correlate well with the bone levels which are much higher than both the soil or modern levels (Waldron et al., 1979). Deciduous and permanent teeth were analysed for levels of lead, cadmium and zinc. Cadmium is rarer but more toxic than lead; zinc is commoner and less toxic. These three are routinely screened for in modern analyses so could provide a useful baseline for comparative data. The preliminary studies at Poundbury found far higher levels of all three, particularly lead, in deciduous teeth than in the permanent dentition. The ratio of zinc to the other two metals appeared lower than is usual in modern samples. The variation in levels between the different specimens was very high so that the mean values are of limited value (Whittaker and Stack, 1984). Work on the uptake of trace elements, their toxic effects, age relationships and their interactions with one another is at an early stage of investigation and reports such as these will provide an accumulation of descriptive data to work

from, rather than conclusive evidence for toxicity in a particular population.

5.2 Age and Sex Assessment.

The Poundbury collection numbers 1228 individuals in total. All the skeletons were sexed and aged as part of the overall bone study and report performed at the British Museum (Natural History) by Dr. Patty Stuart-MacAdam. The sex of the juveniles was left undetermined; the sex of the adults was ascertained on all possible criteria, leaving it as unknown where the skeleton was insufficiently complete. For the Poundbury collection the Phenice sexing criteria (Phenice, 1969) were found to be particularly diagnostic of sex (Stuart-MacAdam, pers. comm.). For the purpose of comparability of this study with other studies carried out on the Poundbury material, the sexes assigned by Dr. Stuart-MacAdam were used.

The juvenile ages were assessed on the basis of tooth eruption, epiphyseal union and general skeletal development (Stuart-MacAdam, pers. comm.). Once epiphyseal fusion had occurred, the adults were aged at the Museum by Dental Attrition scores and by the metamorphosis of the pubic symphysis. Again Dr. Stuart-MacAdam's grading for these two criteria have been used in this analysis.

ex

r. Macadam's own description of the procedures used for sexing the skeletons re given below (Macadam, unpub.ms.):

Sex was determined primarily from pelvic morphology with a greater reliance on henice's diagnostic criteria" (1969). These criteria are:

. presence or absence of a ventral arc on the pelvis;

. presence or absence of a narrow medial aspect of the ischio-pubic ramus;

. presence or absence of a subpubic notch.

his method was used because of its high reliability in conjunction with the act that the pelvis is known to be the most reliable sex discriminator.

ther pelvic and sacral morphology was also examined.

. Subpubic arch
. Body of pubis
. Symphysis
. Obturator foramen
. Acetabulum
. Greater sciatic notch
. Sacro-iliac articulations
. Pre-auricular sulcus
. Ilia
). Sacrum

eatures of other areas of the skeleton were also examined, particularly the kull as well as the overall appearance of bones in terms of size and muscle tachments. The features of the skull which were examined are:

The size of vault and face
Robusticity
General physiognomy
Supraorbital ridges
Forehead - slope, height
Superior border of orbit
External occipital protuberance
Mastoid processes
Zygomatic bone
. Mandible - general appearance
. Gonial angle
. Chin projection
. Chin form.

ne pelvic indicators depend on differences related to childbearing where the emale pelvis is broader overall which affects individual anatomical features. ne other criteria tend to relate to size and robusticity, the male displaying greater degree of these. In the situation of conflicting evidence from the ifferent criteria, greatest weight was placed on the pelvic indicators, articularly phenice. Where no clear pattern emerged, sex was assigned on the asis of the majority of criteria.

r. Macadam additionally notes that in the absence of skull or pelvis, tentative ex could be assigned from metrical features being recorded also in the oundbury study. In the subsequent analysis it was found that metrical features nowing markedly bimodal distributions such as femur head diameter, clavicle ength, corresponded well to the sex assignments already made by Dr. Macadam Molleson, Gauthier, pers.comm.). Whether this validates Dr. Macadam's sex ssignment and describes the Poundbury population as sexually dimorphic for nese features or rather suggests that she may have been unduly influenced ubjectively by evident size and robusticity factors is difficult to assess. either clavicle size nor femur head diameter are necessarily very prominent eatures in the examination of an entire skeleton, and Dr. Macadam's own iteria place major emphasis on features which would not be influenced by gross ze. It is suggested therefore that Dr. Macadam's visually based assignment of x would not have been heavily influenced by evident dimorphism in the pulation.

ere is always the problem of bias in sexing discussed by Weiss, with the ndency to sex in favour of males by as much as 12%. Again there is very ttle way of gauging the risk of this bias in an individual's sexing technique. e bias towards males is seen as arising from a tendency to look for male

raits, particularly as regards size. Since great reliance was placed on the elvis here, where specifically female traits are tending to be looked for, it ight be argued that if anything the bias might be towards females. Given a ood number of skeletons where preservation gave skull and pelvis for xamination, it can only be hoped that Dr. Macadam was able to establish for erself the sex differences in this population of size assessments on the skull uch as mastoid processes etc. Dr. Macadam certainly was very familiar with the ollection before commencing the data collection having already studied all the kull material for her doctorate on cribra orbitalia.

inally, however, the possibility of mis-assignment of sex can never be ruled ut and if this were as much as by 12% it would, of course, seriously affect the esults in terms of any sex differences found by the X^2 and Mann-Whitney U tests r any sex differences commented upon in the strength of correlations or in oefficients of variation.

5.2.1 Dental Attrition.

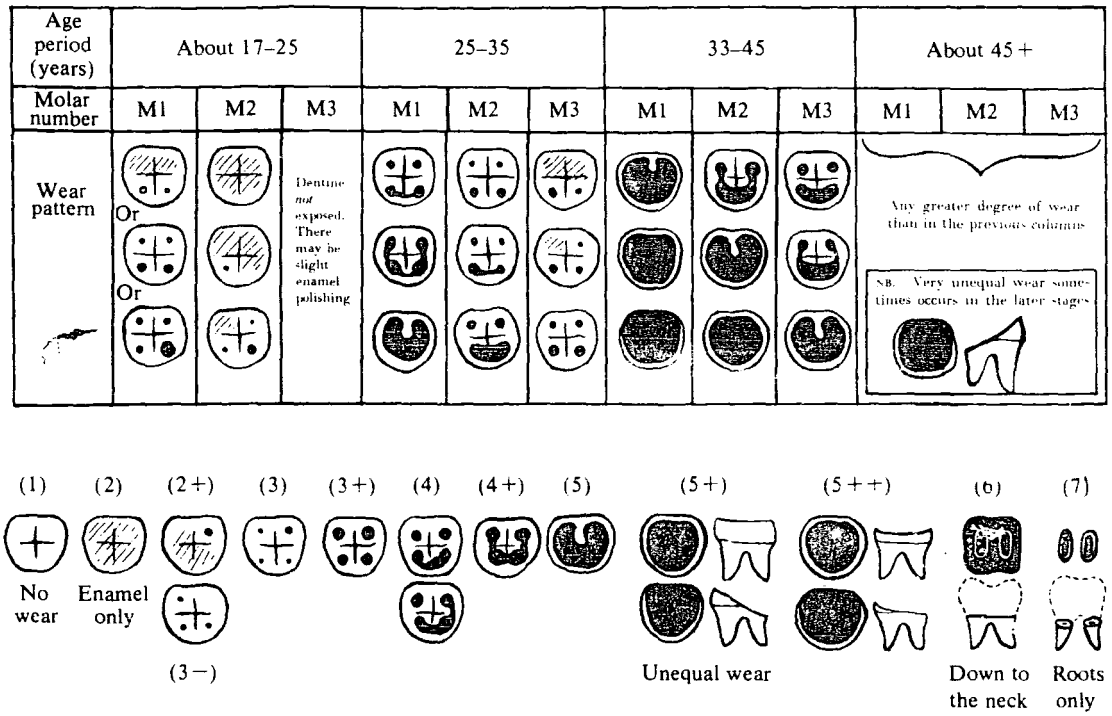
As discussed earlier, there are a number of different scoring methods available to assess the degree of dental attrition. The one used for the Poundbury skeletons was that published by Brothwell in "Digging up Bones" (1972). The + grades have been coded as x.5 as the implication is that it falls between the two 'whole' stages; the only ++ grading (5++) has been included in the top category of 6.0.

The three molars from the left side of the mandible were each graded on this scale for attrition. However, the sample size for M_3 has not been sufficiently large by comparison to M_1 and M_2 to be included in statistical analysis. In any jaw displaying obviously uneven wear, malocclusion, ante-mortem tooth loss or any pathology that could lead to unequal wear in the jaw, the teeth were deemed as ungradeable for aging purposes. The Brothwell chart gives large age categories into which the various stages of wear fall.

Each individual has been accordingly put in an age category following this system and using Dr. Stuart-MacAdam's raw grades for the three molars. Brothwell's chart does not provide different charts for the sexes, so it is assumed in this case that the two sexes experience attrition at the same rate, an assumption already questioned earlier and which will be investigated in the analysis of the data.

The chart from Brothwell (1972) for grading tooth wear and assignment to an age category is presented over. The dark patches represent the areas of exposed dentine.

The grading was based on a series of populations from neolithic to mediaeval times, a time period over which Brothwell claims, "the rates of wear do not appear to have changed much". In the group from Maiden Castle, the dental attrition has been checked against the pubic symphysis face, although which pubic symphysis age method used is not clear from Brothwell. However, the age of the samples used is unknown for true calibration so the age categories remain broad and should be viewed as relative categories rather than absolute age groups.



Numerical classification of molar wear

(N.B. Some patterns are more common than others, and there are minor differences between upper and lower dentitions.)

Figure 3.9 A tentative classification of age in Neolithic to Medieval British skulls, based on molar wear.

(Brothwell, 1972)

5.2.2 Pubic Symphysis Changes.

The scoring system used to assess the changes in the pubic symphysis are those of McKern and Stewart (1957) for the males, based on their sample of Korean war dead, and on the Gilbert and McKern (1973) method for the females. As discussed earlier in the introduction, the two methods are based on the development of the same three features of the pubic symphysis for both sexes, but the age ranges of the two samples are different. This means the possible range of ages given for the Poundbury material is also different for the two sexes so that the two mortality profiles are uneven in this respect. To bypass this problem and make the two groups comparable, categories corresponding to those of Brothwell for dental attrition have been used taking in the scores covering the same ranges as Brothwell's for both male and female. The juveniles have been divided into five categories, followed by Brothwell's five categories for adults.

Dental age categories and juveniles.

age range	category
0 - 6mths	1
6m - 1y	2
1 - 5y	3
5 - 10y	4
10 - 15y	5
15 - 20y	6
20 - 25y	7
25 - 35y	8
35 - 45y	9
45+	10

Pubic symphysis categories

	age range			age range		category
female	13 - 24	19•8	male	17 - 20	19•0	6
	16 - 25	20•2		18 - 21	19•8	
	18 - 25	21•5		18 - 23	20•8	7
	22 - 29	26•0		20 - 24	22•4	
	25 - 36	29•6		22 - 28	24•1	
	23 - 39	32•0		23 - 28	26•1	8
	22 - 40	33•0		22 - 29	29•2	
	30 - 47	36.9		29+	35•8	9
	32 - 52	39•0		36+	41•0	
	44 - 54	47•8				10
	52 - 59	55•7				

5.3 Bone Structure Measurements.

Thin sections of bone were prepared from the femoral cortical bone. Sections were taken from the anterior midshaft of the femur using an ordinary hacksaw. Thin sections were prepared in Durham Anthropology department laboratory by Lesley Bailey and Tracy Horsfall. A thinner section was cut from the original section by a slow circular saw. These sections were then left in a fixing solution for a minimum of 24 hours. The fixing mixture was 6 gm PVA : 50ml acetone. The sections were subsequently air dried for a minimum of 12 hours. These sections were then ground down further, to as near 150μm as possible from the fragility, using wet and dry carborundum paper. If the sections are not too fragile they should then be rinsed under the tap. After drying on a tissue, they are mounted on a slide using DPX. This fixing and grinding method is that used by Samson (pers. comm.). It was not possible to make thin sections from the more fragile bones. These were those of poor preservation, juveniles and osteoporotics. It is recognised therefore that underrepresentation is given to the very young and very extreme, possibly the very old, skeletons.

The measures taken were those necessary to construct the age estimations of Kerley, Samson, Ahlquist and Damsten and Thompson.

Kerley (1965, 1969) makes use of four parameters of bone structure. Each is used in a different equation for age

estimation. The first is based on the number of secondary osteons present. This is referred to as KAO throughout this thesis. The number of secondary osteons are counted in four circular fields from the periosteal surface at quarter points around the entire cortical section, as described earlier. The numbers from the four fields are then added together and put into the equation. As only a semi-circular section has been taken here, the four fields were taken at regular intervals along the anterior periosteal surface, rather than around an entire cortical section. It is hoped that no major differences will be present in the anterior/posterior portions of the periosteal surface bone. Samson suggests it does not really make much difference where the measurements are made, so long as the linea aspera is avoided (pers. comm.), and presumably so long as consistency is maintained. Kerley's microscopic field size was 2·47 times the size of the microscopic field in this study (Kerley and Ubelaker, 1978). Therefore the sum of the number of osteons in the four fields was multiplied by 2·47 before used in the equation. This measure is referred to as KNSO. (Kerley's number of secondary osteons). The same method was used for two of the other Kerley methods, the number of osteon fragments (KNOF), and the number of non-Haversian canals (KNNH). The fourth Kerley measure was the % of the bone area still circumferential lamellar bone. This was assessed for the four fields and the mean taken. No adjustments were necessary for differences in field size (KPCL). The four equations for age estimates by Kerley are as follows:

KAO (Kerley Age, osteons)

$$= 2{\cdot}278 + 0{\cdot}187 \times KNSO + 0{\cdot}00226 \times (KNSO)^2$$

KAF (Kerley Age, fragments)

$$= 5{\cdot}241 + 0{\cdot}509 \times KNOF + 0{\cdot}017 \times (KNOF)^2 - 0{\cdot}00015 \times (KNOF)^3$$

KAN (Kerley Age, non-haversians)

$$= 58{\cdot}390 - 3{\cdot}184 \times KNNH + 0{\cdot}0628 \times (KNNH)^2 - 0{\cdot}00036 \times (KNNH)^3$$

KAL (Kerley Age, lamella)

$$= 75{\cdot}17 - 1{\cdot}790 \times KPCL + 0{\cdot}0114(KPCL)^2$$

Kerley suggest the best age estimate would be achieved if the four were used in conjunction to define the region of overlap and thus to narrow down the accuracy of age estimation. The resolution is given as $\pm$10 years with 95% accuracy.

Ahlquist and Damsten (1969) developed a simple measure of the % area of a square field covered by secondary osteons and osteon fragments. They use a 10 x 10 grid and count the number of squares more than half covered by osteon bone for the %. This is an improvement on Kerley's estimation of age from the % of circumferential lamella which was to be judged by eye. Otherwise it represents much the same measure in reverse. Again no adjustments are needed to be made to the measure for differences of field size as the measure is a relative one. The measure of % of the grid covered by osteons and fragments is referred to here as ADPO (Ahlquist and Damsten, percent osteons). The Ahlquist and Damsten

equation is as follows:

ADAGE (Ahlquist and Damsten age) = 9·991(ADPO) - 4·96

The resolution is given as $\pm$6·71.

Thompson (1978, 1979, 1980) has five equations for age estimation, each successive one adding an additional parameter. The first, TAA (Thompson Age A.) uses the same measure as the Ahlquist and Damsten method, ADPO. The equation therefore is:

TAA = 6·677 + 101·936(ADPO/100)

The next measure to be added is the external bone structure measure of cortical thickness (CTHICK). This was measured using a graticule on an Olympus light microscope at a magnification of x10 and where 1·5 graticule units represented 1mm. The measure was adjusted accordingly and the results given in mm. This second Thompson age estimate (TAB) is constructed as follows:

TAB = 20·969 + 95·278(ADPO/100) - 2·314(CTHICK)

The third and fourth parameters are the mean perimeter length of the secondary osteons and the total perimeter length in the field. Again the 10 x 10 squared grid is used. The total length of perimeter in the square field is given by the equation

TOPL = $(\pi/2)$IL,

where TOPL is the total length of the perimeter (Thompson osteon perimeter length) and IL a measure of the number of times one of the grid lines is intersected by an osteon perimeter, divided by the total length of grid lines. The

length of each grid line is 0·59mm and there are twenty-two lines, therefore the total is 12·98mm. The mean osteon perimeter length (TMOPL) is then obtained by dividing TOPL by the number of osteons in the field. The third and fourth age estimates (TAC and TAD) are given as follows:

TAC = 47·644 + 96·394(ADPO/100) - 2·457(CTHICK) - 47·590 x (TMOPL)

TAD = 72·059 + 127·853(ADPO/100) - 1·797(CTHICK) - 83·949(TMOPL) - 2·739(TOPL)

The final Thompson parameter is the number of secondary osteons in the field (TNSO). Thompson's square field was 2·827 x the size of the field in the present study, so the number of osteons was adjusted accordingly. The equation for the fifth Thompson age estimation (TAE) is as follows:

TAE = 28·978 + 128·557(ADPO/100) - 1·796(CTHICK) - 7·543(TMOPL) - 7·633(TOPL) + 2·688(TNSO)

The method of Samson (1983) involves measures of the number of secondary osteons per mm^2 (CNSO) and the mean minimum osteon diameter in μm (AMOD). The area of the field in this study was 0·8322mm^2 so the mean number of osteons over a number of fields was divided by this figure to give CNSO. AMOD was measured with an eye-piece graticule where 175 of the graticule units represented 1mm. The measurement by the graticule was accordingly adjusted to obtain the measure in mm, and multiplied by 1000 to give the measure in microns. These two measures are multiplied together to give a combined figure referred to by Samson as the

morphological value (MCV). This is used in the equation below to give the age estimation (CLAGE):

$$CLAGE = 0{\cdot}1264763(MCV)^{0{\cdot}9656}$$

This equation is given for use with males only, however, it has been applied to the Poundbury females also for the sake of comparison.

The Thompson methods give a standard error of estimate as 8·6 for TAA decreasing to 7·1 for TAE.

Samson's method of age estimation gives a resolution of $\pm$6 years, equivalent to the Ahlquist and Damsten method, Considering that the Ahlquist and Damsten method is by far the simplest to perform, this looks the most promising.

The age estimates will be referred to en masse throughout the analysis as the microages. The measures of bone structure features will be referred to as the microparameter. The codes to be used for each of the microparameters and microages are given again in Table 5.1.

Fig. 5.1 illustrates bone structure at different stages of development.

Table 5.1 Codes used throughout for the variables of Microages and Microparameters.

Microages

CLAGE	Samson's method	
ADAGE	Ahlquist and Damsten's method	
KAO	Kerley's methods	osteon number
KAF		fragment number
KPCL		% area circumferential lamellar bone
KNNH		non-Haversian canal number
TAA	Thompson's methods	
TAB		
TAC		
TAD		
TAE		

Microparameters

KNSO	Kerley number secondary osteons
KNOF	Kerley number osteon fragments
KPCL	Kerley % area circumferential lamellar bone
KNNH	Kerley number non-Haversian canals
ADPO	% area osteons and osteon fragments
TNSO	Thompson number secondary osteons
TOPL	Thompson total osteon perimeter length
TMOPL	Thompson mean osteon perimeter length
CTHICK	Cortical thickness
AMOD	Average mean osteon diameter
CNSO	Samson number secondary osteons

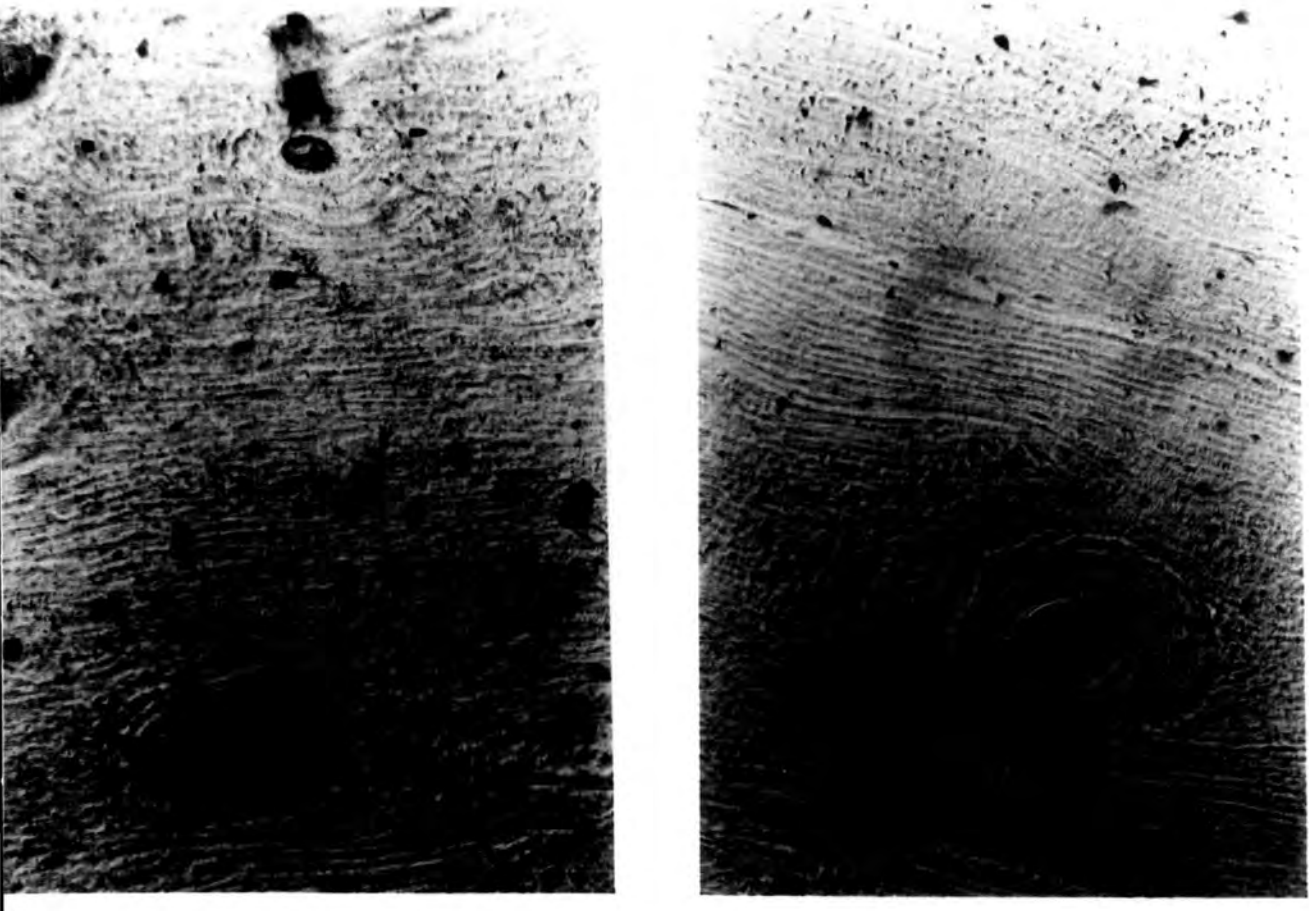
FIG. 5·1 STAGES OF BONE STRUCTURE DEVELOPMENT.

A CHILDHOOD BONE SHOWING:

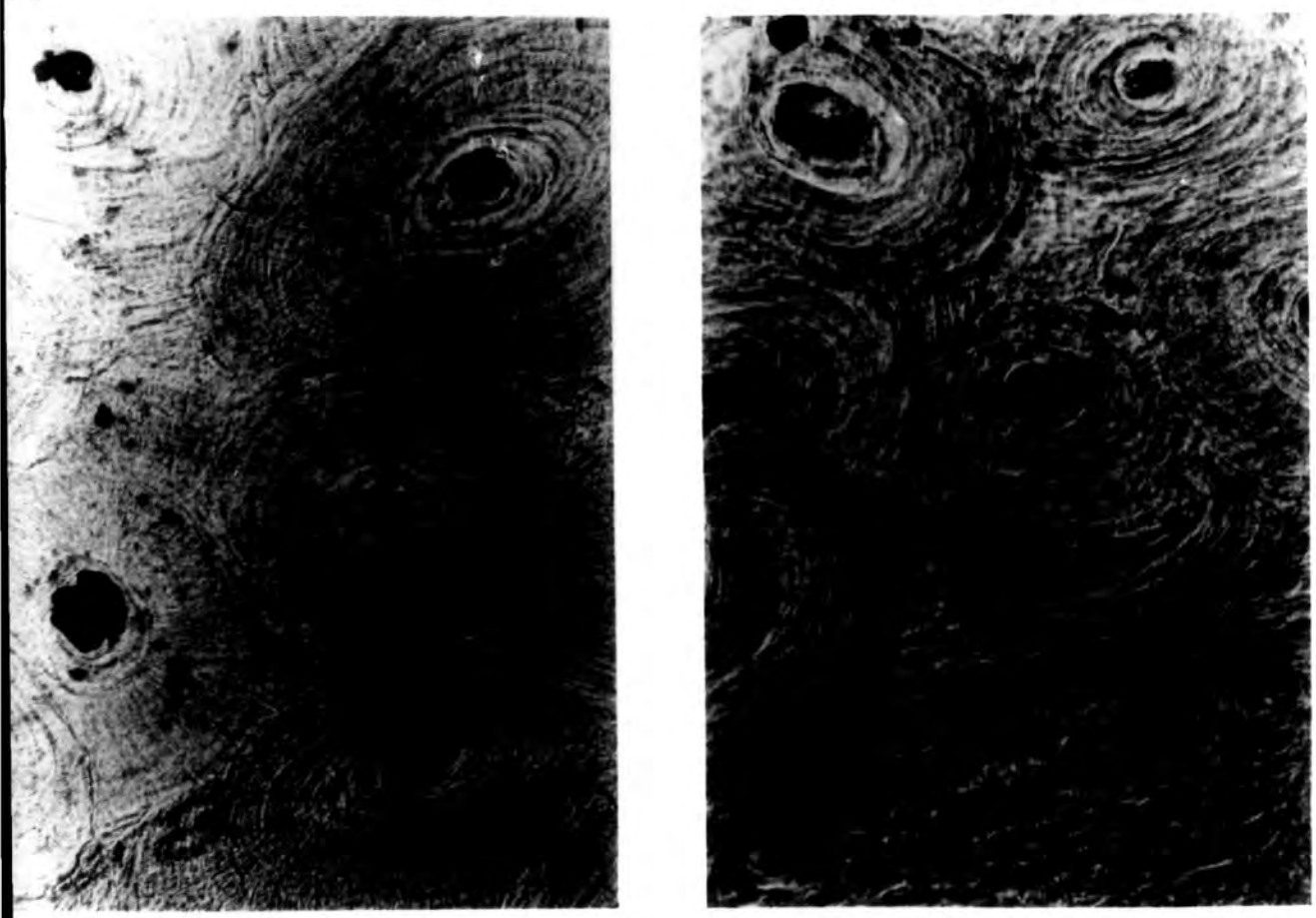
PARRALLEL-LAYERED LAMELLAE
NON-HAVERSIAN CANALS
YOUNG SECONDARY OSTEONS

B ADULT BONE SHOWING:

COMPLETE OSTEON
OSTEON FRAGMENTS
CIRCUMFERENTIAL LAMELLAE



A



B

5.4 Scoring for Osteoarthrosis and Osteophytosis.

The method used to score for the presence of osteoarthrosis and osteophytosis in the spine was based on the stages described by Sager (1969). Sager identifies four stages in the progression of the disease of both osteoarthrosis and osteophytosis from 0 when no osteoarthrosis or osteophytosis is present up to 4 when the bony lipping has formed a fused bridge across the joint.

Sager (1969) based his stages on the progressive degeneration of four features of the bone: - porosity of the articular surface, osteophyte formation around the edge of the articular surface, sclerosis of the bone of the articular surface and eburnation of the bone of the articular surface, Sager scored each of these separately, added up the score to give a total value. He also presents a simple four stage system of the overall state of the joint.

In the Poundbury material the facets were scored for porosity, osteophytes and eburnation/sclerosis and the body scored for porosity and osteophyte formation. In the analysis the body joint osteophytes have been kept separate as the two did not always appear to progress together as indicated by Sager and osteophytosis is taken to be the degree of osteophyte formation on the body edge. The facets were seen to display the features described by Sager more consistently and so a simple overall grade was also assigned from 0 - 4 and this score has been used in the analysis. The individual

features were treated as a hierarchy for cases where there was a discrepancy in the degree of the separate features degeneration. Porosity was given the lowest value, osteophytes next, with eburnation/sclerosis given most importance. It was felt that the early granulated appearance of the porosity feature was too easily mimicked in an archaeological sample by post-mortem process so a facet showing possible porosity without any of the other markers present was not graded as having osteoarthrosis. Only once some evidence of osteophyte formation was present was it counted as displaying osteoarthrosis. The presence of eburnation/sclerosis was taken as the most important indicator so that a facet with small osteophytes but a large area of eburnation and/or sclerosis was given a higher score than the osteophyte score alone would give. In general, however, the osteophyte development, porosity of the surface and eburnation/sclerosis were seen progressing together. In both the body joint and the facet joint when the osteophytes had grown to fuse to the opposite joint surface this was graded as 4 in Sager.

This type of scheme of grading the severity of osteoarthrosis and osteophytosis is very similar to those used by other researcher on degenerative joint disease (Jurmain, 1975; Stewart, 1958). The decision of category is in all cases a subjective one, as for the more established aging methods, based as it is on observation rather than measurements.

In the scheme used here, waiting until the appearance

of osteophytes in the facet joints no doubt excludes the earliest stages of osteoarthrosis but for the purposes of its use as a possible aging method for the older age ranges, it was felt this was preferable to possible overestimation of its presence in younger age groups and will provide an easier method for adoption by other anthropologists.

Each facet joint surface was graded so that there are two scores for each joint and four scores for the two joints between any two vertebrae (see Fig. 5.2). The body joint was scored in twelve location: three on the anterior edge of both the superior and the inferior surface and likewise three on the posterior edge of both superior and inferior surfaces (see Fig. 5.3).

In analysis the four facet scores are reduced to one score by taking the maximum score found. Likewise, on the body the anterior and posterior scores are each reduced from six to one by taking the maximum grade observed. In turn these are reduced to one score for each of the three regions of the spine: cervical, thoracic, lumbar; and again reduced to a single score for the whole column.

These composite scores for the spine regions and the whole column are constructed in six ways. These six measures can be arranged in a hierarchical order, each successive measure utilizing more information. The introduction of more information provides a greater range of scores to correlate against the dental age estimates used as the baseline data.

FIG. 5·2 SITES OF FACET JOINT SCORES

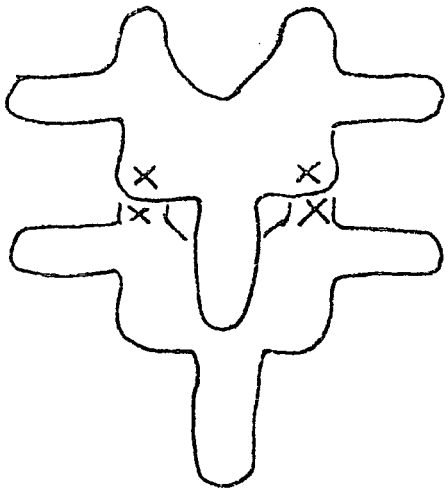
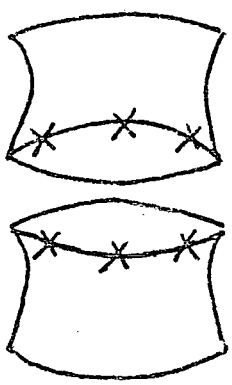


FIG. 5·3 SITES OF DISC JOINT SCORES



The greater information could involve a greater amount of variation from causes other than age, alternatively it may produce a more precise fit with age. Some of the information may duplicate information introduced at an early level in the hierarchy as regards the relation to age and therefore offers no extra advantage.

The six measures of spine degenerative joint disease for the regions and column are as follows, arranged in the hierarchical order of increasing information content.

1. Maximum Severity:

The maximum grade of severity in each of the regions and in the column for the facet and for the disc joints.

2. Combined Maximum Severity:

The maximum severity grade in the regions and column for the facet joints added to the comparable score for the disc joints to give just one score for each of the regions and the column.

3. Extent (%) of Involvement:

The percentage of the facet and of the disc joints displaying some involvemnt of degenerative joint disease in each region and the column.

4. The Combined Extent (%) of Involvement:

As no. 2, the extent score of the facet joints added to the score of the disc joints in each region and the column.

5. Extent (%) x Maximum Severity:

The percentage of facet and of disc joints involved in each region multiplied by the corresponding maximum grade of severity in that region or in the column, a combination

of no. 1 and no. 3.

6. Combined Extent (%) x Maximum Severity:

The measures of no. 5 for facet and disc joints added to each other in each region or in the column.

The information available is, therefore, severity and extent of involvement.

In an archaeological sample, there is often substantial amounts of missing data, in the form of missing vertebrae. To calculate the measures for the individual joints and for the composite regions and column, a minimum number of values is specified as necessary to make a valid score. For each of the individual facet or disc joints there had to be at least two of the possible four or six contributing values present. For each of the regional scores, there also had to be at least two of the individual joint scores available, and for the column scores, at least seven.

Examples of the grades of severity of degenerative joint disease in the facet and disc joints are shown in Figs. 5.4 and 5.5 respectively. Vertebral body porosity is illustrated in Fig. 5.6.

Additional figures illustrate the distinctive beak-shaped osteophytes of the disc joints when at the most severe development in Fig. 5.7, an example of fusion of the anterior ligaments as found in DISH disease, Fig. 5.8 and examples of an extra sacral segment, Fig. 5.9 and a

FIG. 5.4 STAGES OF SEVERITY OF OSTEOARTHROSIS

A IN THE ATLAS

B IN THE AXIS

C IN THE REST OF THE SPINE

GRADESO

1

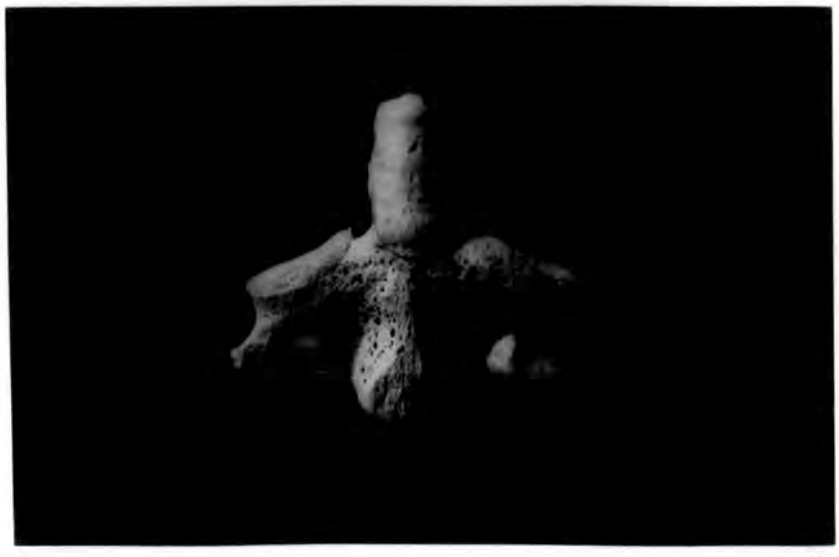
A

2

3

B GRADES O

1

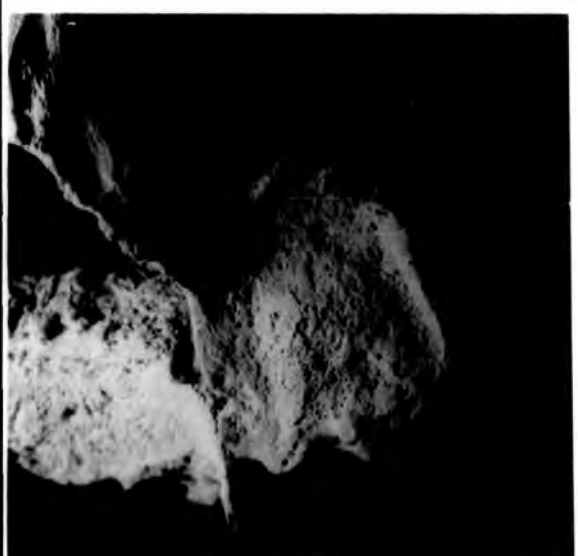


2

C

GRADES O

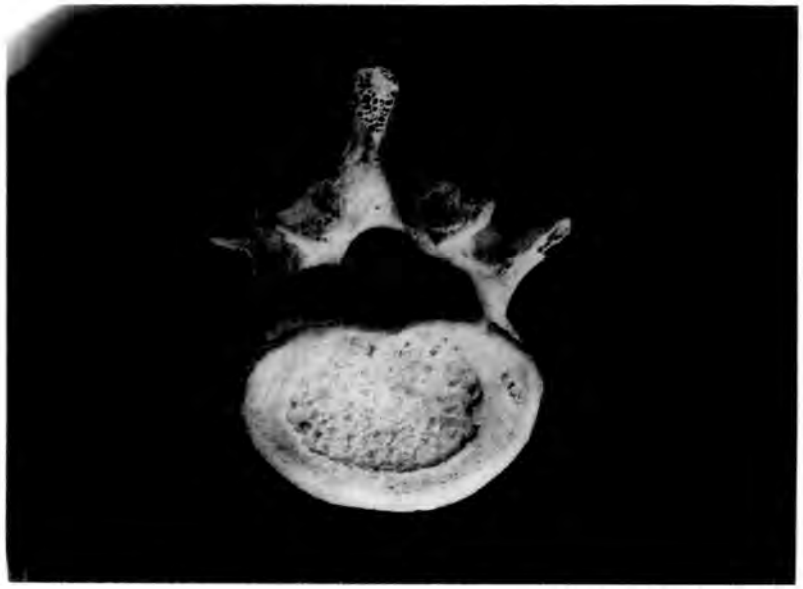
1



2

3

FIG. 5·5 STAGES OF SEVERITY OF OSTEOPHYTOSIS.

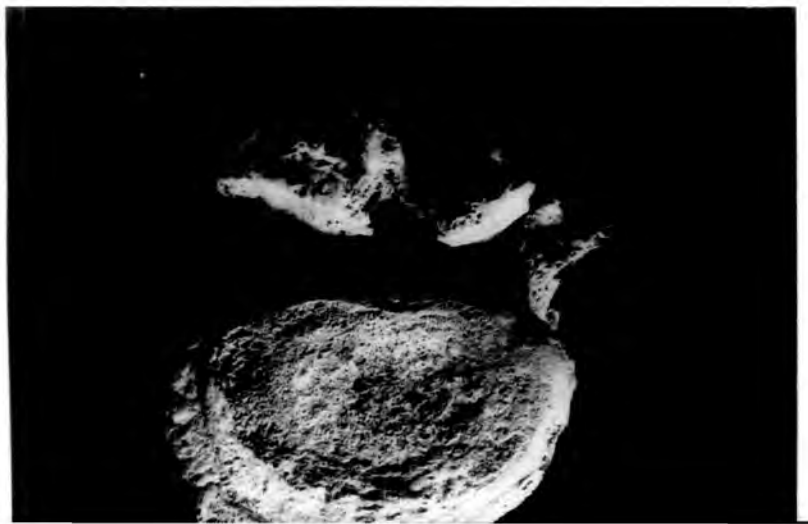


GRADES 0

1

2

3



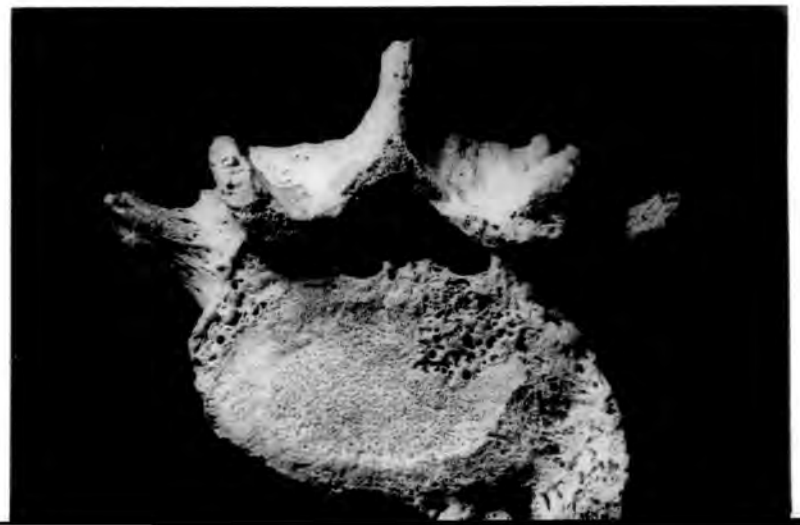
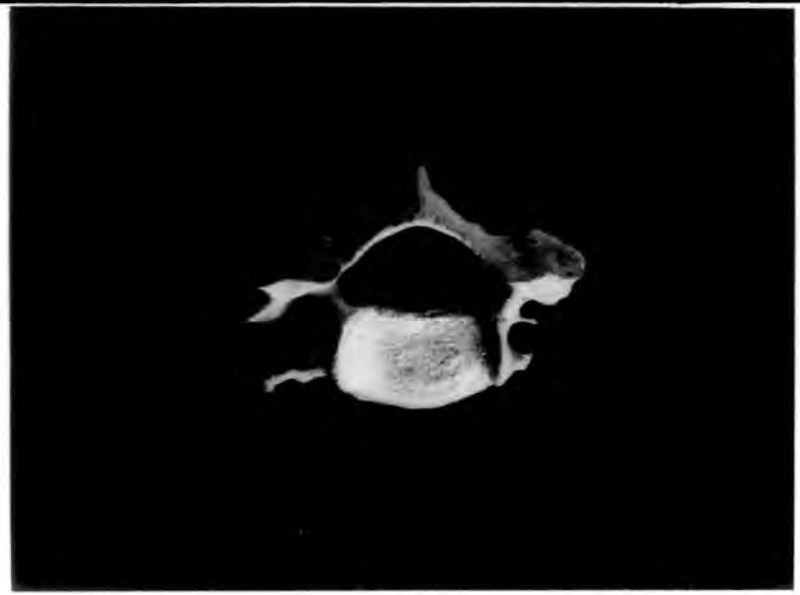
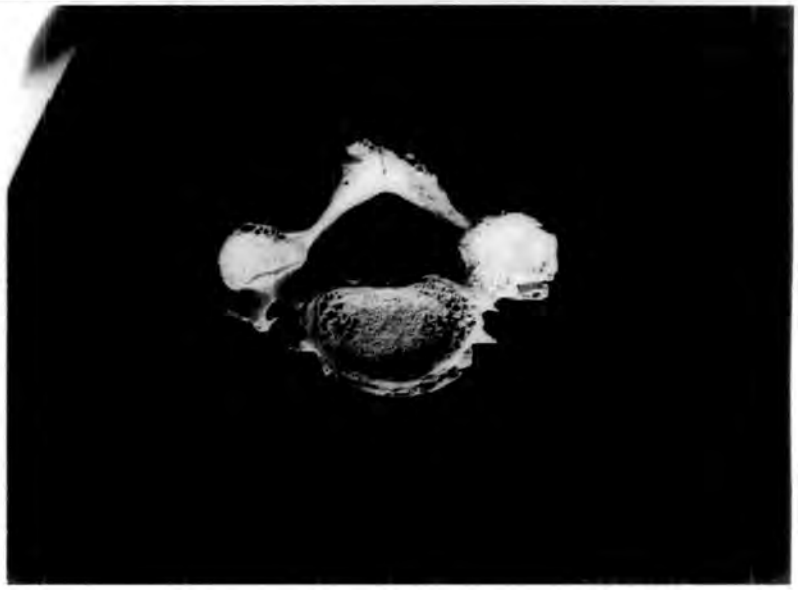


FIG. 5.6 STAGES OF VERTEBRAL BODY POROSITY



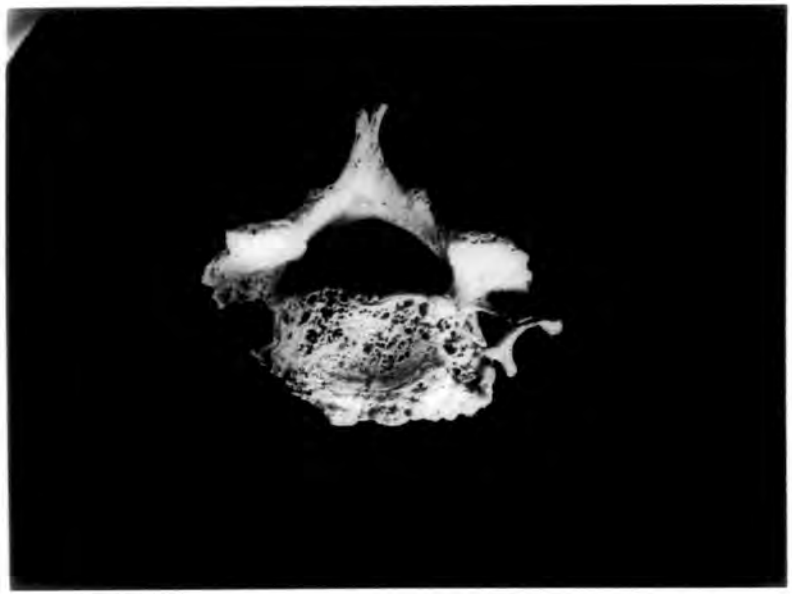


GRADES 0

1

2

3



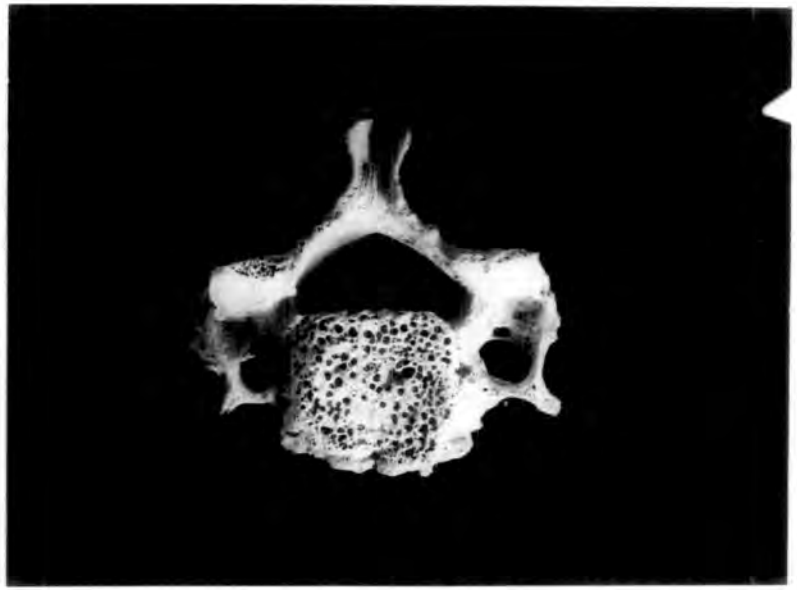


FIG. 5•7 BEAK SHAPED OSTEOPHYTES.

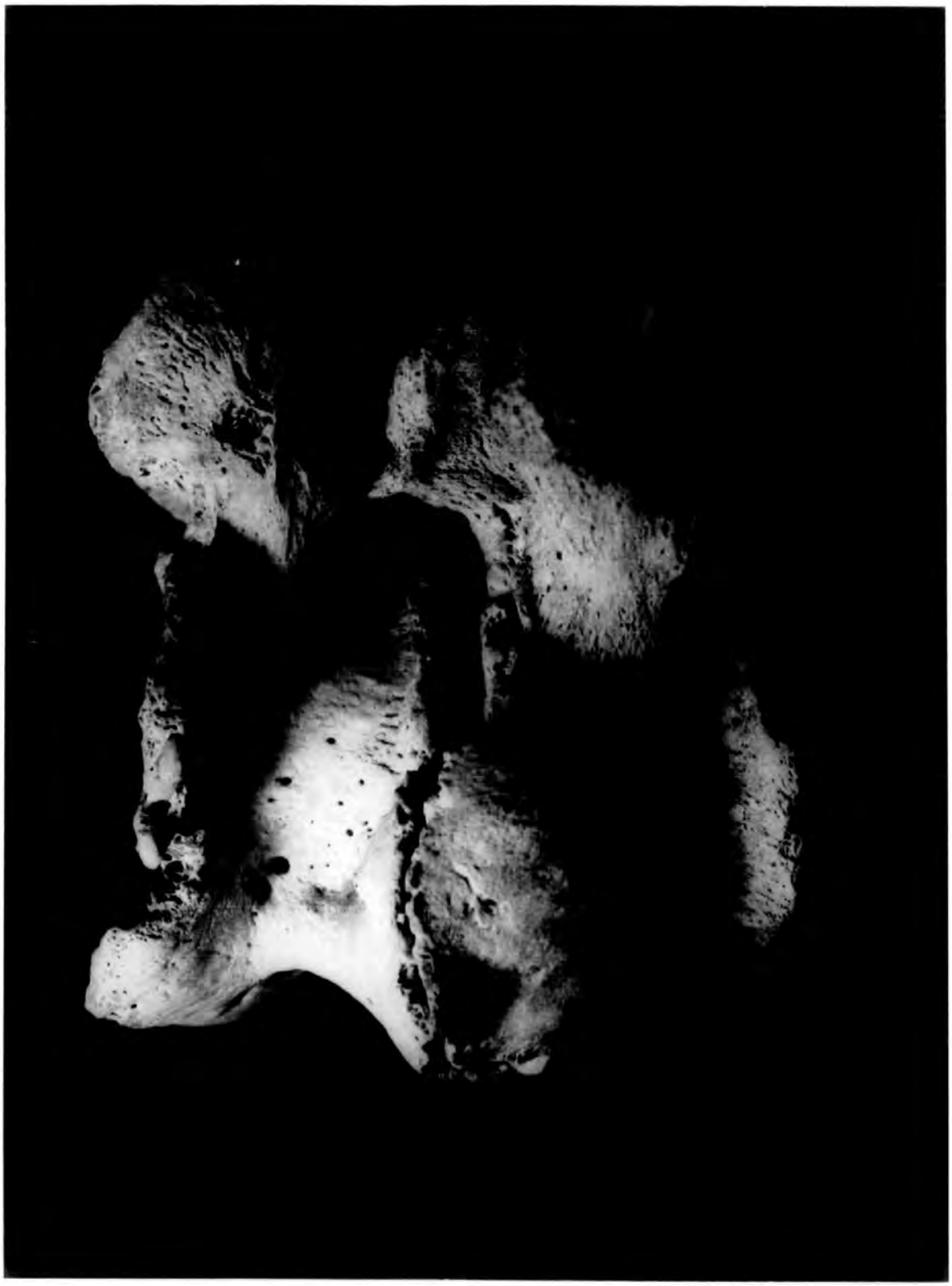


FIG. 5.8 FUSED VERTEBRAE FROM DISH DISEASE.

FIG. 5·9 AN EXTRA SACRAL SEGMENT.

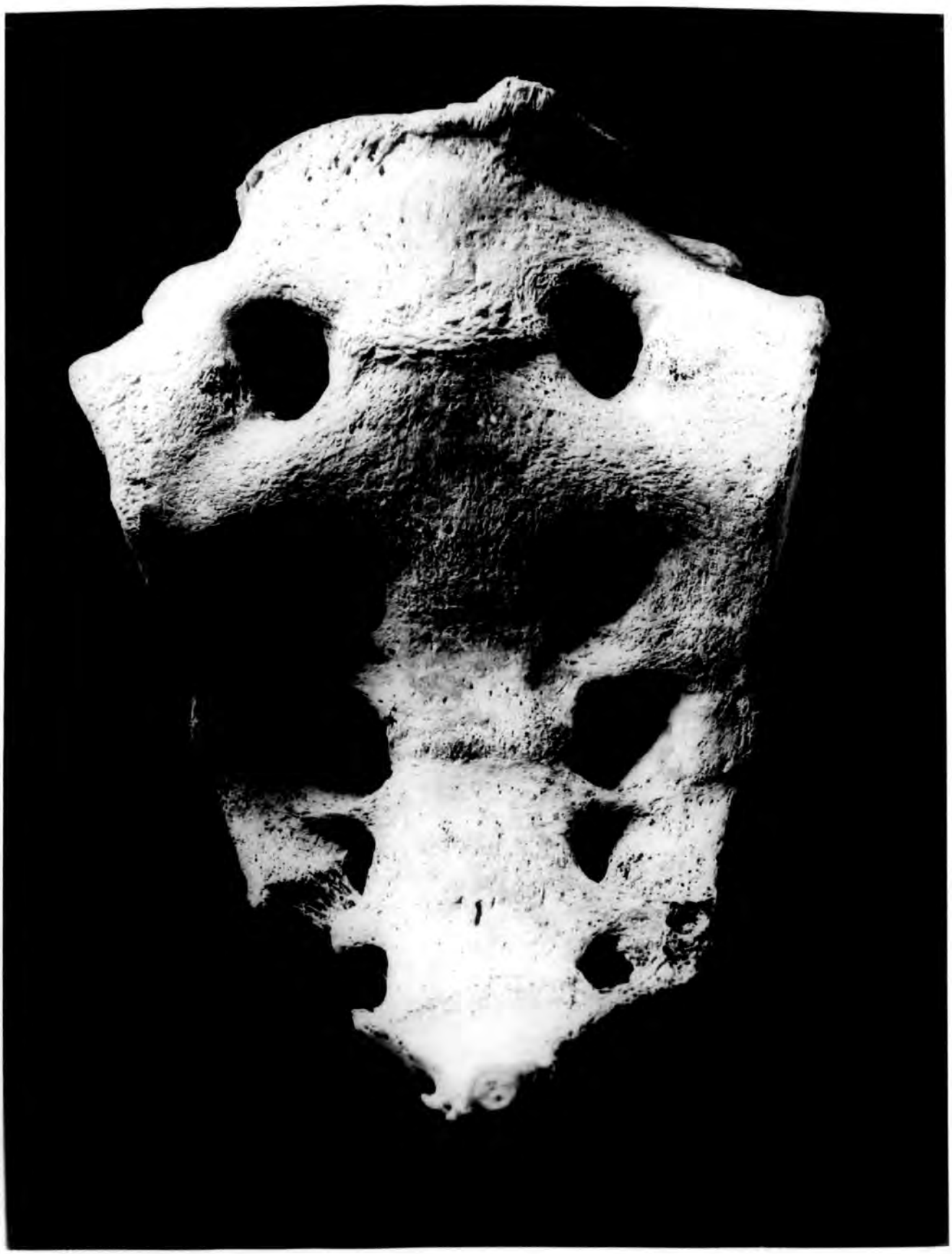
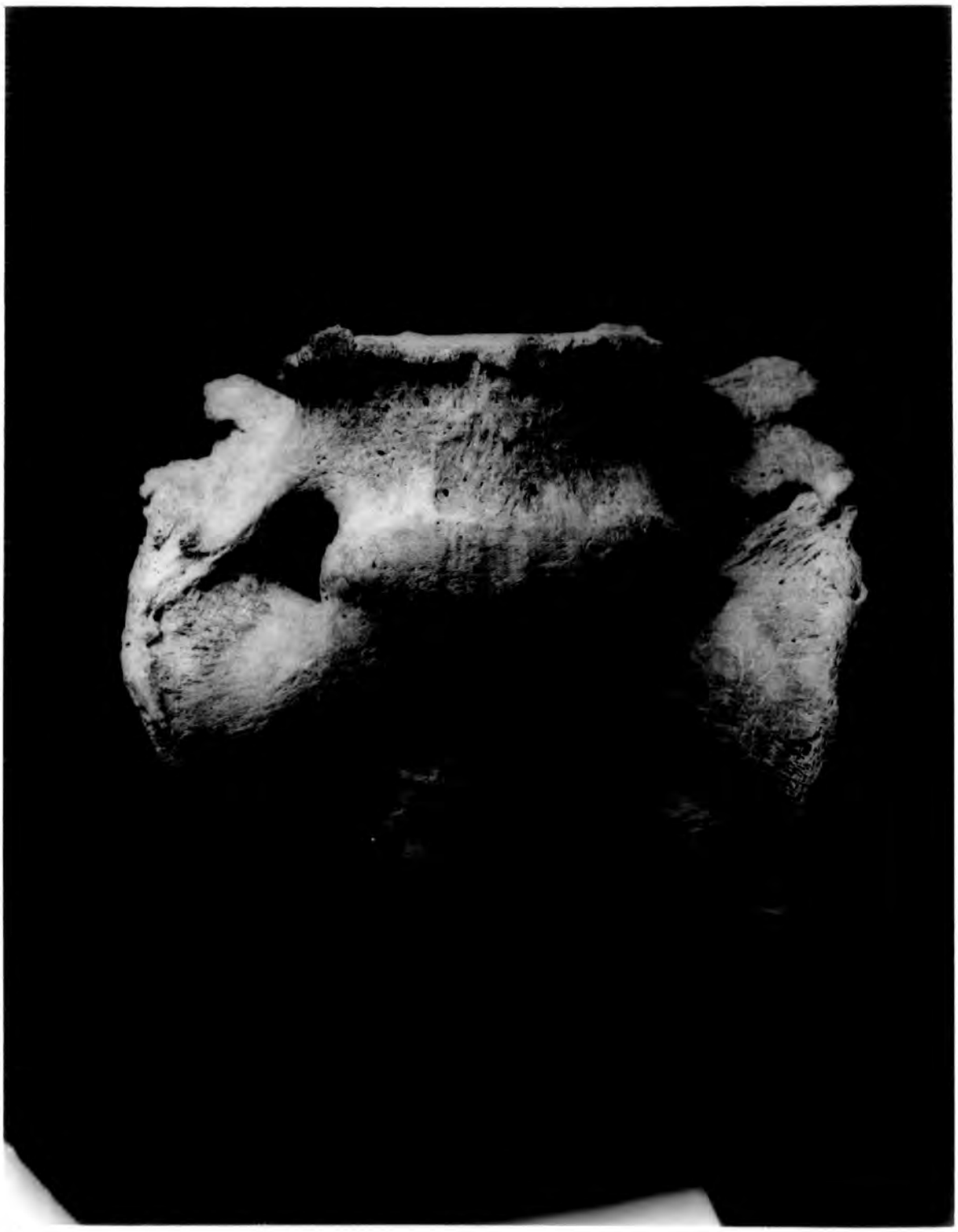


FIG. 5.10 SACRALIZATION OF THE LOWEST LUMBAR VERTEBRA

A ANTERIOR VIEW

B POSTERIOR VIEW



A

B

sacralized lumbar in Fig. 5.10.

5.5 Analysis

All the data has been put onto the IBM computer at Durham University. The analysis was performed using the Statistical Package for Social Sciences, edition 10 (SPSSX). The analyses performed were kept to simple tests of correlation and difference. Nonparametric tests were used throughout.

Statistics used to examine difference were the χ^2 and the Mann-Whitney U test. The χ^2 test was used on data where only a few categories were involved. Specifically, this was in investigations of sex differences in distribution of severity in the individual joints and in the composite regional and columnal measures. The combined scores of maximum severity, the extent measures and the extent x maximum severity measure were assessed for sex differences in distribution using the Mann-Whitney U test. This test was also used with the microages and microparameters, the dental attrition scores and the pubic symphysis metamorphosis scores. The choice of tests was largely determined by those available in the SPSSX package. The χ^2 test given with crosstabulations, necessitates an absolute frequency of number of cases being greater than five in each of the tabular cells, which is not found in the sex distributions of the combined maximum severity scores, nor, of course in those measures using continuous scales.

has been subdivided by age groups and by sex in tests for sex differences in each age group. In these cases, where smaller sample sizes are involved, significance is accepted as $p < 0{\cdot}05$.

No cases have been excluded from the analysis on the grounds of gross pathology etc. This was decided since it is wished to assess the applicability of aging methods throughout an entire sample, regardless of pathologies. The study of palaeopathology itself needs good age estimations of the individuals concerned. Of particular importance here are cases of joint fusion in the spine from causes other than strictly degenerative joint disease. These occur mainly in the thoracic region and represent ossification of ligaments. The photograph in Fig. 5.8 is an example of such a case. Individuals with severe cortical bone loss are less likely to be involved since sections are not easily made from brittle, porous bone. The external measure of cortical thickness will have been taken, but internal examination will not have been possible.

The bulk of the tables and figures have been presented in appendices at the end of the thesis, since, with so many variables studied, a large amount of data is necessarily generated. Therefore, only the most pertinent have been selected to illustrate the text in the chapters of results.

Correlation was tested in all cases using Spearman's statistic for rank correlation, r_s. This test compares the two sets of data by placing the cases in rank order for each variable and comparing the rank. Therefore, measures may be seen to move in the same direction together even though absolute values are very different. This is appropriate for the estimates of age used here, since different methods may provide very different absolute ranges of age estimations, but otherwise have a close relationship. Since setting absolute age values is very problematic with skeletal remains, a relative relationship is what must be first examined. The mean values, standard deviations and coefficients of variation are given for most of the distributions. The coefficient of variation is calculated by dividing the standard deviation by the mean value.

All of the univariate and bivariate analyses used are described in Siegel (1956) and Blalock (1972).

In addition to these basic tests, a factor analysis was performed for the purposes of summarizing the relationships. The axes used in the factor analysis are kept at right angles to one another and rotated in multi-dimensional space to obtain the greatest differentiation of grouped variables (Williams, pers. comm.).

The level of significance to be accepted was set at $p < 0.01$, in the majority of tests because of the large sample size. The exceptions are instances where the sample

Statistics Used

X^2 Test

The X^2 statistic tests in differences between two samples when the values are arranged in discrete categories. The X^2 value itself gives no indication of the direction of any observed difference between the samples. The frequencies for each category are arranged in so-called contingency tables.

	Sample A	B	Total
Value P			
Q			
R			
Total			

The best estimate of the expected values in each cell of the table can be calculated from the total frequencies of the rows and columns.

The observed value is then compared with the expected for the entire set of cells and the X^2 value calculated by the formula

$$X^2 = \frac{E(O-E)^2}{E}$$

O = Observed
E = Expected

The limitation on the use of X^2 is the frequency in each of the cells. This will distort the statistic if below a certain level. This level is set by convention as an absolute frequency of 5. The statistic must be used on frequencies not percentages.

Mann-Whitney U Test

The Mann-Whitney U statistic tests for differences in the distributions of two samples by arranging the values of the two samples in rank order. The U value

is the sum of the number of values of sample A which precede each value of sample B.

The null hypothesis is that the values of the two distributions are randomly intermixed on this rank ordering and the distribution of U values under the null hypothesis is known.

Spearman's rs

The Spearman rank correlation coefficient similarly acts on ranking the data. In this case, it is two measures for the same sample which are to be compared for correspondence. The cases are ranked by score for each measure and the sum of the squares of the differences in rank calculated (Edi^2) rs is computed from the formula

$$rs = 1 - \frac{6(Edi^2)}{N^3-N}$$

Statistics books commonly provide tables giving the critical values of X^2, U or rs for given sample sizes when $p<0.05$ or $p<0.01$. The SPSSX package simultaneously computes the exact p value as part of the statistical calculation which is given here.

Factor Analysis

Factor analysis can be used for two purposes in the biological sciences. First, to resolve complex interrelationships into the interaction of fewer and simpler factors and secondly, for the identification and isolation of causal factors behind biological correlations. In this study where there are a large number of variables, the factor analysis provides a simple way of summarising the correlation relationships found between individual pairs of variables in the bivariate analyses. The factor analysis starts by constructing a correlation matrix of all the variables involved. Covariation is then expressed in terms of underlying factors and correlation coefficients express correlation of each variable with such a factor. These factors explain various proportions of the variance and covariance of the variables.

There are various methods for the extraction of factors from the correlation matrix and these are obviously time-consuming to perform by hand. Thus, the performance of factor analyses is very much a child of the computer era. The SPSSX package for factor analysis was used here, setting the axes at right angles one to another to be rotated in the hypothetical multidimensional space in which the variables are plotted. This positioning of the axes serves to maximise the differentiation of the factors (Williams, pers.comm.).

The correlation coefficients of each variable with each factor are presented in the appendix and the groupings of the variables by factor discussed. This summary thus serves both to present the bulk of the data set in the form of simpler factors and suggests possible causal agents behind the correlations through the grouping by factors.

Sample Sizes

The sample sizes for each of the various spine and bone structure measures are given both as the absolute sample size by sex and by dental age. The sexing of the population indicates a larger proportion of females so long as the 91 unknowns are randomly from both sexes. The sample sizes of the measures reflect this, having a larger proportion of females.

The distribution of sample sizes of both the spine and bone structure measures similarly show this pattern. It is felt, therefore, that the samples of the spine and bone structure measures do come randomly from each age-sex subgroup. The sample sizes are large compared to those of other studies (Kerley; Ahlquist and Damsten; Thomson), with the exception of KAN against dental age. The sample values may therefore not accurately reflect the underlying population values.

Sample Sizes

	Female	Male	JUV
Sex	383	354	400
Unknown	91		
Dental age	250	226	
Pubic symphysis age	142	174	
KAO	149	137	
KAF	68	66	
KAL	76	61	
KAN	27	15	
CLAGE	164	141	
ADAGE	144	133	
TAA	144	132	
TAB	144	132	
TAC	141	128	
TAO	139	126	
TAE	136	126	
Cerv. FACETS	253	234	
Tho	282	268	
Lumb.	294	271	
Column.	279	271	
Cerv. Disks	224	216	
Tho	242	240	
Lumb.	239	226	
Column.	240	238	
Cerv. Combined	220	213	
Tho	239	236	
Lumb.	235	221	
Column.	235	236	
Cortical Thickness	247	217	

Numbers against Dental Age

	Female	Male
KAO	134	94
KAF	53	45
KAL	64	46
KAN	25	10
CLAGE	125	99
ADAGE	110	91
TAA	110	91
TAB	110	91
TAC	108	88
TAD	108	88
TAE	108	87
Cerv. Facs	181	166
Tho	193	181
Lumb.	207	181
Column.	194	183
Cerv. Discs	157	154
Tho	168	159
Lumb.	169	152
Column.	165	160
Cerv. Combined	156	152
Tho	165	159
Lumb.	169	149
Column.	162	160

Chapter 6. RESULTS: DENTAL ATTRITION AND PUBIC SYMPHYSIS METAMORPHOSIS.

6.1 Mortality Distributions.

The distributions of age at death, as assessed from dental attrition and pubic symphysis metamorphosis are illustrated in Fig. 6.1, with the sexes apart. Dental age gives a modal value of age at death of 25 - 35 for both sexes, whereas pubic symphysis age gives the same modal value of 25 - 35 for males, but a far higher value of 45+ for females.

The distributions for the two sexes are compared using the Mann-Whitney U test and the values given in Table 2 of Appendix A. Significant differences are found between the sexes from both mortality distributions. The age assessments from dental attrition result in a greater number of males in the older categories. The reverse is the case in the distributions produced from pubic symphysis age, females having a far greater proportion than males in the older categories. The two methods therefore produce very different pictures of mortality in Poundbury.

Some correspondence between the two exists as the Spearman rank correlation statistic shows a significant correlation for both sexes between the two methods (see

FIG. 6·1 DISTRIBUTION OF AGE AT DEATH FROM DENTAL ATTRITION AND PUBIC SYMPHYSIS METAMORPHOSIS.

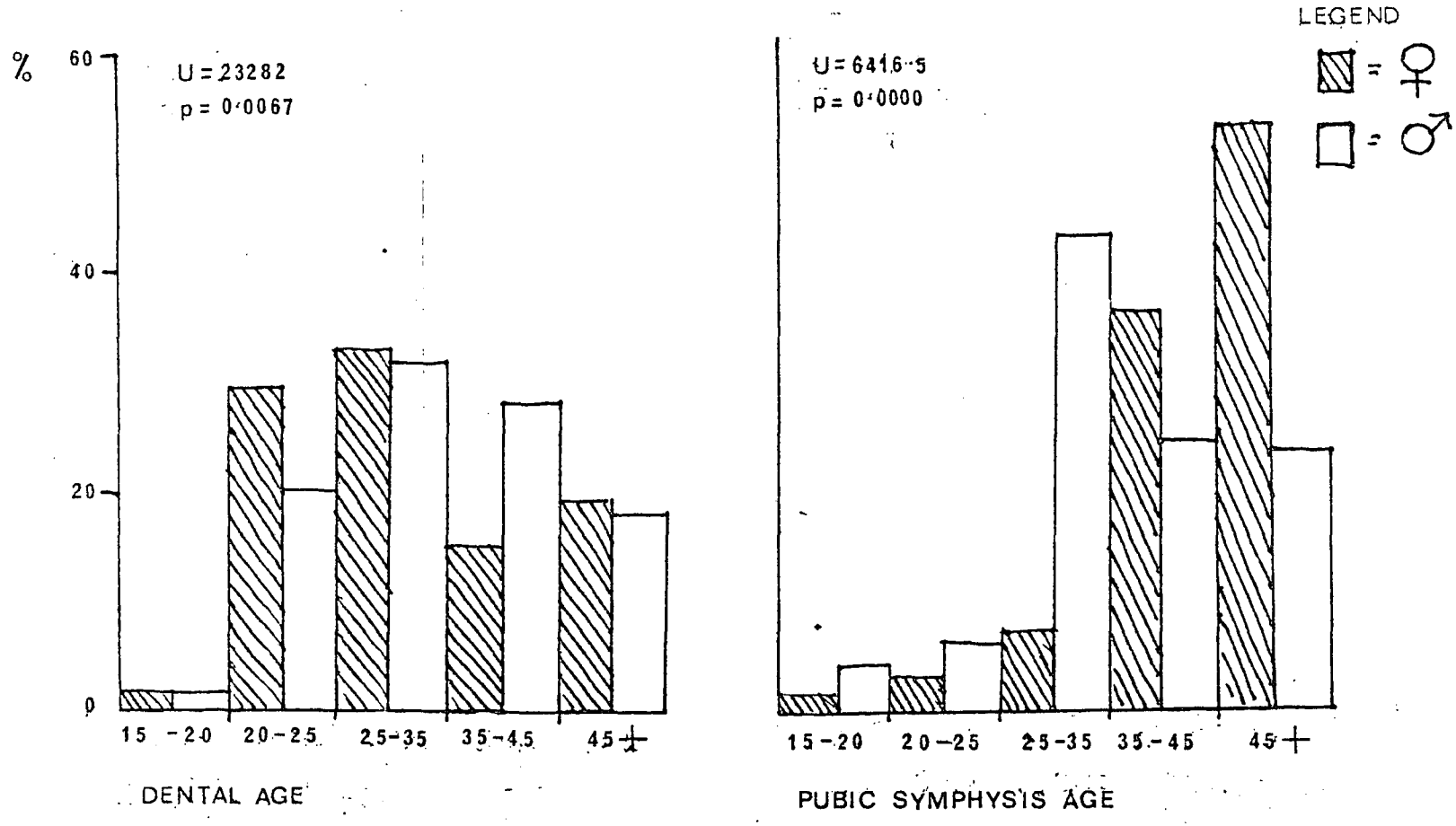


Table 3 of Appendix A). This correlation is lower for females than males. This implies attrition and pubic symphysis metamorphosis progress linearly together, but some acceleration of pubic symphysis metamorphosis in females occurs in relation to the rate of dental attrition. Alternatively, the conversion from either the score of the metamorphosis or the score of dental attrition to an estimation of age is producing a distortion in the mortality pattern.

6.2 Dental Attrition.

The distributions of attrition scores of each molar are given in Fig. 6.2. A significant difference between the sexes is found for all three molars using the Mann-Whitney U test. The U and p values are given in Table 5 of Appendix A. The difference found is, of course, the same as the dental age sex difference based on these scores, with the males showing a greater proportion in the categories of more severe wear. The difference between the sexes in attrition scores and, subsequently in the mortality pattern, can either arise from an unequal rate of wear on the teeth or from a real difference in mortality patterns. Dietary differences between the sexes could easily have existed producing an unequal rate of wear.

The difference between the scores of M_1 and M_2, between M_2 and M_3 and between M_1 and M_3 are calculated and the distributions given in Table 6 of Appendix A. The U and p values of the Mann-Whitney U test for sex differences are

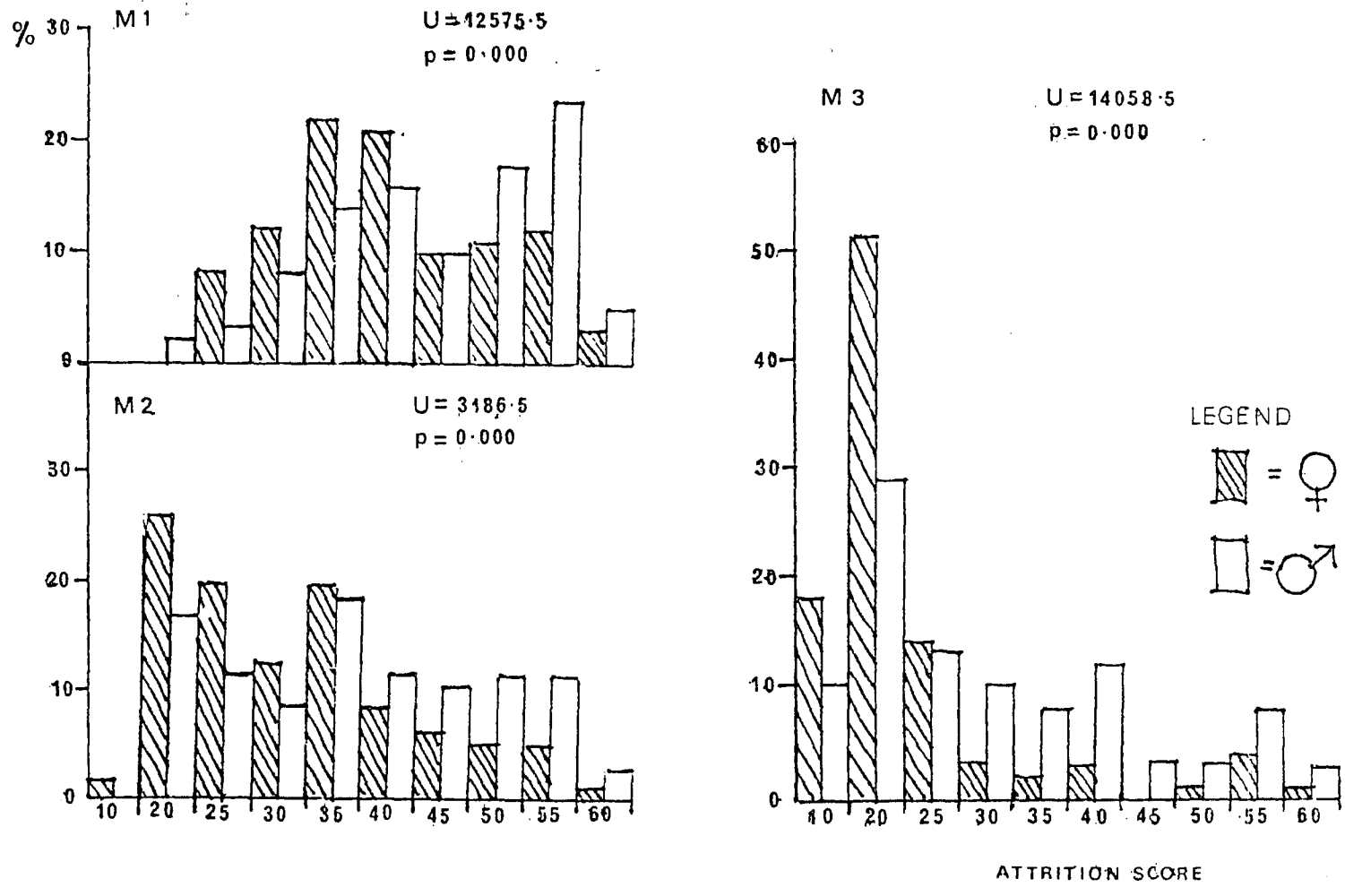


FIG. 6·2 % FREQUENCIES OF THE MOLAR ATTRITION SCORES.

given in Table 7 of Appendix A. None is found to be significant. However, it would also not be true to dismiss the possibility of different rates of wear altogether since a trend is indicated which is significant at $p<0{\cdot}05$, whereby females have a greater difference in wear than males between M_1 and M_2 and M_1 and M_3. This suggests the possibility of a longer interval between the eruption of M_1 and M_2 or M_3 in females than in males. It is difficult to assess if this would affect the categorization into dental age groups. Females could possibly have been placed in a group older than a male of equal age on the basis of the wear on M_1, or in a younger one on the basis of M_2 and M_3. Since the difference is not significant at the level stipulated for acceptance here, and because errors in dental age assignment could have been in either direction, it will be taken here that no differences exist between the sexes in the relative rates of wear on each of the teeth. It may be that one of the sexes experiences more severe wear at a constantly greater level than the other.

6.3 Pubic Symphysis Metamorphosis.

A true comparison of the sexes by the scores of pubic symphysis metamorphosis is not really possible as both the scales and conversion to an age estimate are drawn up separately for each sex. However, the same aspects of the pubic symphysis are examined and the same progression of changes of each of the three components so some rough level of comparison may be made. The frequency distribution of the

pubic symphysis scores in Fig. 6.3 displays the bulk of the population in the higher scores, supporting the implication of the method that it does not age well beyond 40, and a greater proportion of males are in the higher scores than females. It has been shown that this relationship of the sexes is reversed once conversion to age estimates is made. This illustrates the need for a comparable scoring system for the two sexes to assess real sex differences in the rates of metamorphosis.

6.4 Dental Attrition and Pubic Symphysis Age.

The median value of dental attrition scores are plotted for each category of pubic symphysis age in Fig. 6.4. For each tooth, males show a higher median grade of attrition in each age group. If the pubic symphysis age were over-estimating the females, the female distribution of median values could be moved one category to the left. A slightly greater attrition value would still exist for males. This implies a greater rate of attrition in males. However, the pubic symphysis aging is thought to be less reliable than dental attrition aging, particularly in females so this indication is inconclusive.

6.5 Pubic Symphysis Metamorphosis and Dental Age.

The median grade of pubic symphysis metamorphosis for each dental age group is illustrated in Fig. 6.5. No difference is found between the sexes. Again as stated earlier,

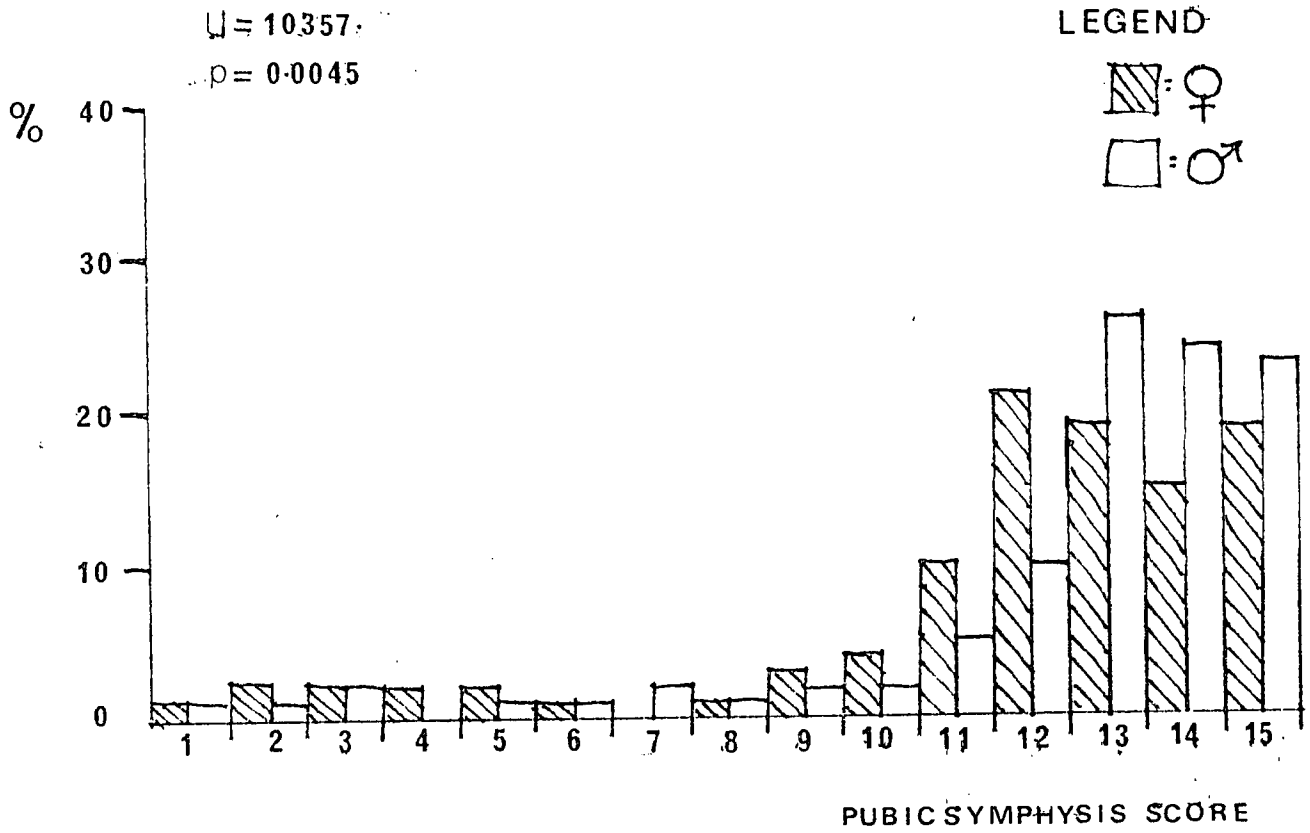


FIG. 6·3 % FREQUENCY OF THE PUBIC SYMPHYSIS SCORE.

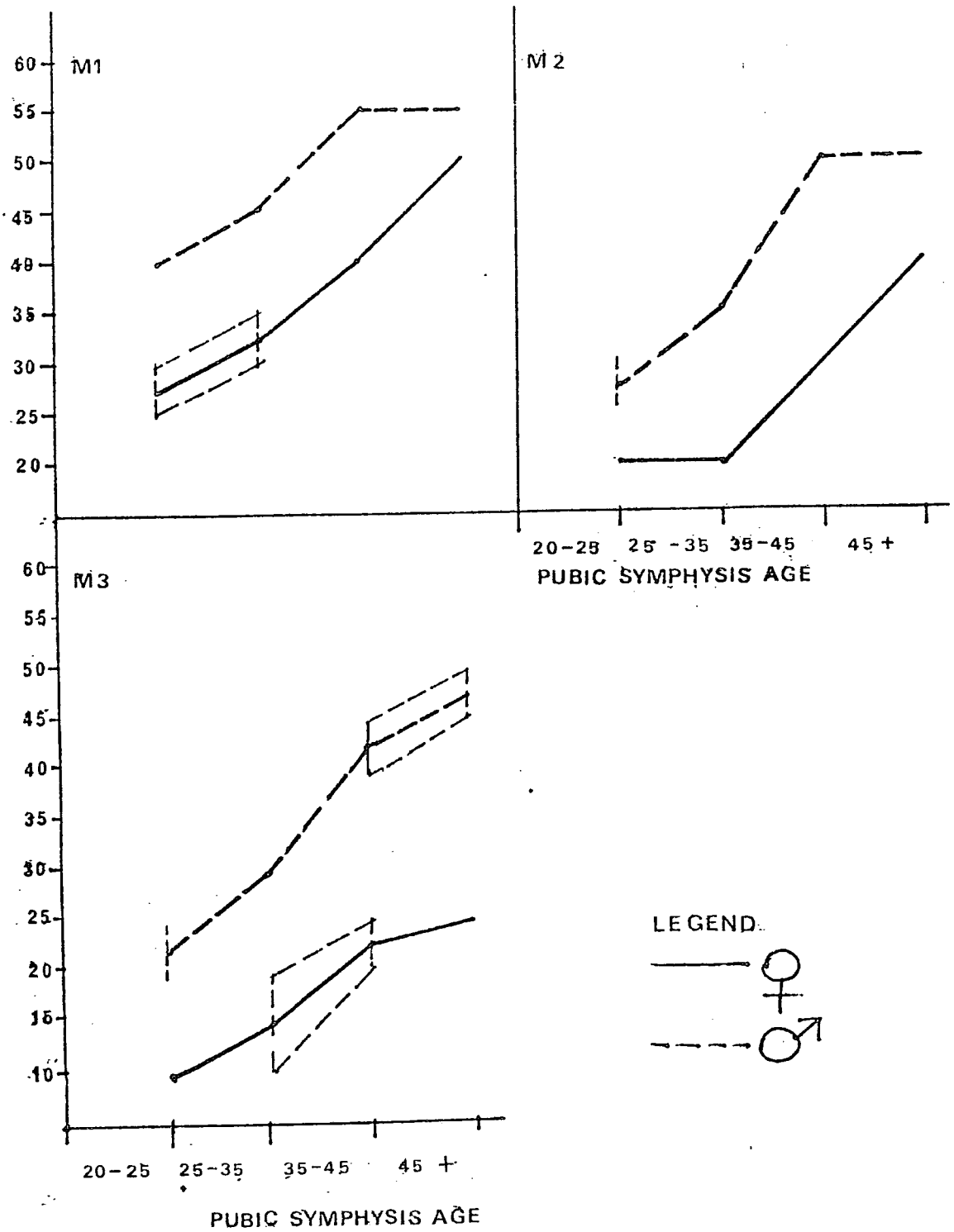
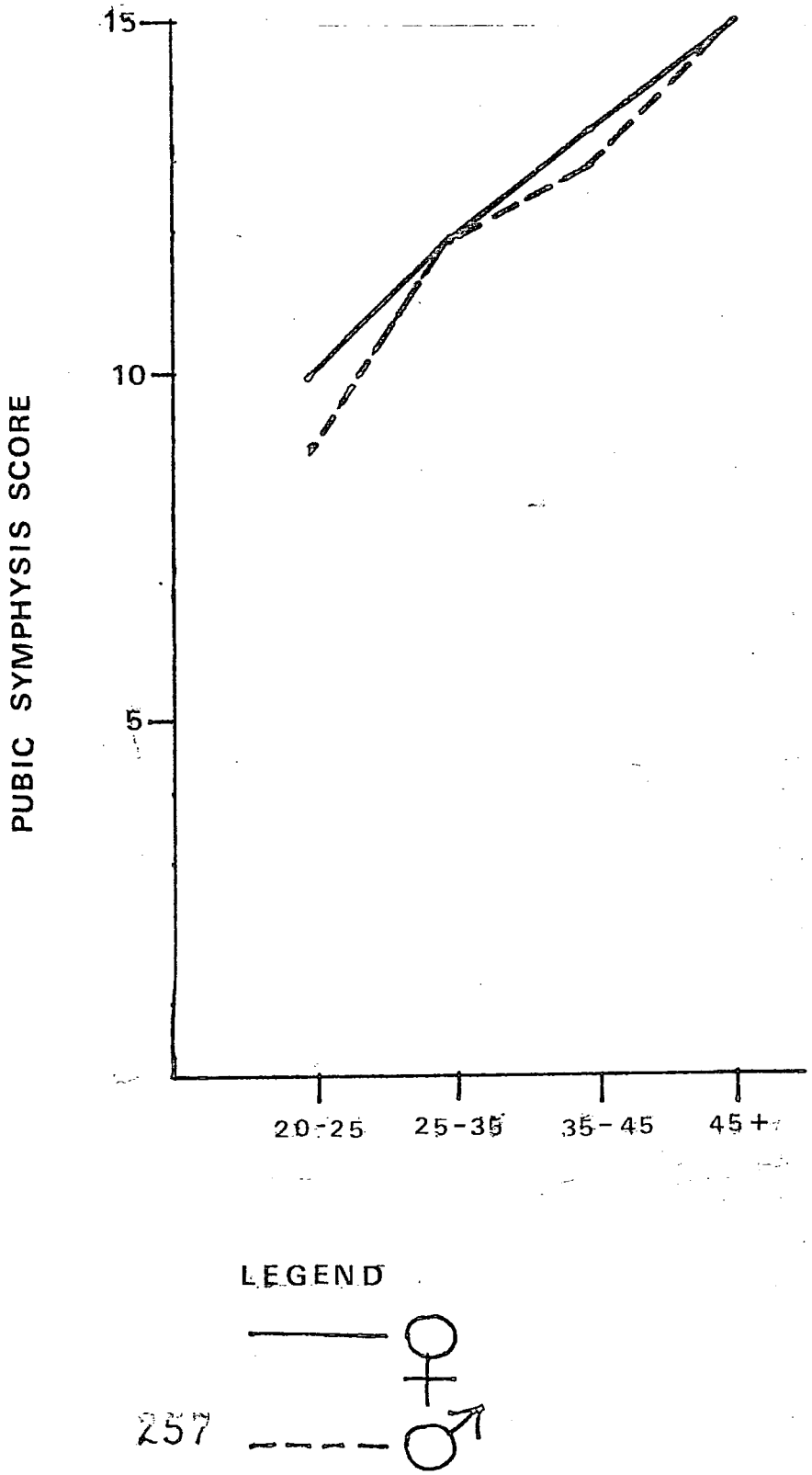


FIG. 6·4 MEDIAN VALUES OF THE MOLAR ATTRITION SCORES IN EACH DENTAL AGE GROUP.

FIG. 6·5 MEDIAN VALUES OF THE PUBIC SYPMPHYSIS SCORE IN EACH DENTAL AGE GROUP.



the conversion to real age changes this, the implication being that possibly the female pubic symphysis metamorphoses more slowly. This is partly an artefact of the different reference samples, but the descriptions of the stages of metamorphosis proceed to the commencement of break down in both sexes suggesting that this final stage is reached equally in the younger male sample as in the broader age range of the female sample.

6.6 Pubic Symphysis Metamorphosis and Dental Attrition.

All the tooth wear scores correlate very highly with each other and also with the score of pubic symphysis metamorphosis in both sexes. The coefficients and p values are given in Table 15 of Appendix A.

Summary.

The mortality distribution from dental age depicts a greater proportion of males in older age groups than females. The distribution from pubic symphysis age shows the reverse pattern. Nonetheless, the progression of attrition and pubic symphysis metamorphosis move linearly together.

There is a suggestion that dental attrition may proceed at a faster rate in males than females but the evidence is inconclusive. Females have a tendency for greater wear on M_1 which may be from a greater interval in eruption time between M_1 and the other molars.

Aging from the pubic symphysis is felt to be less reliable since the methods of each of the sexes are not totally comparable. As far as they can be compared, it seems that the conversion from the raw score to age estimate may overage females. However, the correlation between dental age and pubic symphysis score is also better for males than females, so the actual scoring system may also be unreliable for females. These indications are merely speculative, not conclusive.

Dental age will be used as the baseline of age in the Poundbury population for the examination of bone structure and degenerative joint disease of the spine.

Chapter 7. RESULTS: BONE STRUCTURE.

7.1 Frequency Distribution of Age Estimates.

The distributions of age at death for each of the eleven methods except TAD are given in Fig. 7.1. None shows any significant differences between the sexes in distribution using the Mann-Whitney U test. This would indicate equivalent mortality profiles between the sexes. It can be seen that the majority encompass a wide range of age estimates some of which are impossible, well over 100 or minus scores. This was particularly true of TAD which had a large proportion of the estimates below 0, so was not illustrated here. No cases have been excluded on the grounds of pathology etc. and the most extreme values presumably represent extreme outliers in structural measurements from such causes.

The distributions for KAF, KAL and KAN are rather uneven. KAN is based on a very small sample size of 27 female and 15 male only. KAL and KAF indicate a more even distribution of age at death than any of the other age spreads.

None of the age distibutions indicates a large number of childhood deaths. This is in part a sampling problem as the small fragile bones of the infant and child are both less well preserved and less easily ground down into thin sections

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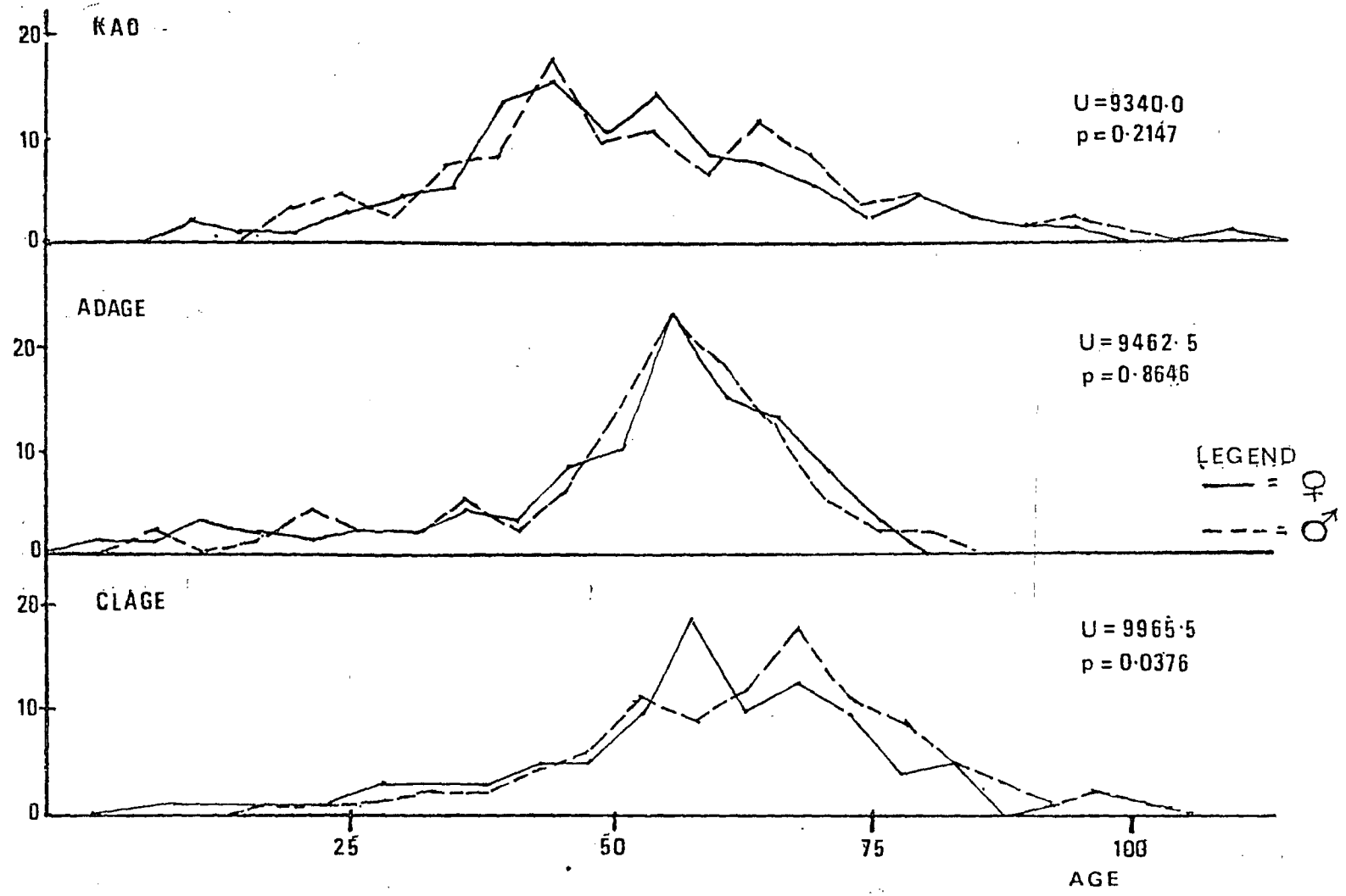
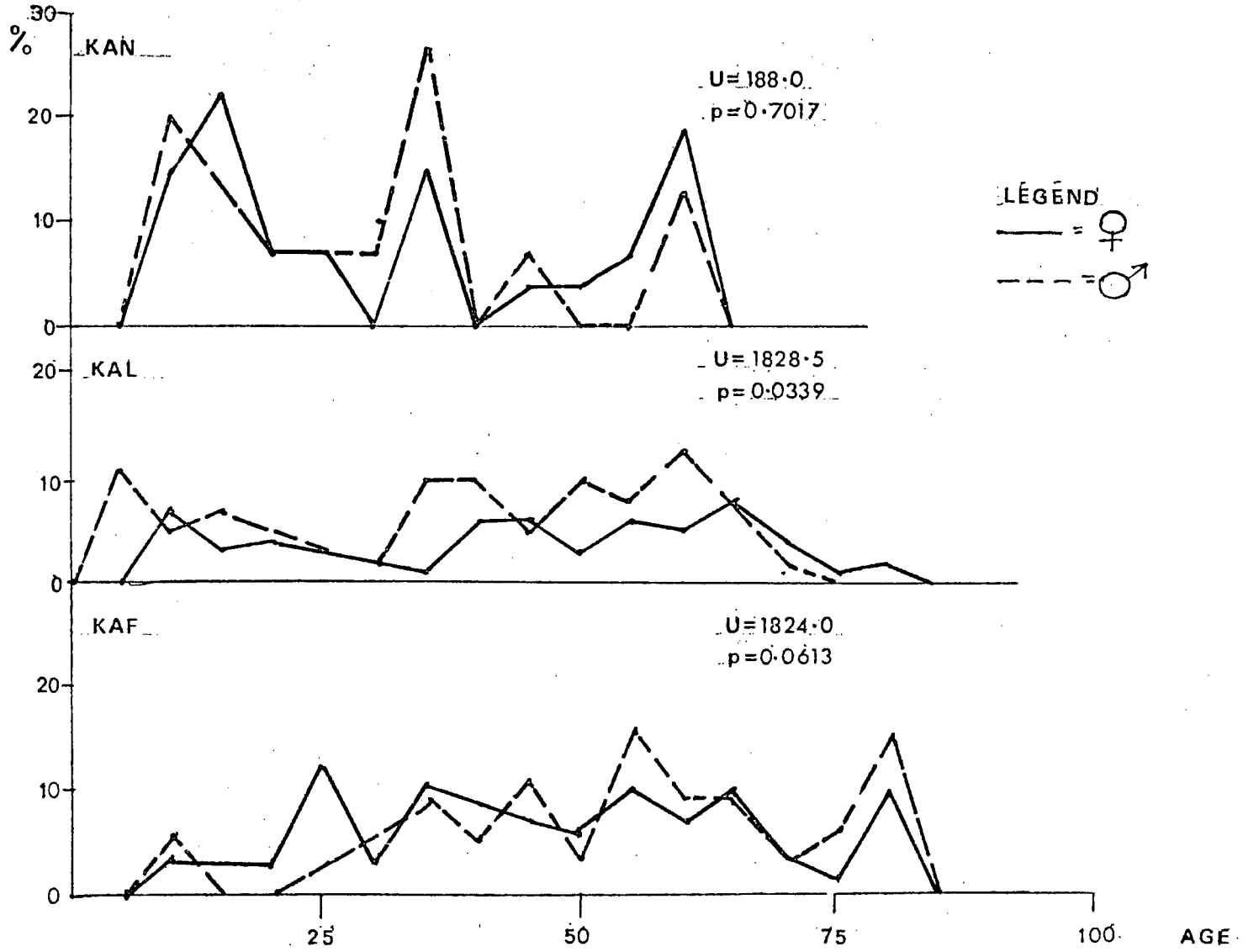
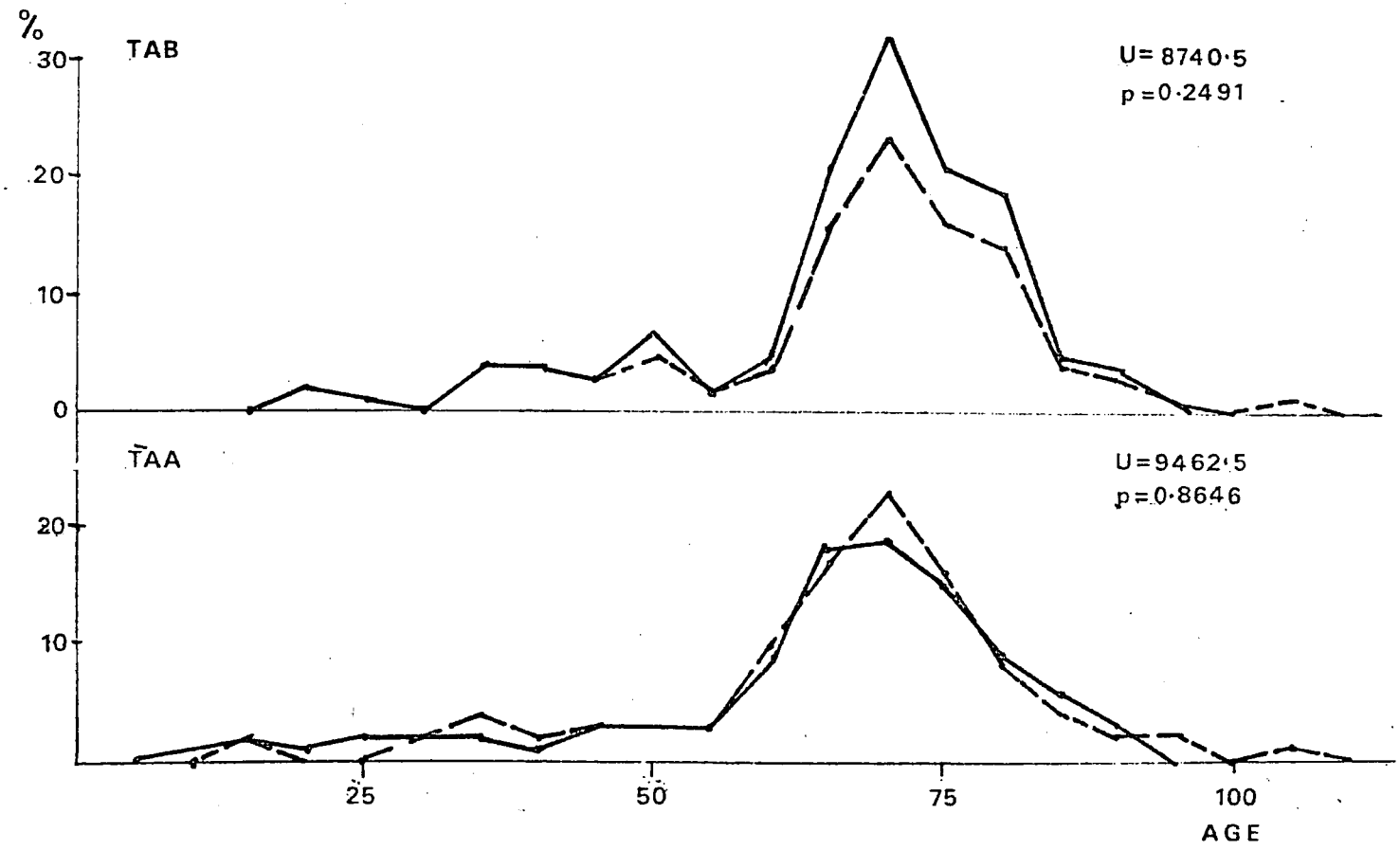
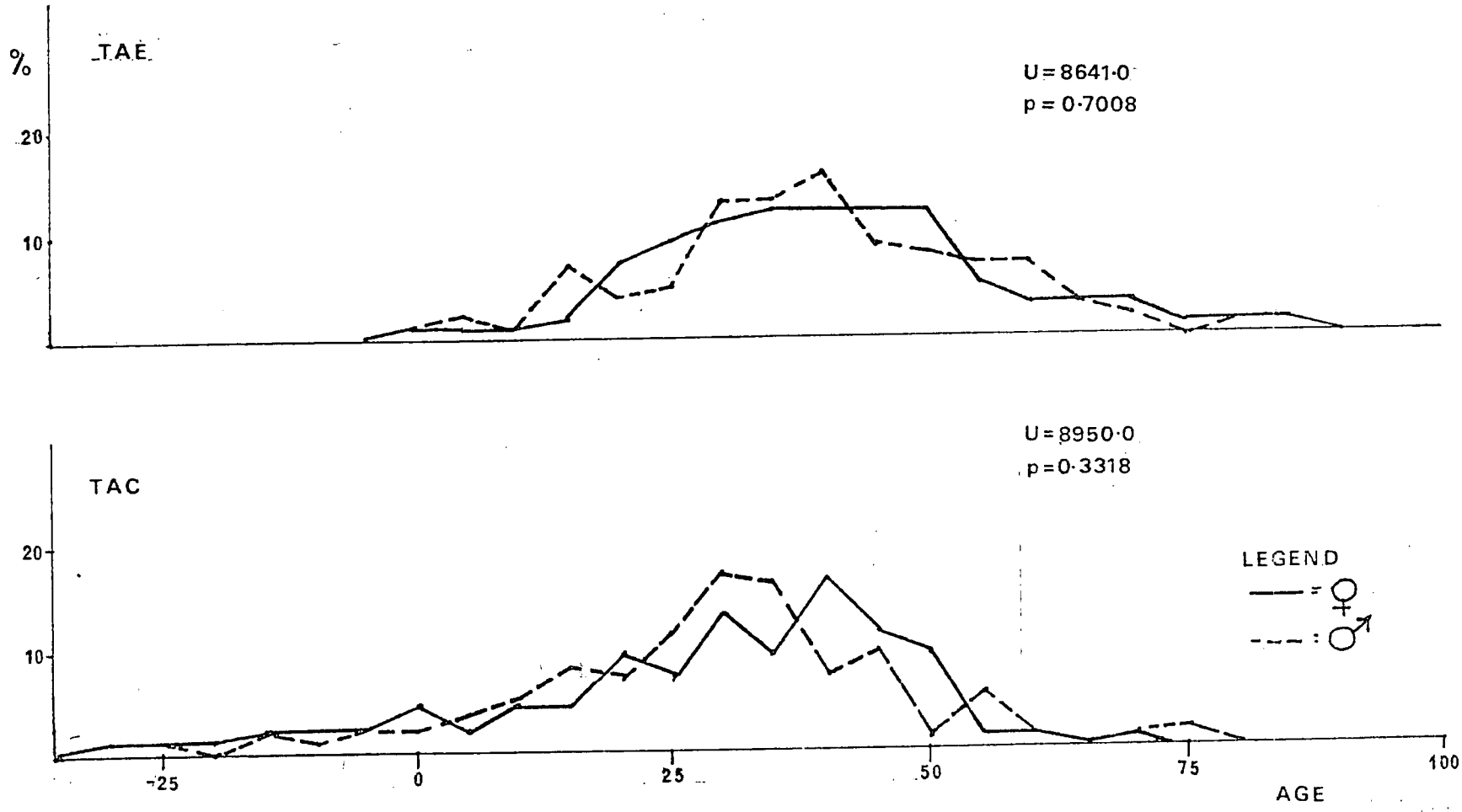
FIG. 7·1 DISTRIBUTIONS OF AGE AT DEATH FROM MICROAGE ESTIMATIONS IN 5 YEAR CATEGORIES.




%
30
20
10
0
KAN
U=188·0
p=0·7017
LÉGEND
= ♀
= ♂
KAL
U=1828·5
p=0·0339
KAF
U=1824·0
p=0·0613
25
50
75
100
AGE


%
TAB
30
20
10
0
U= 8740·5
p =0·2491
TAA
20
10
U=9462·5
p=0·8646
25
50
75
100
AGE


%
TAE
U=8641·0
p=0·7008
20
10
TAC
U=8950·0
p=0·3318
20
10
LEGEND
= ♀
= ♂
-25
0
25
50
75
100
AGE

for microscopic examination. In addition, the majority of parameters to be measured for age estimation are features of adult bone, which are forming during childhood, but not very prevalent. The two methods which do measure the retention of childhood bone features (KAN and KAL) do indicate small peaks of distribution in the younger ranges.

KAO, CLAGE and ADAGE provide distributions showing a peak of mortality, then a fall approaching a normal distribution. This pattern is also produced by the five methods of Thompson. However, as mentioned above TAD has a large number of estimates below zero. This is true to a lesser extent for TAC also. So although in rank correlation tests, similar rank may be found with other estimations, the absolute values are ridiculous.

Peak and mean ages of death are given in Table 7.1. TAA and TAB have peaks in the seventies. The Kerley estimates also show tendencies to peak late in life. This might suggest an overestimation of age by these equations. CLAGE, ADAGE and TAE fit more closely to traditional expectations with peaks in the forties and fifties. Mean values of age at death again have the highest values from TAA and TAB in the sixties. CLAGE also gives a mean age of sixty. ADAGE, KAO and KAF give middle-aged values of the early fifties whereas KAL, KAN, TAC and TAE all have lower values of the twenties and thirties. KAL may be said to give the best range of estimates with a childhood peak indicated, a mean age at 44 in females and 51 in males and a peak of mortality in the sixties. This

Table 7.1 The Mean and Peak Values of age at death from the Microages.

Microage	Peak		Mean	
	Females	Males	Females	Males
CLAGE	45	45	60	63
ADAGE	55	55	49	50
KAO	57•5	67•5	50	52
KAF	25	55/80	44	51
KAL	10/65	5/60	34	41
KAN	15/35/60	10/35/60	30	27
TAA	70	70	62	63
TAB	70	70	66	65
TAC	40	30	25	24
TAD	- 5	-10	- 6	- 5
TAE	35 - 50	40	36	38

approximates modern values, rather than the values normally thought to hold for ancient populations of high mortality in the thirties.

7.2 Distributions of bone structure measurements.

The distributions of the microparameters are given in Table 4 of Appendix B. No significant differences are found between the sexes for any of the measures of internal remodelling using the Mann-Whitney U test. CTHICK , however, shows a significant difference between the sexes. The U and p values for microages and microparameters are given in Tables 2 and 3 of Appendix B.

7.3 Correlations between the microage estimations.

Spearman's nonparametric statistic for correlation by rank order is used to test the microage estimations for correlation with one another. The correlation coefficients and p values are given in Table 5 in Appendix B. Significant values are indicated in the matrix in Fig. 7.2 by shaded boxes for simpler visual effect.

Significant correlations are found between all the microage estimations for the females. The males, by contrast to the females, do show a number of non-significant correlations. KAF does not correlate significantly with any of the other age estimates except KAO. Neither KAL nor KAN correlate significantly with the other two age estimates of

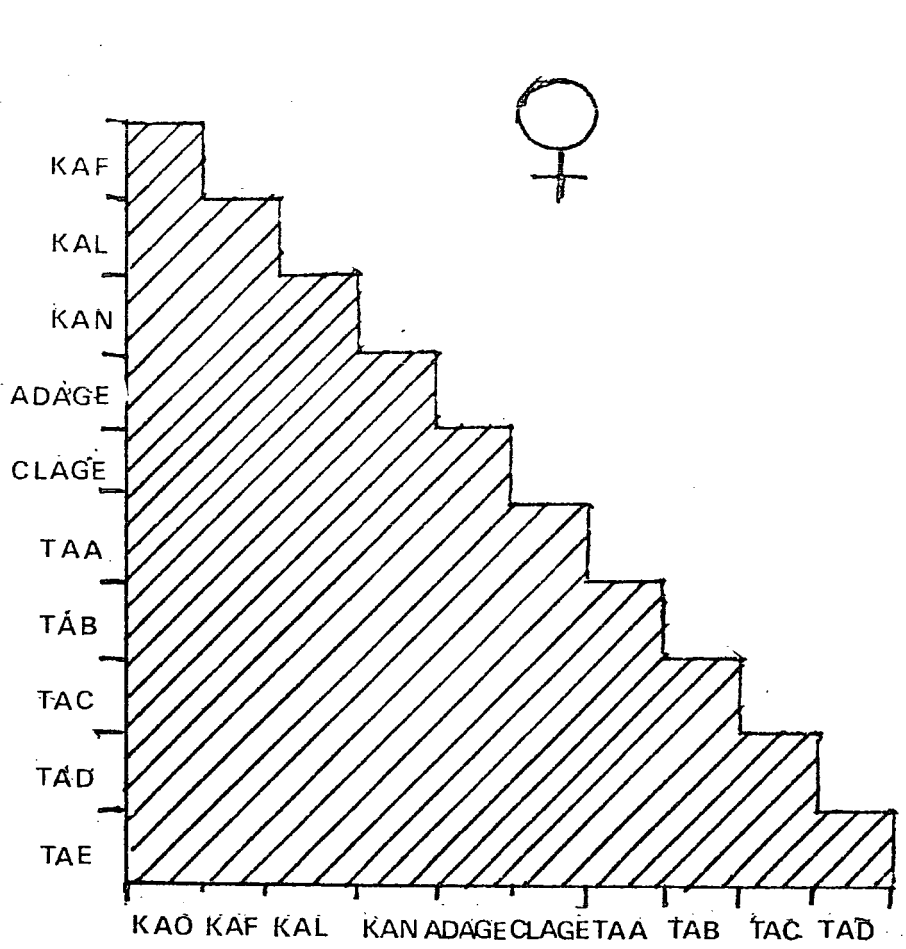
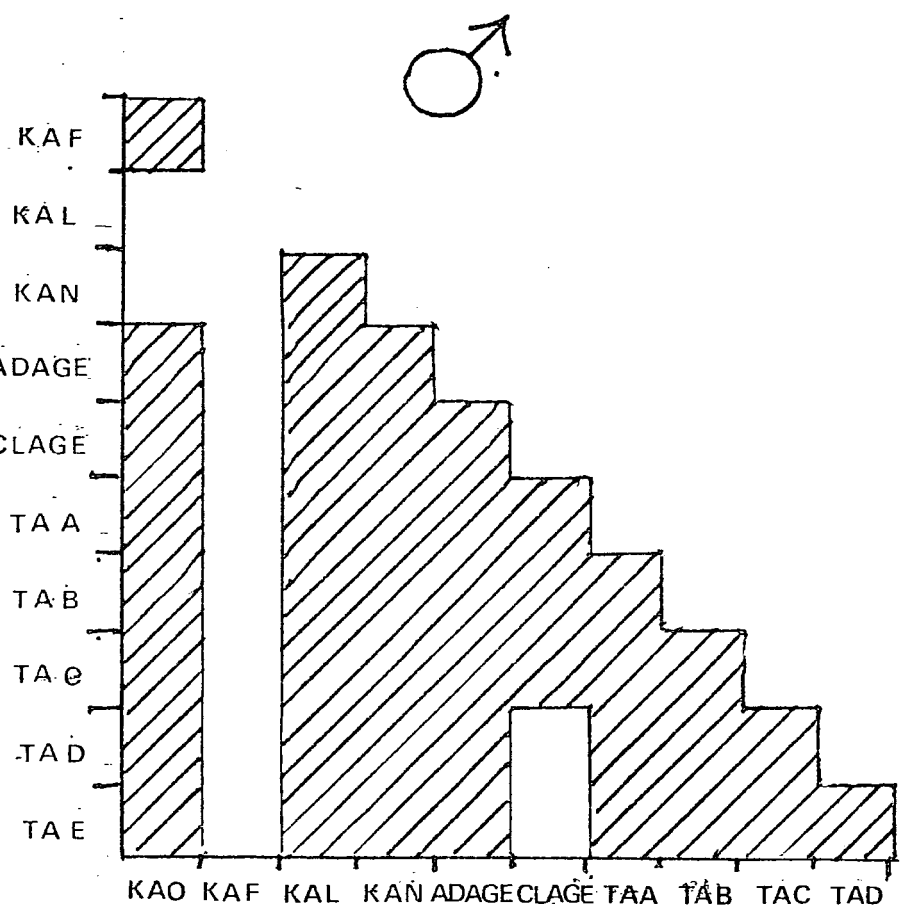


FIG. 7·2 CORRELATIONS BETWEEN THE MICROAGE ESTIMATIONS.



Kerley i.e. KAF and KAO, but do correlate with each other. So amongst the Kerley estimates of age KAO and KAF correlate and KAN and KAL correlate, but no relationship is found between the two pairs. TAE also does not correlate with KAO, and neither TAD nor TAE correlate with CLAGE. So although there is no significant difference to be found between the sexes in distribution within any one age estimation method, the sexes do show differences in the closeness of one estimation to another. This is seen as less consistency between different estimations for the males particularly involving the Kerley estimations. For this reason it does not seem useful to form a single age estimation by taking the overlap area from the four separate age estimations given by Kerley's equations, as he suggests should be done (Kerley, 1965). Instead, the four have been treated separately throughout.

7.4 Correlations between the microparameters.

If differences are found between the rank relationships of the microage estimates, it must either be a result of corresponding differences between the measures of parameters constituting the age estimation, or from a distortion of the relationship of the parameters brought about by the equations for conversion to age estimations. A consideration of the relationships between the parameters could help distinguish where the failure in consistency lies. As for the microage cross-correlations, the coefficients and p values are given in Table 6 in Appendix B, with a visual display of the

significant correlations using Spearman's statistic is given below in Fig. 7.3.

The external parameter of cortical thickness (CTHICK) shows only one significant correlation in both sexes with any of the internal parameters. The correlation is with the average mean osteon canal diameter, a negative relationship of a low order (-·2308) which informs of a slight but significant tendency for the average mean osteon canal diameter (AMOD) to increase as cortical thickness decreases. The failure of cortical thickness to correlate significantly with any of the other internal parameters indicates an independence of process between the two levels.

Amongst the internal measures, significant correlations for both sexes are found between the three measures of osteon number (KNSO, TNSO, CNSO) the percentage area of circumferential lamellae (KPCL) the percentage area of osteons and fragments (ADPO) the number of non-haversian canals (KNNH) and the total osteon perimeter length (TOPL). The measures of childhood features, (KPCL and KNNH) are correlated negatively with the other parameters, and correlated positively with each other. In addition to this main group, the number of osteon fragments (KNOF) correlates with KNSO for both sexes. KNOF, therefore is somewhat isolated from the main group but does correlate with osteon number of Kerley. Here can be seen the isolation of KAF in males from the other parameters, as the correlation with KNSO is the only significant one for KNOF in males. However, KNSO correlates significantly with the

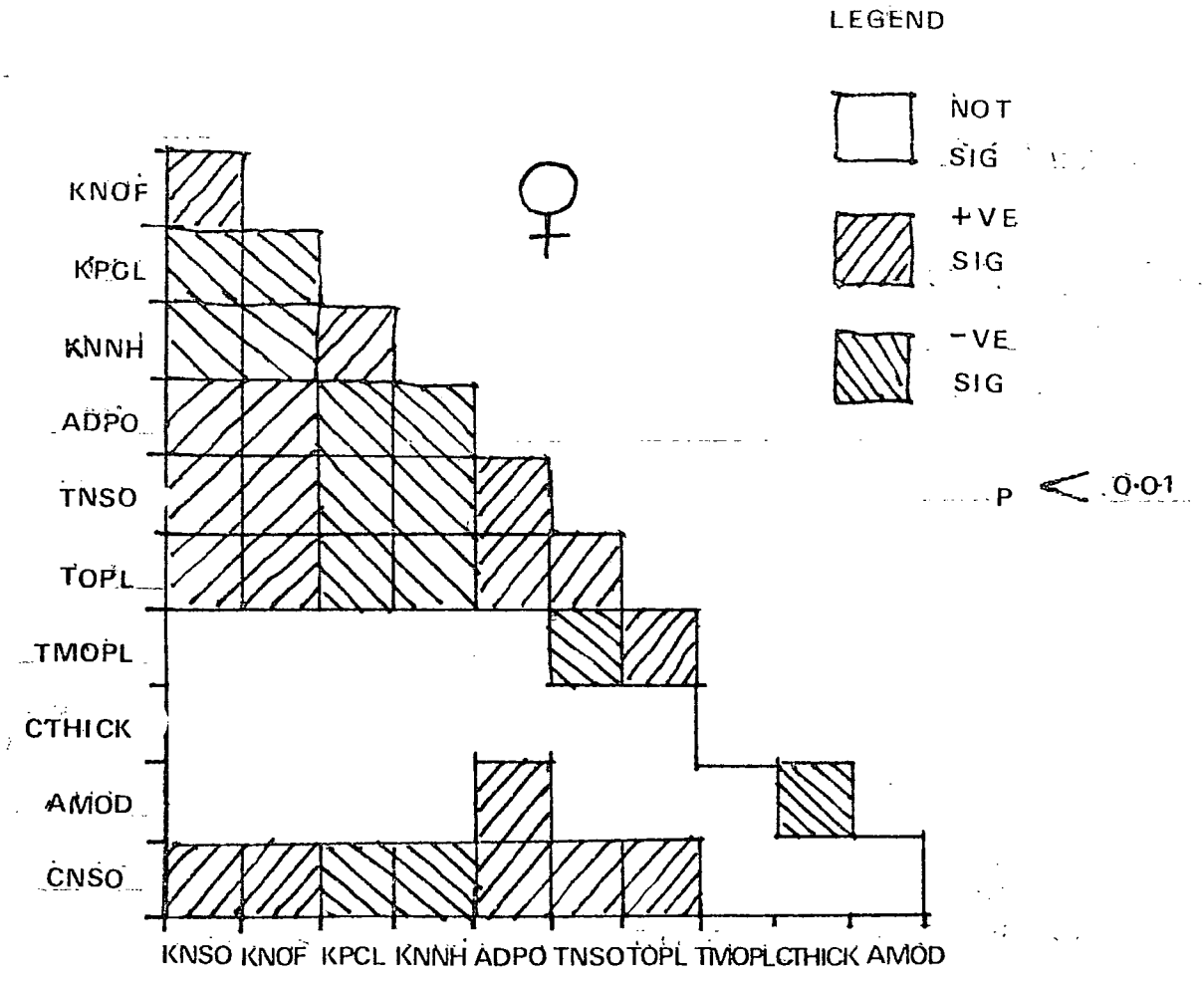
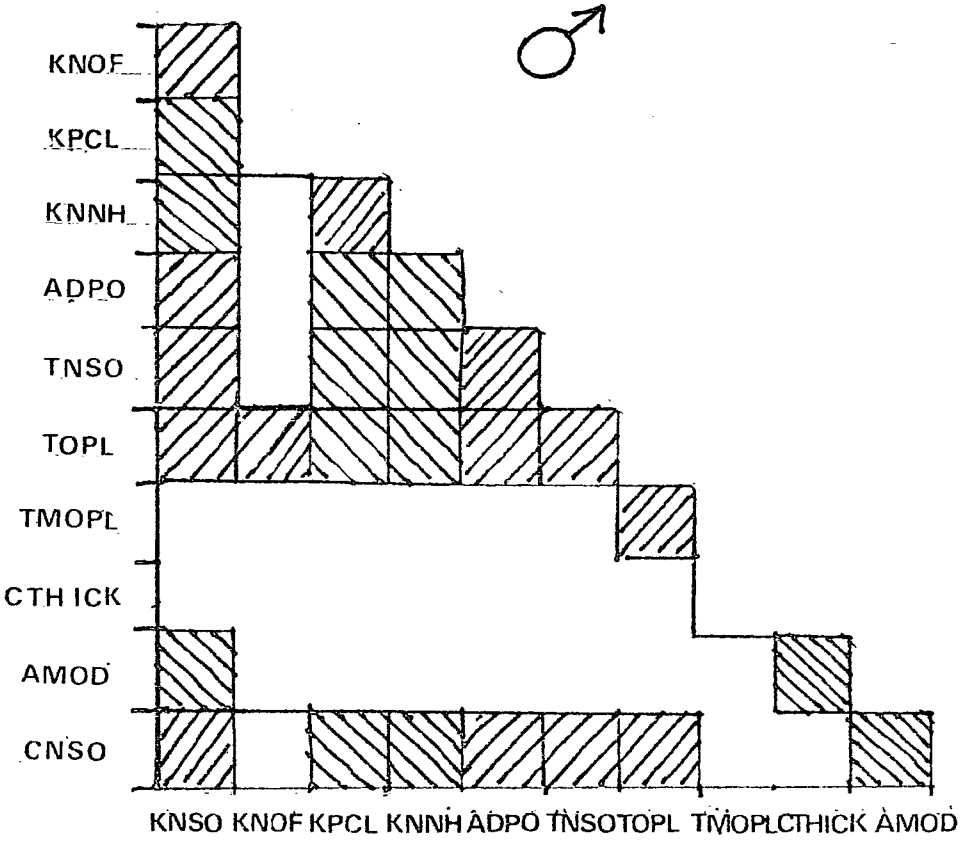


FIG. 7·3 CORRELATIONS BETWEEN THE MICROPARAMETERS.



two measures of childhood features so does not echo the break found in the converted age estimates from these parameters of KAO (KNSO) and KAL and KAN (KPCL, KNNH). It is suggested therefore that the conversion from the parameter measure to the absolute age estimation is distorting the significant relationship found between the parameters, to a non-significant one between the corresponding age estimations.

Correlations specific to one or other of the sexes are found beyond the main core group already discussed. The mean osteon canal diameter (AMOD) has a negative correlation in males with the osteon number measures of Kerley and Samson (KNSO, TNSO). Since the hypothesised age relationships of the osteon number and mean canal diameter were an increase and a decrease respectively this is a consistent relationship. However, if the correlation mentioned earlier between cortical thickness and canal diameter is also included some confusion arises. Cortical thickness is also supposed to be decreasing with age for use in age estimation equations. The relationship between canal diameter and cortical thickness is also negative, therefore at least one of the pairwise relationships found by the correlation tests is being mediated by some factor other than age since it is impossible for KNSO to increase with age, AMOD and CTHICK to decrease and two negative correlations to exist with AMOD. Important, of course, is first the observation that CTHICK does not correlate significantly with KNSO, and secondly, that the correlation coefficients for AMOD with KNSO and CTHICK are very low. The later examination of the parameters' change in

relation to dental age and the factor analysis discussed in the final results chapter should cast more light on this apparent paradox.

Amongst the females, the number of osteon fragments (KNOF) is part of the main group of related parameters, not an isolated variable as in males. The mean osteon perimeter length (TMOPL) shows a negative correlation with the number of osteons measured according to Thompson (TNSO), so that as the number of osteons increases, the mean perimeter length decreases whereas the total perimeter length (TOPL) was seen above to be positively correlated with TNSO, so increases with an increase in osteon number. TMOPL correlates positively with TOPL in both sexes, so that as mean perimeter length increases so does the total, obviously. Again an inconsistency is seen here, TMOPL and TOPL positively correlated to one another but correlated with opposite signs to the third parameter of TNSO. Again, other factors must be involved affecting the size of the three parameters beyond the presumed linear relationships to age allowing for different relationships to co-exist. Again a factor analysis will illustrate the importance of any single agent in explaining the variations. The correlation coefficients for TMOPL with either TNSO or TOPL are of a low order, even though significant. Finally, amongst females a significant correlation is found between mean osteon diameter (AMOD) and the % of area covered by osteons and osteon fragments (ADPO). This is a consistent relationship with the correlation already established with cortical thickness. However, it implies

if age mediates the relationships, that as the % area of osteons and osteon fragments increases and cortical thickness decreases with age, the mean osteon diameter shows an increase, rather than the decrease expected by the equation.

The failure of CLAGE to correlate with TAD or TAE would not necessarily be predicted from the relationships of the parameters. The Thompson age estimates are more complicated to break down than the Kerley ones, based on a number of variables rather than just one. However, there is no more significant correlation between the last two Thompson parameters (TOPL, TMOPL) and the Samson parameters (AMOD, CNSO) for females than for males, so the lack of relationship only for males is not explained simply by the relationship of the parameters in males. It would appear therefore that again some distortion is occurring in translation from raw measure to absolute age estimation. Some differences between the sexes in the relationships of the various parameters is seen to exist, so although no significant difference by sex is seen within the distribution of any one variable, the sexes should be kept apart and treated separately because of these relationship discrepancies.

7.5 Microage and Dental Age.

The correlation coefficients and p values of correlation between the microages and dental age and pubic symphysis age are given in Table 7. of Appendix B using Spearman's rank correlation statistic. Those showing significant correlations

Table 7.2 Significant Correlations Between the Microages and the Dental and Pubic Symphysis Ages.

Females			Males		
Microage	Dental Age (coefficient + p)		Microage	Dental Age (coefficient + p)	
TAB	•4921	•000	KAL	•4426	•001
ADAGE/TAA	•4617	•000	CLAGE	•2349	•009
CLAGE	•4098	•000			
KAL	•4055	•000			
KAO	•3782	•000			
TAC	•3448	•000			
TAE	•2647	•003			

	Pubic Symphysis Age (coefficient + p)		Pubic Symphysis Age (coefficient + p)	
TAB	•3905	•002		
ADAGE/TAA	•3577	•005		
TAC	•3484	•007		

are given in Table 7.2. The female age estimations have a greater number of significant correlations with dental age than males. Only CLAGE and KAL are significantly correlated in males, whereas all are correlated in females except KAF, KAN and TAD. KAN, as mentioned earlier has a small sample size, while KAF and TAD, although not significant only just miss with $p = 0{\cdot}011$ in both cases. The highest correlation coefficient of all is for females between TAB and dental age at 0·4921, not a very high level of correlation.

None of the male microage estimates correlates with pubic symphysis age. Females have four significant correlations with ADAGE, TAA, TAB and TAC. Again TAB has the highest coefficient with pubic symphysis age at 0·3905 and again a very low level of correlation.

The mean microage values in each dental age group are presented in Fig. 7.4. The tendency for the mean age to drop in the last dental age group is apparent in most, particularly so in males. Where female values do drop in the last dental age group (e.g. TAB) it is only by a small amount relative to the previous increase by dental age group. The confusion of microage estimates through the dental age groups can be seen clearly in Fig.s 7.5 and 7.6 where the cumulative frequencies of the microages are plotted by dental age group for KAL and TAB. TAB shows a clear demarcation of the four categories for females, but the others, even though a significant correlation with dental age is indicated for KAL, show overlapping of the lines. Similar graphs for CLAGE, ADAGE,

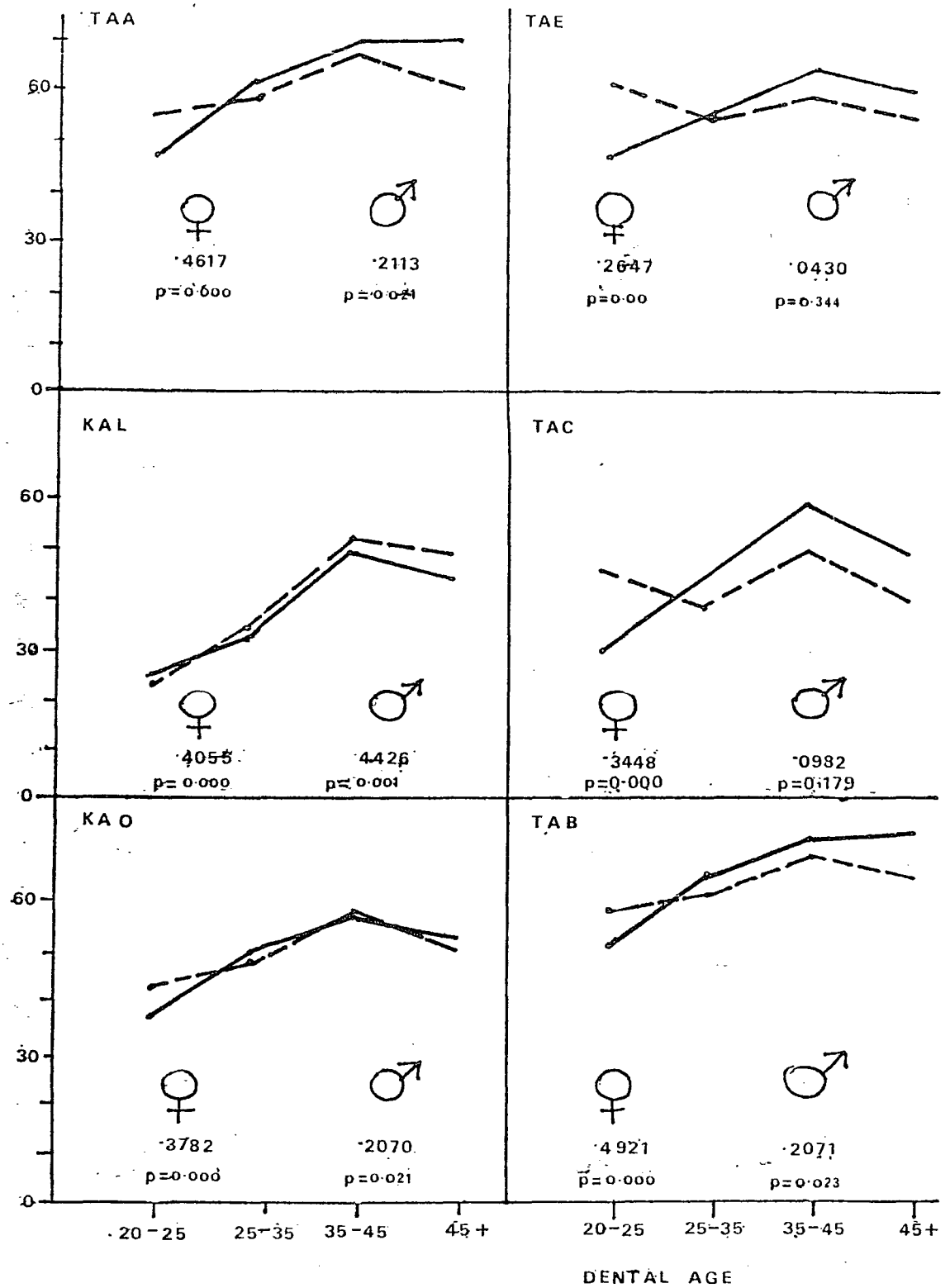
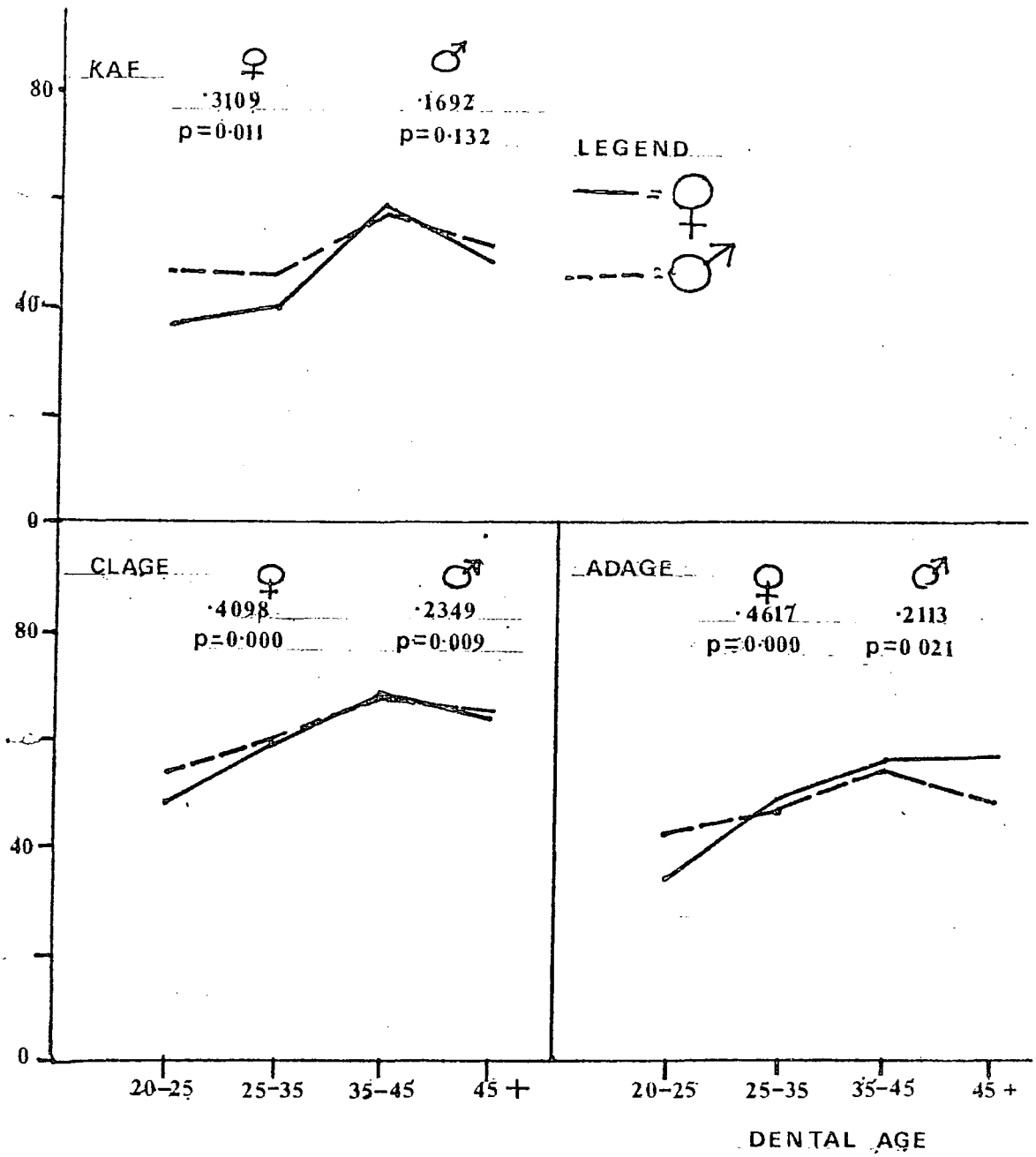


FIG. 7·4 MEAN VALUES OF MICROAGE ESTIMATIONS IN EACH DENTAL AGE GROUP.


KAE
♀
·3109
p=0·011
♂
·1692
p=0·132
LEGEND
80
40
0
CLAGE
♀
·4098
p=0·000
♂
·2349
p=0·009
ADAGE
♀
·4617
p=0·000
♂
·2113
p=0 021
80
40
0
20–25
25–35
35–45
45 +
20–25
25–35
35–45
45 +
DENTAL AGE

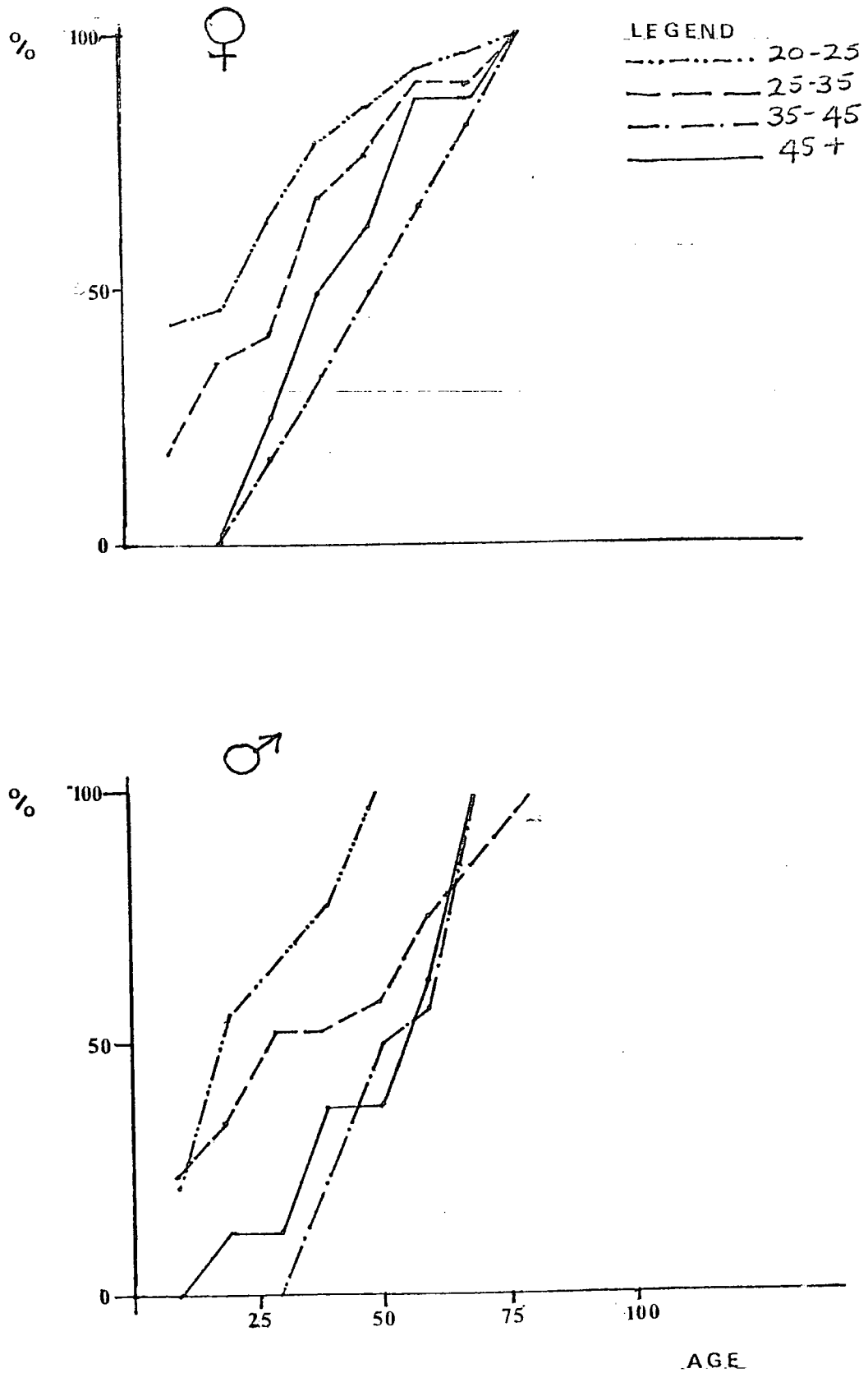


FIG. 7·5 CUMULATIVE % FREQUENCY OF KAL IN 10 YEAR INTERVALS PLOTTED BY DENTAL AGE GROUP.

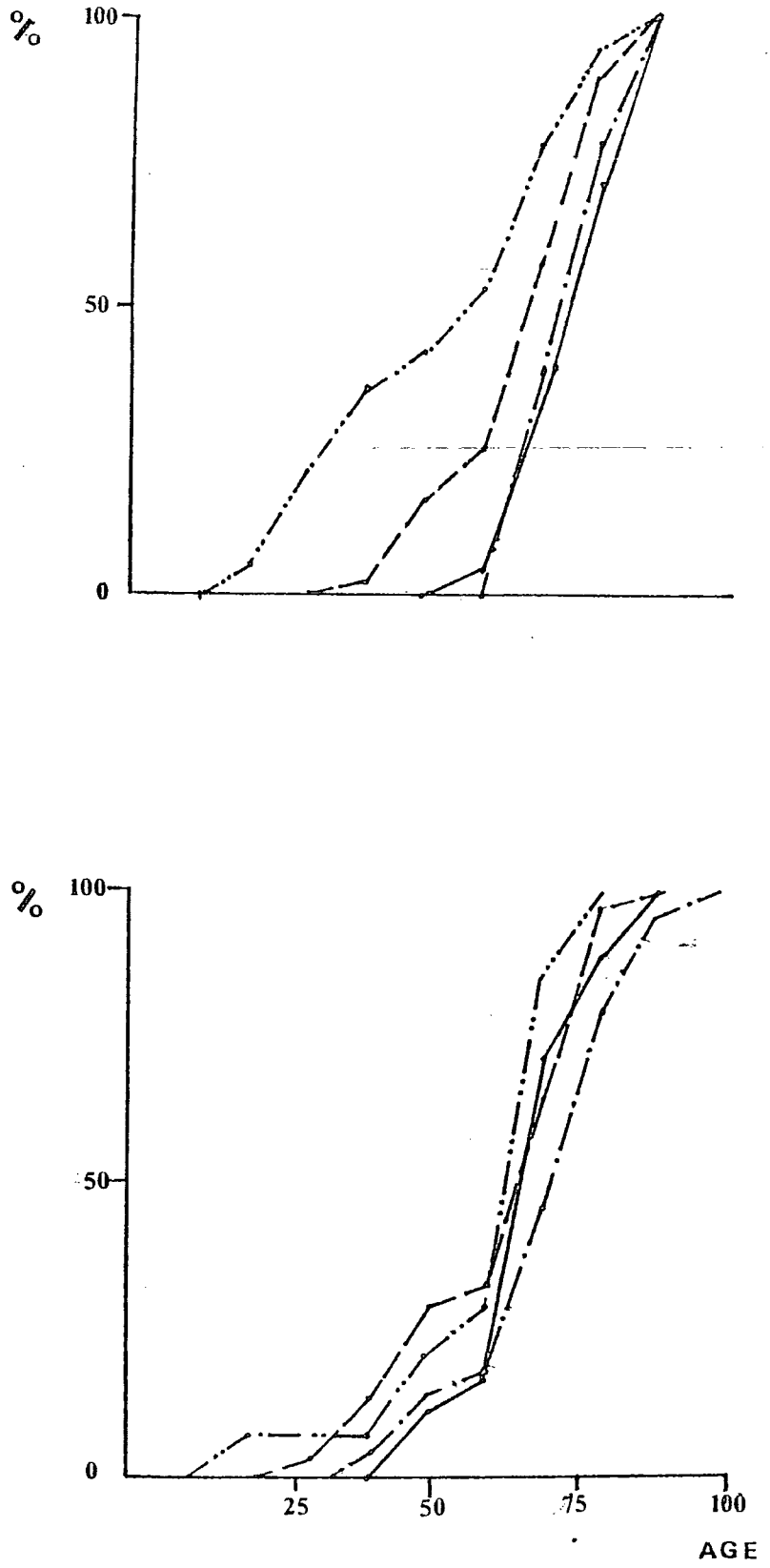


FIG. 7·6 CUMULATIVE % FREQUENCY OF TAA IN 10 YEAR INTERVALS PLOTTED BY DENTAL AGE GROUP.

KAO and TAA are given in Figs. 1 to 4 of Appendix B, where similar confusion of dental age groups can be seen.

Mann-Whitney U tests have been run for sex differences in the microage distributions in each dental age group. Because of small sample sizes in each subsample, significance is accepted at $p < 0{\cdot}05$. The U and p values are given in Table 8 of Appendix B. Three microage estimations show significant sex differences in the oldest dental age group. These are ADAGE, TAA and TAB. In all cases, this represents a higher distribution of microages amongst females, reflecting the greater drop in male values in the 45+ dental age group. TAC shows a higher range of female values in the 35 - 45 dental age group whereas TAE shows higher values of the male age estimations in the 20 - 25 age group.

The mean ages from the microage estimates in each dental age group are all higher for males compared to the age range of the dental age group except for KAL and TAC. Females have higher values from the Thompson methods, particularly in the last two dental age groups. None of the others really shows any differences.

The coefficients of variation are plotted by dental age group for each method except KAN and TAD in Fig. 7.7. The mean values, standard deviations and coefficients of variation are given in Table 9 in Appendix B. TAD has been excluded because of the minus values of the mean age estimates and very high coefficients. KAN with the small sample size

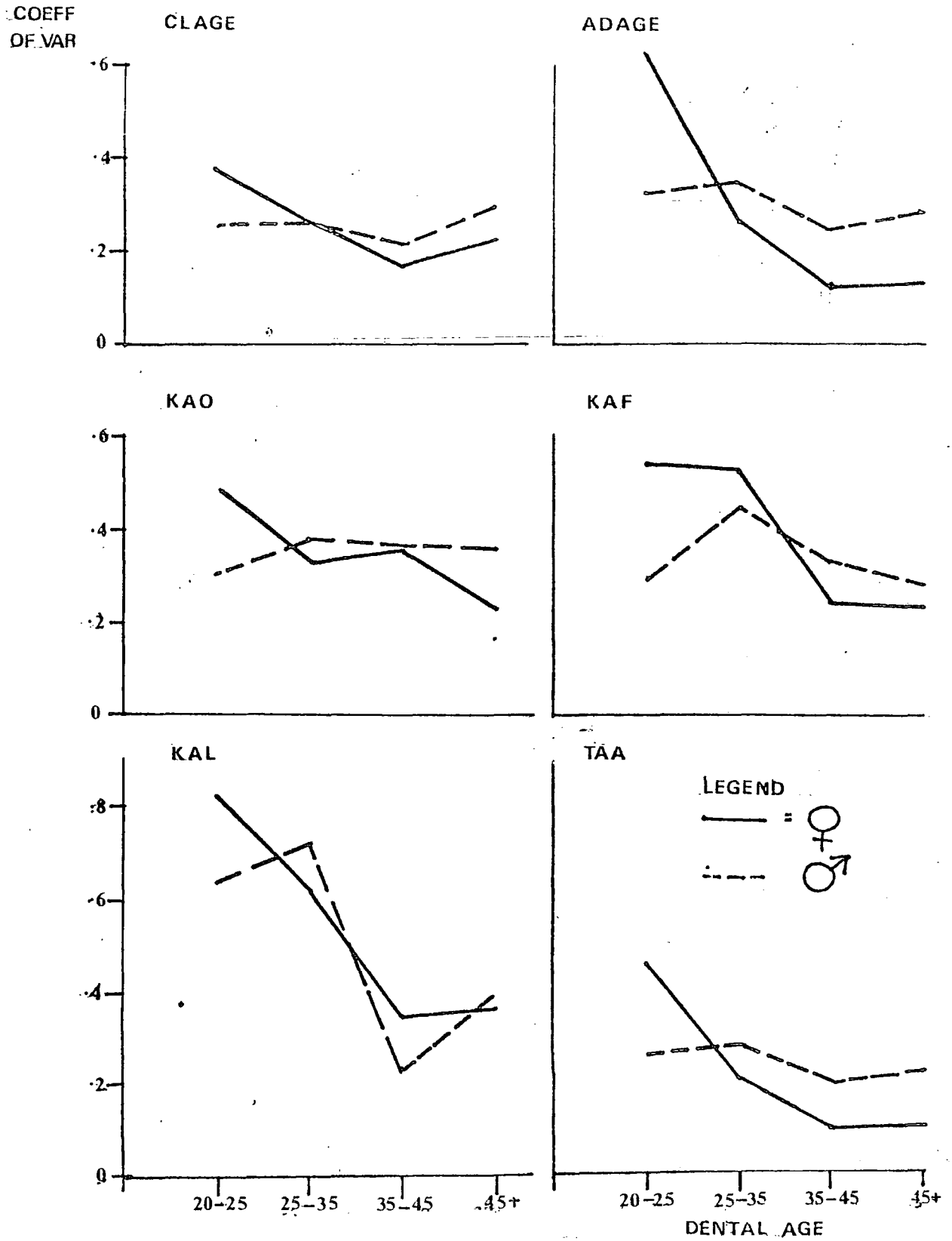
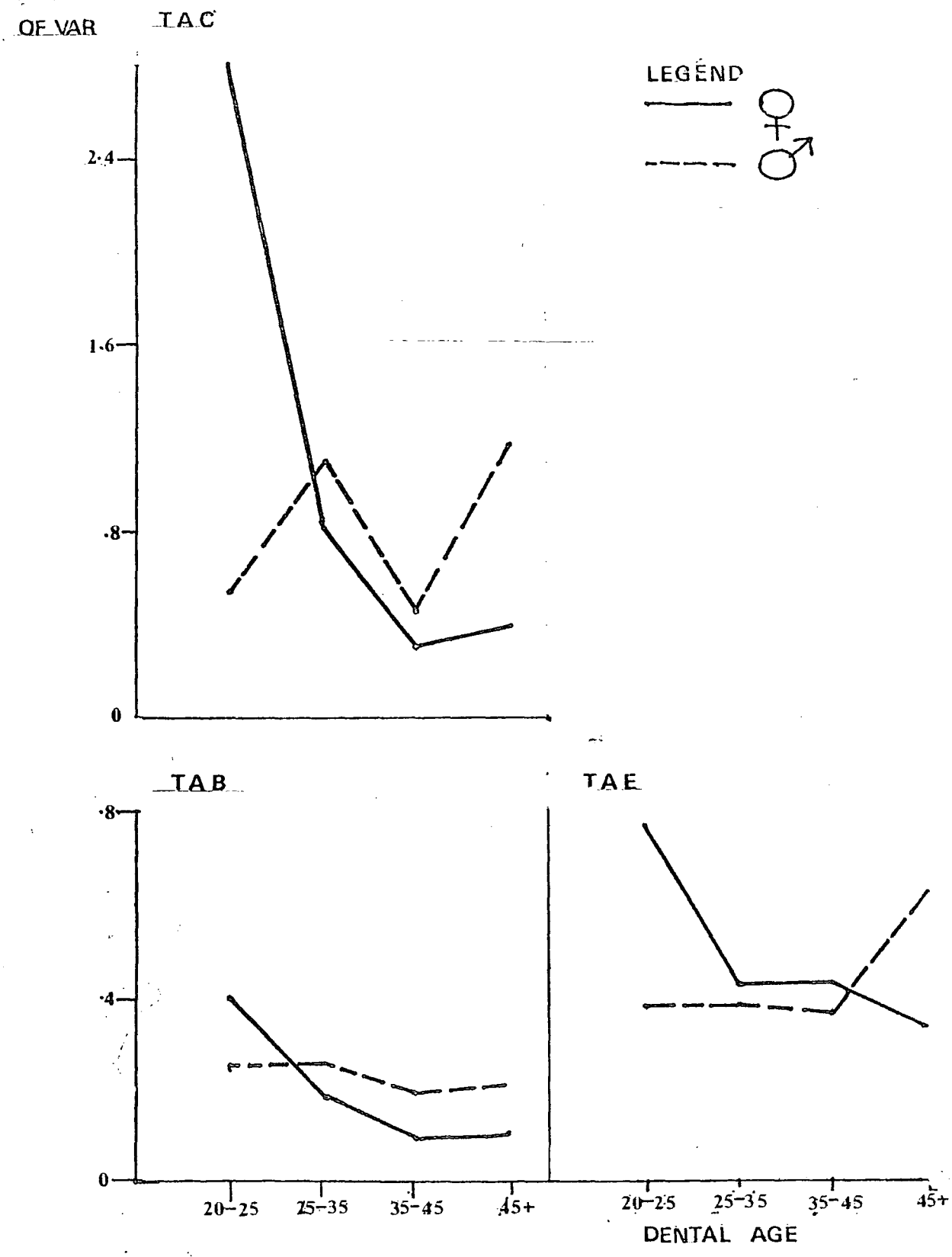


FIG. 7·7 COEFFICIENTS OF VARIATION FOR THE MICROAGE ESTIMATIONS IN EACH DENTAL AGE GROUP.


COEFF
OF VAR
TAC
LEGEND
♀
♂
2·4
1·6
·8
0
TAB
TAE
·8
·4
0
20–25
25–35
35–45
45+
20–25
25–35
35–45
45+
DENTAL AGE

involved has only one case in some dental age groups so neither mean nor standard deviation are available.

The male coefficients are consistently higher than females in the last dental age group and often in earlier ones too. All show a tendency to decrease with dental age, with a rise in the last dental age group particularly in males. This, of course, reflects the increase in the mean value and its decrease at 45+ dental age. However, the standard deviation is not increasing proportionately so therefore is relatively smaller.

7.6 Microages and dental attrition and pubic symphysis metamorphosis.

The correlation coefficients and p values for correlation of the microages with dental attrition and pubic symphysis metamorphosis are listed in Table 10 of Appendix B. Those showing significant correlations using Spearman's rank correlation statistic are given in Table 7.3. Females show higher coefficients with M_1 than M_2, males the reverse. In males, only KAL and CLAGE were significantly correlated with dental age. Four estimates correlate significantly with attrition scores for both M_1 and M_2 in males. They are KAL, CLAGE, TAB, TAA and ADAGE. It would have been predicted that all these five would correlate significantly with dental age. The female correlations are the same as the dental age list except for TAB which is significant with both molar attrition scores but not quite with dental age. The actual

Table 7.3 Significant Correlations Between the Microages and the Dental Attrition and Pubic Symphysis Metamorphosis Scores

M_1

Females		Males	
CLAGE	•4668 •000	TAA/ADAGE	•2955 •006
TAE	•4627 •000	CLAGE	•2796 •006
TAB	•4511 •000	KAL	•3923 •007
TAA/ADAGE	•4413 •000	TAB	•2717 •010
KAO	•4359 •000		
TAD	•3795 •000		
KAL	•4115 •001		
TAE	•3165 •001		

M_2

Females		Males	
CLAGE	•4290 •000	KAO	•4033 •000
TAC	•4277 •000	TAA/ADAGE	•3720 •001
TAB	•4063 •000	TAB	•3516 •001
TAA/ADAGE	•3931 •000	CLAGE	•3468 •001
TAD	•3641 •000	KAL	•4665 •002
KAO	•3363 •001		
TAE	•3241 •001		

M_3

Females	
TAD	•4063 •005
TAE	•3884 •007
TAC	•3698 •010
KAO	•3569 •010

Pubic Symphysis Metamorphosis

Females	
TAB	•3655 •004
TAA/ADAGE	•3655 •004

coefficients show no particular relationship in terms of size to one another; in some cases the dental age coefficient is higher, in others the attrition score coefficient. The pubic symphysis metamorphosis scores show no significant correlation with any of the male microage estimates. The same microages for females are significantly correlated with pubic symphysis metamorphosis as were with pubic symphysis age except for TAD which is not significant with the raw score but is with the converted age.

7.7 Microparameters with Dental Age and Pubic Symphysis Age.

The correlation coefficients and p values for the relationship of the microparameters with dental age and pubic symphysis age are given in Table 11 of Appendix B, using Spearman's rank correlation test. Those showing significant correlations are listed in Table 7.4. Females show a greater number of significant correlations than males. Four of the microparameters do not correlate for females with dental age (KNOF, KNNH, TMOPL and AMOD), whereas with males only four do : KPCL (-ve), CNSO, TNSO and TOPL. Only ADPO correlates significantly with pubic symphysis age in females; there are no significant correlations amongst the male coefficients.

The two best correlations for males with dental age are KPCL and CNSO, the measures used towards the age estimations of KAL and CLAGE respectively which also correlated significantly. The Thompson age estimates are more difficult

Table 7.4 Significant Correlations Between the Microparameters and the Dental and Pubic Symphysis Ages

Females			Males		
Micro-parameter	Dental Age (coeff. + p)		Micro-parameter	Dental Age (coeff. + p)	
ADPO	•4617	.000	KPCL	-•4426	•001
TNSO	•4127	•000	CNSO	•2422	•005
KPCL	-•4125	•000	TNSO	•2499	•008
KNSO	•3782	•000	TOPL	•2540	•008
TOPL	•3681	•000			
CNSO	•3127	•000			
CTHICK	-•1849	•006			

	Pubic Symphysis Age (coeff. + p)			Pubic Symphysis Age (coeff. + p)	
ADPO	•3577	•005			

to relate to specific parameters, based as they are on an increasing number of parameters. TOPL and TNSO, which correlate significantly with dental age in males, do not feature in the Thompson methods until TAD and TAE which also include three other measures which do not correlate with dental age (ADPO, CTHICK, TMOPL) with the result that TAD and TAE do not correlate with dental age, along with the other Thompson age estimates. The list of significant correlations found amongst the microparameters in females likewise corresponds well to the relationship of the relevant age estimates with dental age. ADPO correlates with pubic symphysis age in females as do the age estimates based on it, ADAGE and TAA. However, TAB, involving the addition of CTHICK shows an even better correlation although CTHICK itself does not correlate with pubic symphysis age. Similarly, TAC, although less strongly correlated than the others, involves the addition of TMOPL, which shows no significant correlation for either sex with either dental age or pubic symphysis age.

The mean values for each parameter in each dental age category are plotted in Fig. 7.8. The mean values, standard deviations and coefficients of variation are listed in Table 12 in Appendix B. The Mann-Whitney U test has been run for sex differences in distributions in each dental age group. U and p values are given in Table 13 of Appendix B.

The external parameter of CTHICK shows higher absolute mean values in males than females. The difference is

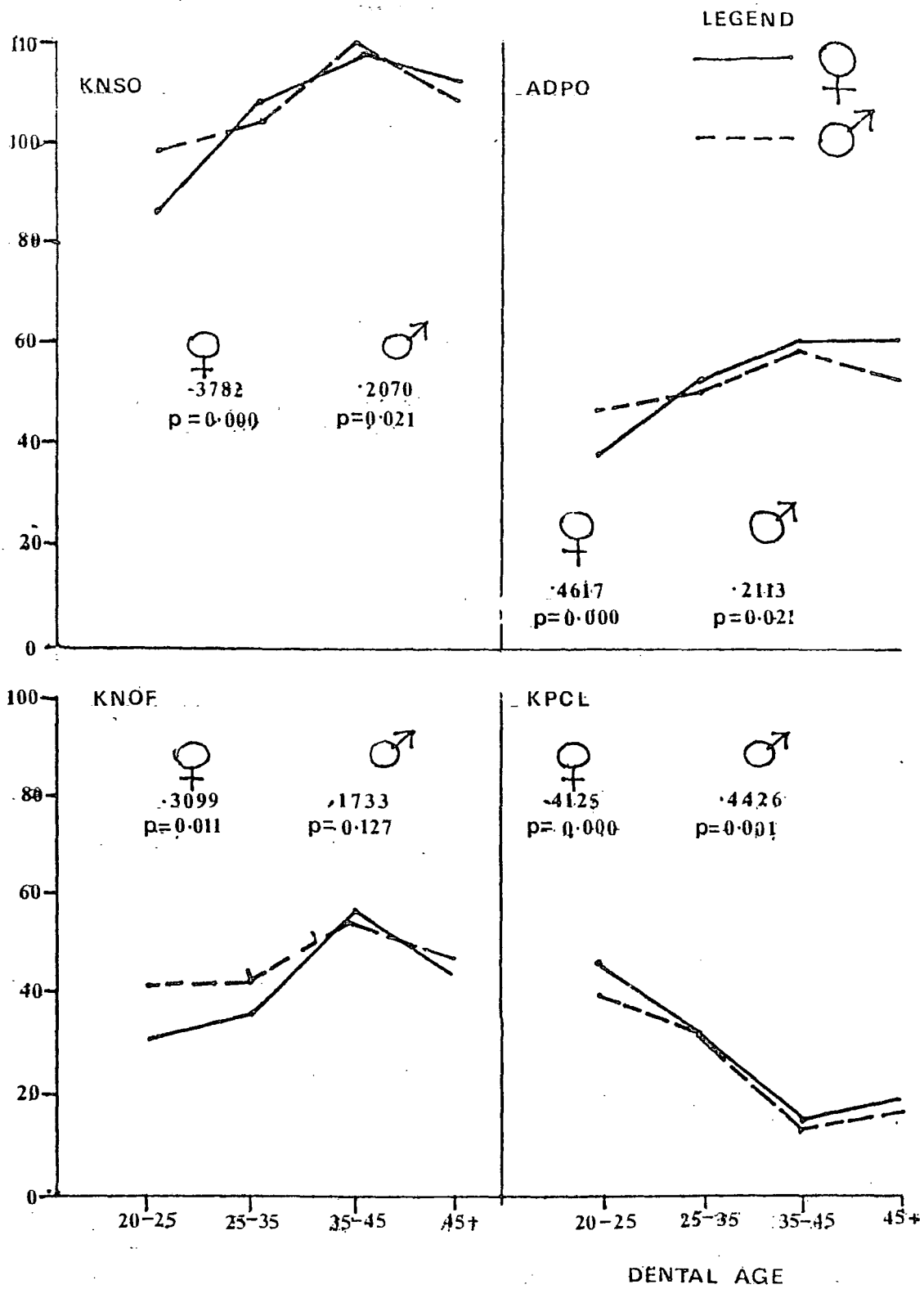
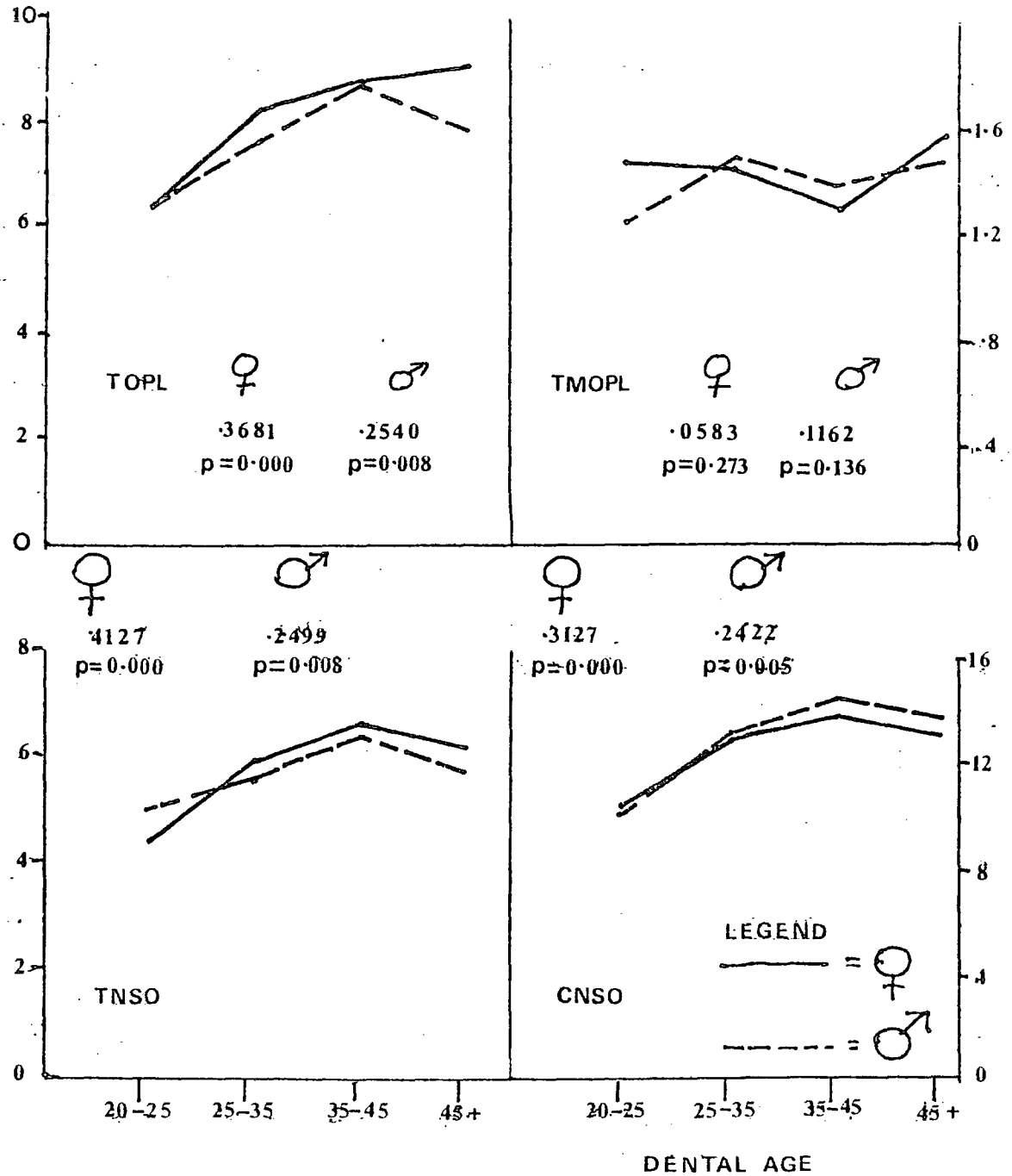
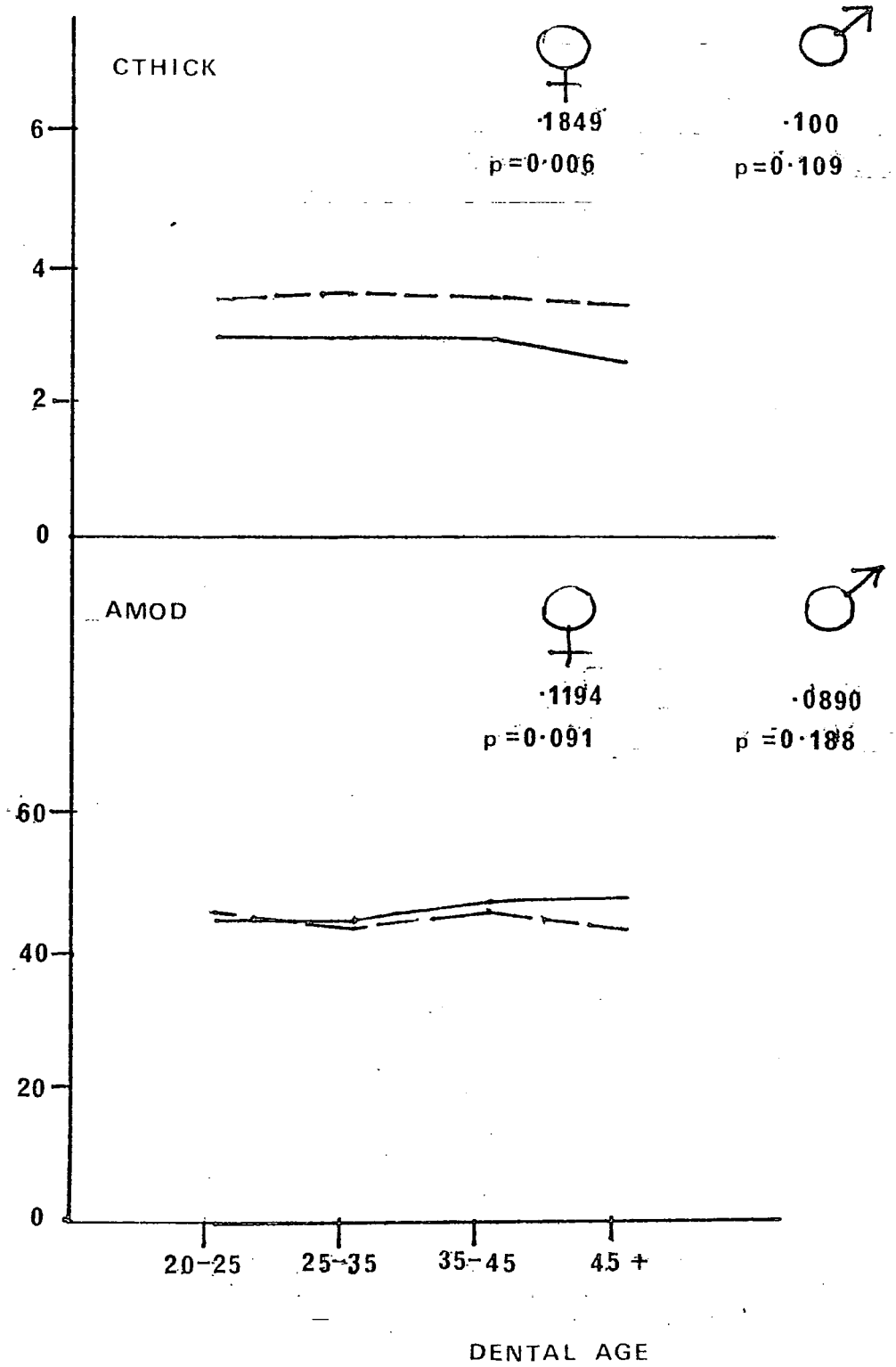
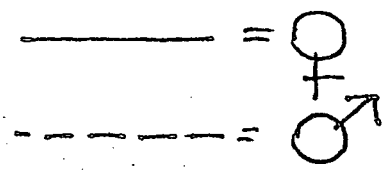


FIG. 7·8 MEAN VALUES OF THE MICROPARAMETERS IN EACH DENTAL AGE GROUP.


TOPL
·3681
p=0·000
·2540
p=0·008
TMOPL
·0583
p=0·273
·1162
p=0·136
·4127
p=0·000
·2499
p=0·008
TNSO
·3127
p=0·000
·2422
p=0·005
CNSO
LEGEND
20–25
25–35
35–45
45+
DENTAL AGE


CTHICK
·1849
p=0·006
·100
p=0·109
6
4
2
0
AMOD
·1194
p=0·091
·0890
p=0·188
60
40
20
0
20–25
25–35
35–45
45 +
DENTAL AGE

LEGEND



significant in every dental age group. CTHICK increases between the first two dental age categories and thereafter decreases in both sexes. Overall the decrease in females is significant with dental age where the male loss is not. The male loss amounts to 6% of the maximum mean thickness whereas the female loss is 12%. The female loss is not as great as that reported in modern samples. The gain between the first two dental age groups is both absolutely and relatively greater in males representing a 3% gain as opposed to a 0·8% gain in females. The absolute and % figures of gain and loss for all the microparameters are given in Table 16 of Appendix B.

The values of cortical thickness were normalized by dividing the measure by the maximum femur length and multiplying by 100, CTHICK thus expressed as a % of the femur length. The correlation coefficients and p values of the normalized and non-normalized values with dental age using Spearman's rank correlation statistic are given in Table 7.5. A slightly higher correlation and level of significance was gained by using the normalizing process, especially in females, but the overall result remained the same. Thus no particular advantage to using the method was seen.

The age related changes of the interval parameters fall into three groups. First those which show a consistent increase or decrease with dental age up to the 35 - 45 group then a decrease or increase to 45+ dental age group. KNSO, TNSO, CNSO and KNOF all show an increase-decrease pattern for

Table 7.5 Spearman's nonparametric rank correlation test between Cortical Thickness and Dental Age, comparing normalized and non-normalized data.

	Coefficient/ p value	
	Normalized	Non-normalized
Females	-•2215	-•1849
	•002	•006
Males	-•1114	-•1000
	•100	•109

both sexes. In TNSO and KNSO the female increase is greater and the decrease less than males. The female increase in CNSO is also greater but the decrease about the same in the two sexes. The % of increase and decrease for each are given in Table 17 of Appendix B. The increase with age overall is still significant despite the 45+ decrease, for TNSO and CNSO but only for female KNSO values. KNOF similarly increases by a far greater amount in females than males, but females also show a greater decrease than males. Neither retain a significant dental age relationship over the entire range. KPCL, being a measure of childhood features shows the reverse pattern of decrease-increase. The sexes show much the same pattern. Spearman's rank correlation test is performed for those parameters with dental age group up to 35 - 45 only. The results are given in Table 16 of Appendix B. KNSO, TNSO and CNSO show the increase in values is significant with dental age in both sexes. The increase in KNOF is only significant in females. The decrease in KPCL is significant in both sexes.

The male pattern of dental age related changes of ADPO and TOPL come in this first cateogory also, whereas the females constitute the second category showing a constant increase in the value throughout life. The male gain in ADPO is far less than the female, and then drops. The male gain is significant, as is the female gain throughout life. The female gain is mainly achieved between the 20 - 25 and 35 - 45 dental age groups, the gain thereafter not being significant alone. TOPL shows a lower gain for females

compared to males. Again significant through life and from the 20 - 25 to 35 - 45 dental age groups but not after. The males show a very large increase up to 35 - 45 which is significant, with a small decrease up to the last dental age category. The overall dental age increase is still significant.

Thirdly, TMOPL and AMOD have their own patterns with no significant directional correlations with dental age. TMOPL decreases in females over the first three dental age groups, not significantly, then increases sharply in the last dental age group, significantly. Males fluctuate, increasing between the first two dental age groups, decreasing up to 35 - 45 then increasing up to 45+. None of the changes is significant. AMOD changes very little at all with no significant differences between any of the dental age groups.

The coefficients of variation are plotted in Fig. 7.9 for the microparameters by dental age group. Males show a higher level of variation for internal parameters except TMOPL, where no difference is evident. The higher male value is particularly marked in the last dental age group. The external parameter of CTHICK shows the reverse pattern with a higher coefficient throughout all dental age groups for females.

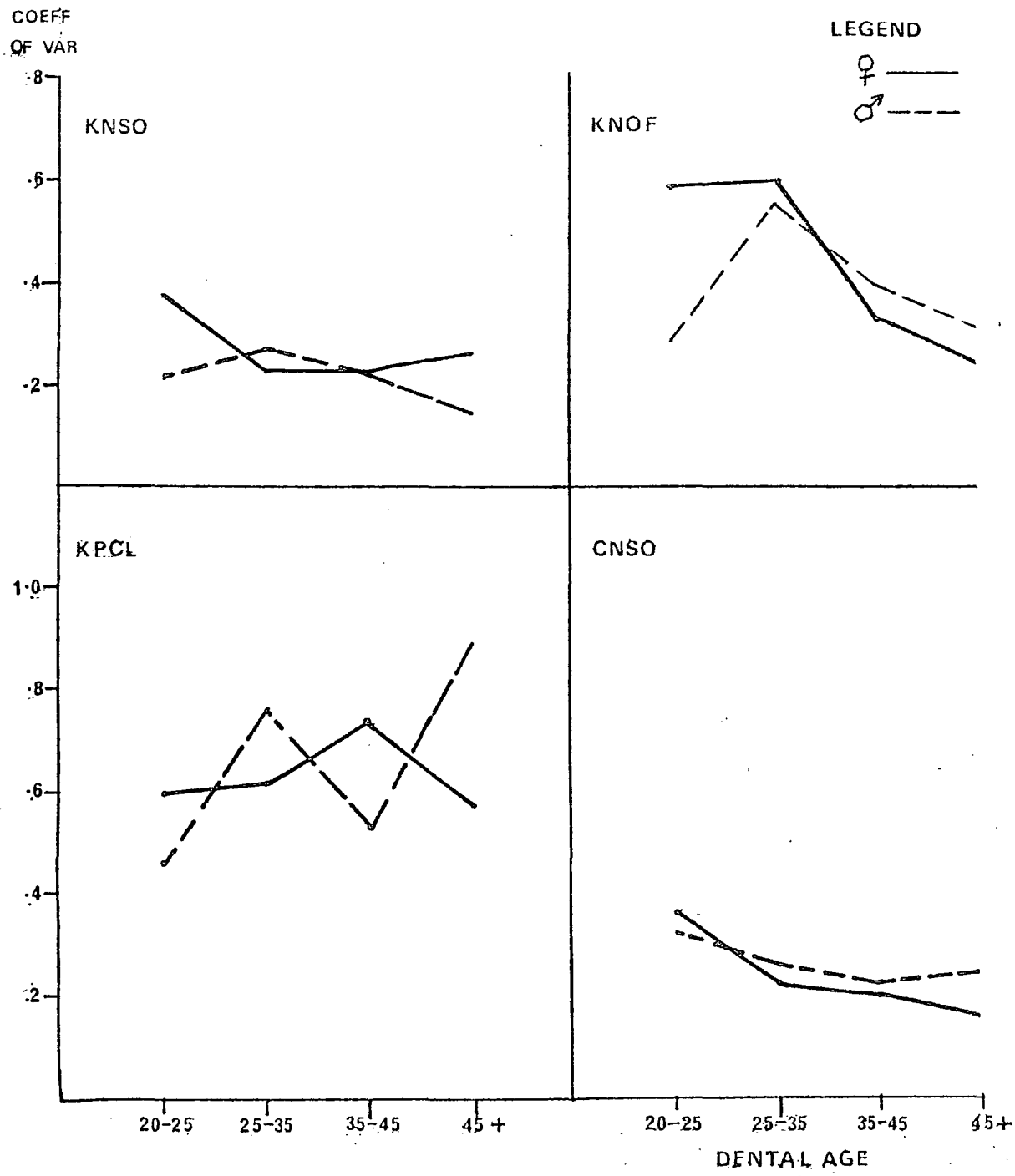
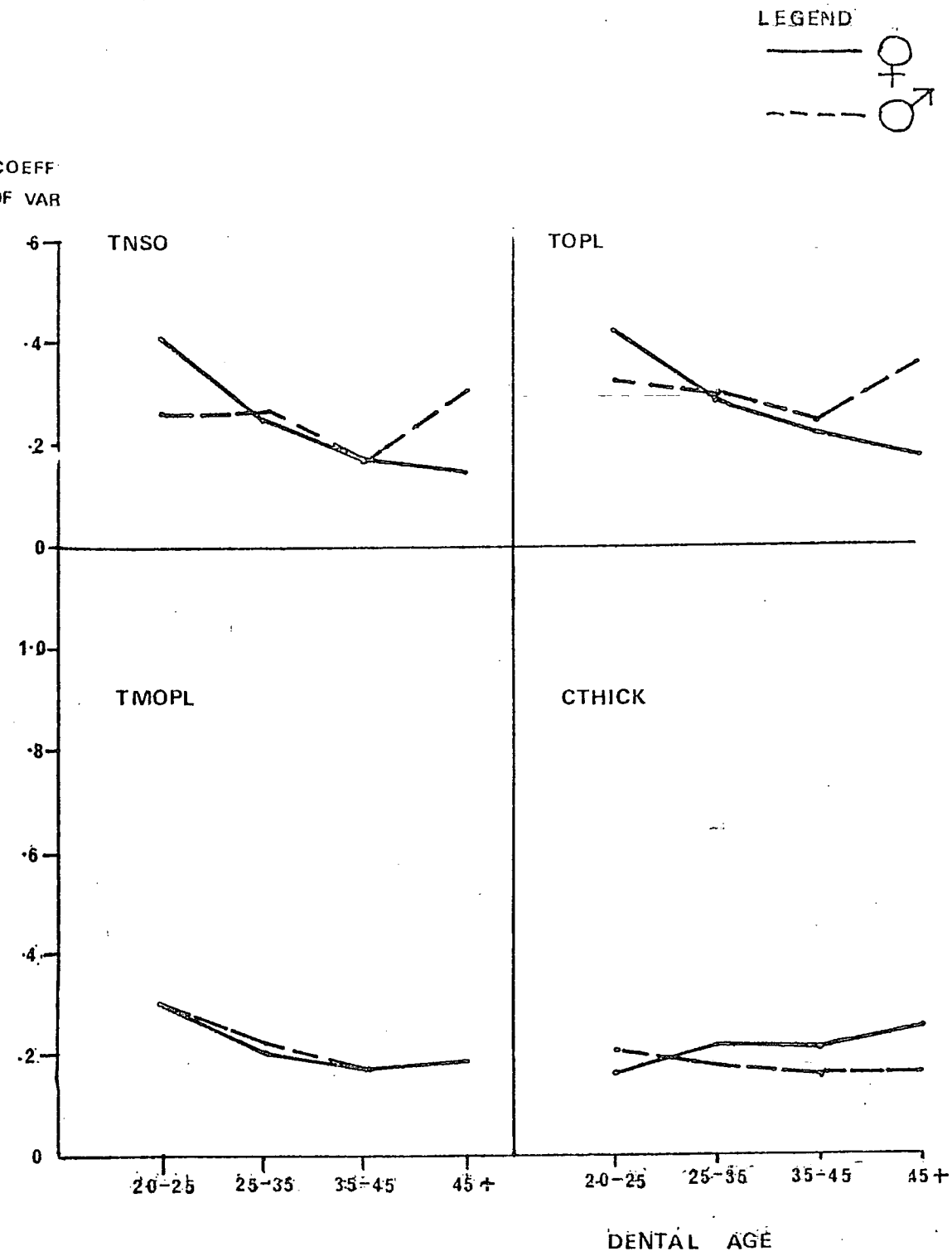
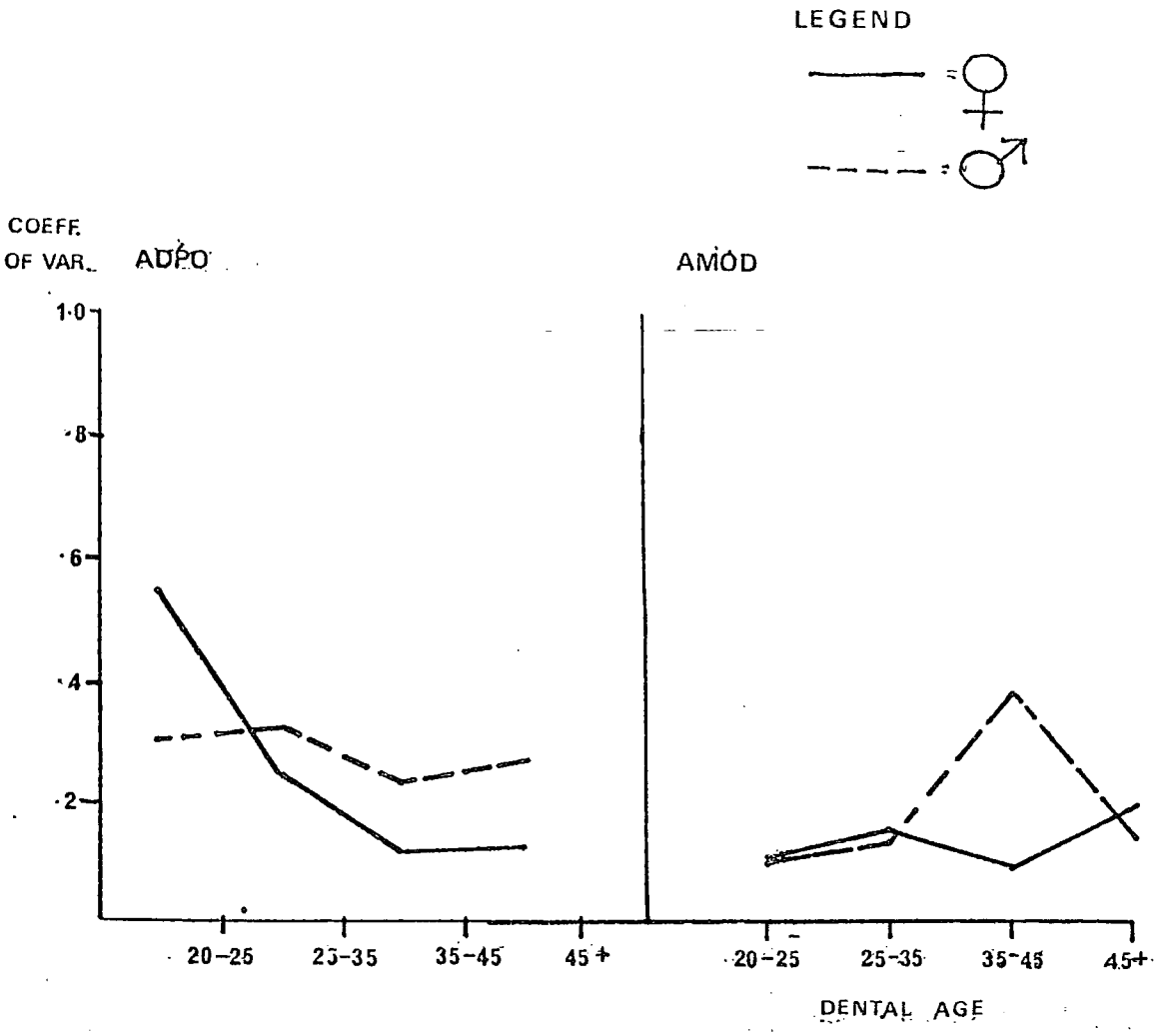


FIG. 7·9 COEFFICIENTS OF VARIATION FOR THE MICROPARAMETERS IN EACH DENTAL AGE GROUP.


LEGEND
♀
♂
COEFF
OF VAR
·6
·4
·2
0
TNSO
TOPL
1·0
·8
·6
·4
·2
0
TMOPL
CTHICK
20–25
25–35
35–45
45 +
20–25
25–35
35–45
45 +
DENTAL AGE


LEGEND
COEFF. OF VAR.
ADPO
AMOD
1·0
·8
·6
·4
·2
20–25
25–35
35–45
45+
20–25
25–35
35–45
45+
DENTAL AGE

7.8 Microparameters with Dental Attrition and Pubic Symphysis Metamorphosis.

The coefficients of correlation and the p values of relationships between the micpoparameters with dental attrition and pubic symphysis metamorphosis are given in Table 18 of Appendix B. Those showing significant values are presented in Table 7.6. Again correlations with M_1 in females have higher coefficients than with M_2 and the reverse is true in males. The lists of those significantly correlated with dental attrition is much the same in both sexes for those with dental age. Exceptions are CTHICK in females which shows a significant negative correlation with dental age but not with the attrition scores, and ADPO in males which is significantly correlated with both molar attrition scores but not with dental age. There are very few significant correlations for either sex with M_3, probably explained by the small sample sizes of M_3. The coefficient values are slightly higher with attrition scores than with dental age for males, but not particularly different for females.

The relationships to pubic symphysis metamorphosis are identical to those with pubic symphysis age. No correlations are found with pubic symphysis metamorphosis amongst males and only with ADPO for females. The coefficient value is slightly better for ADPO with pubic symphysis age than with the raw score, but only marginally.

The normalized score of cortical thickness was also

Table 7.6 Significant Correlations Between the Microparameters and the Dental Attrition and Pubic Symphysis Metamorphosis Scores.

M_1

Females		Males	
TNSO	•4751	TOPL	•3361
	•000		•002
ADPO	•4413	TNSO	•3313
	•000		•003
KNSO	•4359	ADPO	•2955
	•000		•006
CNSO	•3761	KPCL	-•3923
	•000		•007
KPCL	-•4132	CNSO	•2534
	•001		•010
TOPL	•3448		
	•001		
KNNH	-•4696		
	•008		

M_2

Females		Males	
TNSO	•4006	TNSO	•4265
	•000		•000
ADPO	•3931	KNSO	•4033
	•000		•000
CNSO	•3419	TOPL	•3996
	•000		•000
KNSO	•3363	CNSO	•3974
	•001		•000
KPCL	-•3100	ADPO	•3720
	•010		•001
		KPCL	-•4665
			•002

M_3

Females		Males	
KNSO	•3569	TMOPL	•3659
	•010		•006
		TOPL	•3498
			•009

Pubic Symphysis Metamorphosis

Females	
ADPO	•3442
	•006

compared to the attrition and metamorphosis scores. Again the overall result was the same as the non-normalized, no significant correlation with any of the molars or with the pubic symphysis

Summary

No significant sex differences exist in the distribution of age at death from any of the methods. The peak and mean values of age at death vary quite considerably between methods. The microage distributions correlate all one with another for females, but a number do not in males.

The distribution of microparameters show no sex differences for the internal measures but do for the external measure of cortical thickness. The microparameter distributions have a group which all correlate one with another for both sexes: KNSO, CNSO, TNSO, KPCL, KNNH, TOPL, ADPO. KNOF is part of this group in females but not in males. TMOPL, TNSO and TOPL correlate with one another in females but the directions of correlation involved seem paradoxical. A similar situation is found in males between KNSO, AMOD and CTHICK.

The distributions of the microparameters against dental age show that change in the measures of bone structure do not proceed linearly with age, but alter in the direction of age-related change at about 35 - 45 for internal parameters and 25 - 35 for cortical thickness. The age-related change

in the later age group is more marked in males for the internal parameters. This explains the lack of good correlation with dental age of either the microparameters or the microages, and explains how paradoxical relationships can occur since two directions of change are involved. The use of regression equations, therefore is not suitable for age estimations based on measures of bone structure.

Females show a greater consistency between the different microage and microparameter measures. Males tend to show a greater range of variation in the oldest age group.

The loss of cortical thickness is greater in females than males, but nowhere near comparable to modern standards. Females show greater variation in cortical thickness measures at all ages compared to males. The normalized measure of cortical thickness gives the same overall result as the non-normalized. Although the normalized coefficient of correlation with dental age is higher than the non-normalized, the sample size is smaller which means fewer individuals could be aged if a normalized value were to be used.

Correlations with the dental attrition scores suggest some significant relationships would be expected with dental age amongst both the microages and microparameters, which are not found. This concerns ADPO and the microage estimations based upon it, TAA and ADAGE and to a lesser extent TAB. Otherwise the results of correlation with dental age and dental attrition are very similar. There is a tendency

for correlation coefficients to be higher between micro-parameters and dental attrition than dental age. This probably merely reflects the fact that the range of dental attrition scores incorporated into each of the dental age categories up to 35 - 45 is greater than the range describing the older categories. Thus the age-related changes of the first part of life are more pronounced relative to the later changes in the reverse direction.

Correlations with pubic symphysis age and metamorphosis are minimal supporting the idea that the pubic symphysis method is less reliable.

Chapter 8. RESULTS: DEGENERATIVE JOINT DISEASE OF THE SPINE.

8.1 Columnal and Regional Scores with Dental Age and Pubic Symphysis Age.

The various composite scores for degenerative joint disease in the facet and disc joints have been constructed as described in the methods section, i.e. max grade, % of joints affected and % x max grade. All of the scoring systems were tested for correlation with dental age and pubic symphysis age using Spearman's rank correlation statistic. The results are presented in Tables 1 to 3 in Appendix C. All relationships were significantly correlated. Table 8.1 summarizes the findings by presenting the six highest correlation coefficients overall for each sex with dental age and pubic symphysis age.

The first most striking result is that the correlation coefficients are higher for both sexes with dental age than with pubic symphysis age. This is particularly true of the females, reflecting the discrepancy in females between the dental age and pubic symphysis age estimates. Both sexes show a number of scores with coefficients greater than ·7 with dental age whereas none of the scores have a coefficient of ·7 with pubic symphysis age. From hereon dental age is taken as the age of the population, a baseline to which the spinal

Table 8.1 The Highest Correlation Coefficients of Columnal and Regional Scores with Dental Age and Pubic Symphysis Age.

	Female			Male	
RANK	score	correlation coefficient	RANK	score	correlation coefficient
with Dental Age					
coeff. > ·7					
1	column combined % x max	·7408	1	lumbar combined %	·7263
2	lumbar combined % x max	·7400	2	column combined %	·7180
3	lumbar combined max	·7390	3	cervical combined % x max	·7024
4	column combined %	·7373	4	cervical facets % x max	·7002
5	lumbar combined %	·7341			
6	cervical combined %	·7165			
coeff. > ·6					
			5	lumbar combined % x max	·6991
			6	cervical combined %	·6911
with pubic symphysis age					
coeff. > ·6					
1	lumbar combined max	·6340	1	column combined %	·6083
			2	column combined % x max	·6066
coeff. > ·5			3	lumbar combined %	·6006
2	lumbar combined % x max	·5966	4	thoracic combined %	·5938
3	column combined max	·5670	5	lumbar combined max	·5787
4	column combined % x max	·5616	6	lumbar combined % x max	·5783
5	lumbar disc % x max	·5526			
6	lumbar disc max	·5496			

scores are compared.

Secondly, females have higher correlation coefficients for the first four scores than males and more female scores correlate with coefficients greater than ·7, eight in total as opposed to four amongst the male scores. However, throughout the entire series it can be seen that amongst the best correlations females have higher coefficients whereas at the lower end males show the higher figures. This could indicate a greater consistency amongst males between the different spinal scores. This will be examined in more detail.

Thirdly, none of the top scores with dental age coefficients involve the thoracic region. This region is most commonly involved by those pathologies which involve the joints such as to possibly predispose to joint degeneration or cause fusion of joints through ossification of ligaments. No attempt to distinguish these cases or to exclude them has been made for reasons outlined in the methods section.

Fourth, all except the last male score for correlation with dental age are combined scores, supporting the suggestion that a certain independence exists between the two joint types while both are still age-related in their degeneration, thus enhancing the correlation coefficient with dental age.

Lastly, the two sexes have different sequences of results, the cervical region playing a more prominent role

relative to other scores amongst males than amongst females.

In conclusion, the first two scores having high coefficients with dental age common to both females and males are the combined extent (%) scores for the column and lumbar region. These two measures could provide the best means of aging by degenerative joint disease.

The distributions of those two high correlating scores are presented as cumulative % frequency curves in Fig. 8.1. If the combined extent (%) scores for the columns and the lumbar region are possibly to be translated to an age estimate, these graphs could represent the mortality curve of the Poundbury population. As can be seen from the graphs, a sex difference exists in both cases such that a greater proportion of the females have a lower percentage of joints affected than males. Translated into a mortality curve directly, this would imply that females were dying earlier than males, a result consistent with the mortality profile indicated by dental age.

The χ^2 and U statistics for sex differences in frequency distributions are presented for the whole series in Tables 19 to 22 of Appendix C. Those showing significant sex differences are given here in Table 8.2. In all cases the difference in the sexes is one of higher scores in males. Not all the scores do show significant differences and there is a clear pattern in this respect. Most of the facet scores do not show any significant differences between the sexes.

FIG. 8·1 CUMULATIVE % FREQUENCY OF THE COMBINED EXTENT (%) SCORES IN THE WHOLE SPINE AND IN THE LUMBAR REGION.

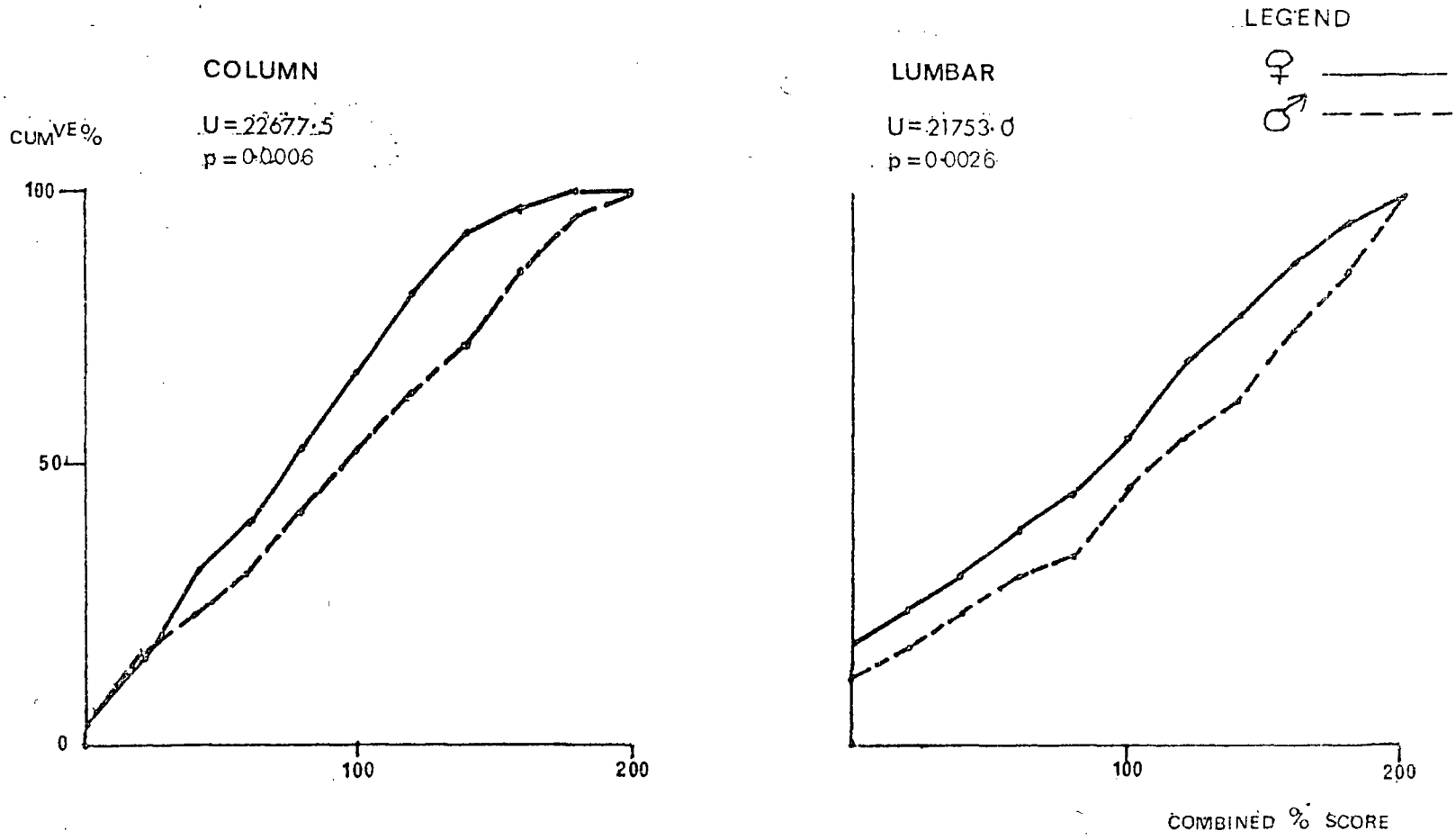


Table 8.2 Significant sex differences found in distributions of max, % and % x max. scores.

score: max	χ^2	p
column disc	30•38255	0•0000
cervical disc	16•01697	0•0030
thoracic disc	25•78744	0•0000
lumbar disc	32•90105	0•0000

score: max	U	p
column combined	22973•5	0•0011
cervical combined	19389•0	0•0016
thoracic combined	24304•5	0•0079
lumbar combined	21953•0	0•0037

score: %	U	p
column disc	23112•5	0•0003
cervical facet	24775•0	0•0014
cervical disc	18771•5	0•0000
lumbar disc	20943•5	0•0000
column combined	22677•5	0•0006
cervical combined	18337•0	0•0001
lumbar combined	21753•0	0•0026

score: % x max	U	p
column disc	22104•0	0•0000
cervical facet	25120•5	0•0031
cervical disc	18975•5	0•0001
thoracic disc	25038•5	0•0087
lumbar disc	19480•5	0•0000
column combined	22182•0	0•0002
cervical combined	18884•0	0•0005
thoracic combined	24105•5	0•0061
lumbar combined	20651•5	0•0002

The exception is the percentage of joints affected in the cervical region, which is echoed in the % x max. score. All the others are disc scores and combined scores. The thoracic region shows no significant sex differences in the % of joints involved but does in the severity observed in discs. The column, cervical, and lumbar disc scores consistently show differences in severity and extent sufficiently great enough to be restated in the combined scores. Again, a distinction between facet and discs is observed and the question is raised whether the scores should be directly translated to age estimates or whether the sexes need to be treated apart. If the sex differences in the cumulative frequency curves of disc and combined scores are taken to represent mortality curves, the assumption is being made that the degeneration proceeds uniformly across the sexes. This would nessitate a greater rate of degeneration amongst females in the facet joints giving no sex differences overall in distribution. On the other hand, if males have a greater rate of degeneration of disc joints than females giving a false impression of later mortality, the facet joints could be proceeding in degeneration at comparable rates in the two sexes, giving a truer picture of similar mortality curves. It is necessary to try to assess if any differences exist between the sexes in age of onset, severity of involvement by age, percentage of joints affected by age and the rate of increase with age of these measures.

8.1.2 Columnal and Regional Scores with Dental Attrition and Pubic Symphysis Metamorphosis.

The correlation coefficients are given for the entire series of spine scores with attrition on M_1 and M_2 and the pubic symphysis scores in Tables 4 to 6 of Appendix C. Some differences in order of highest coefficients are found comparing the order of correlations with dental age and with attrition. The high correlation with dental age of the cervical facets in males would not have been predicted from the coefficients with attrition scores. No particularly higher coefficients overall are found, confirming that no distortion in the relationship is occurring through the conversion from attrition score to age estimate. An interesting sex difference is that females show their highest coefficients with M_2, whereas in males it is with M_1. The coefficients with pubic symphsysis score are higher in males than with pubic symphysis age, but if anything lower in females, suggesting the apparent problem in using the pubic symphysis is not one of distortion from raw score to age estimate but one in attribution the stages of metamorphosis in the first place.

8.2 Individual Joints of the Spine.

8.2.1 Preservation.

As expected, the most frequently present vertebrae were the largest ones. There is an increase in presence from the first cervical vertebra through to the fifth lumbar

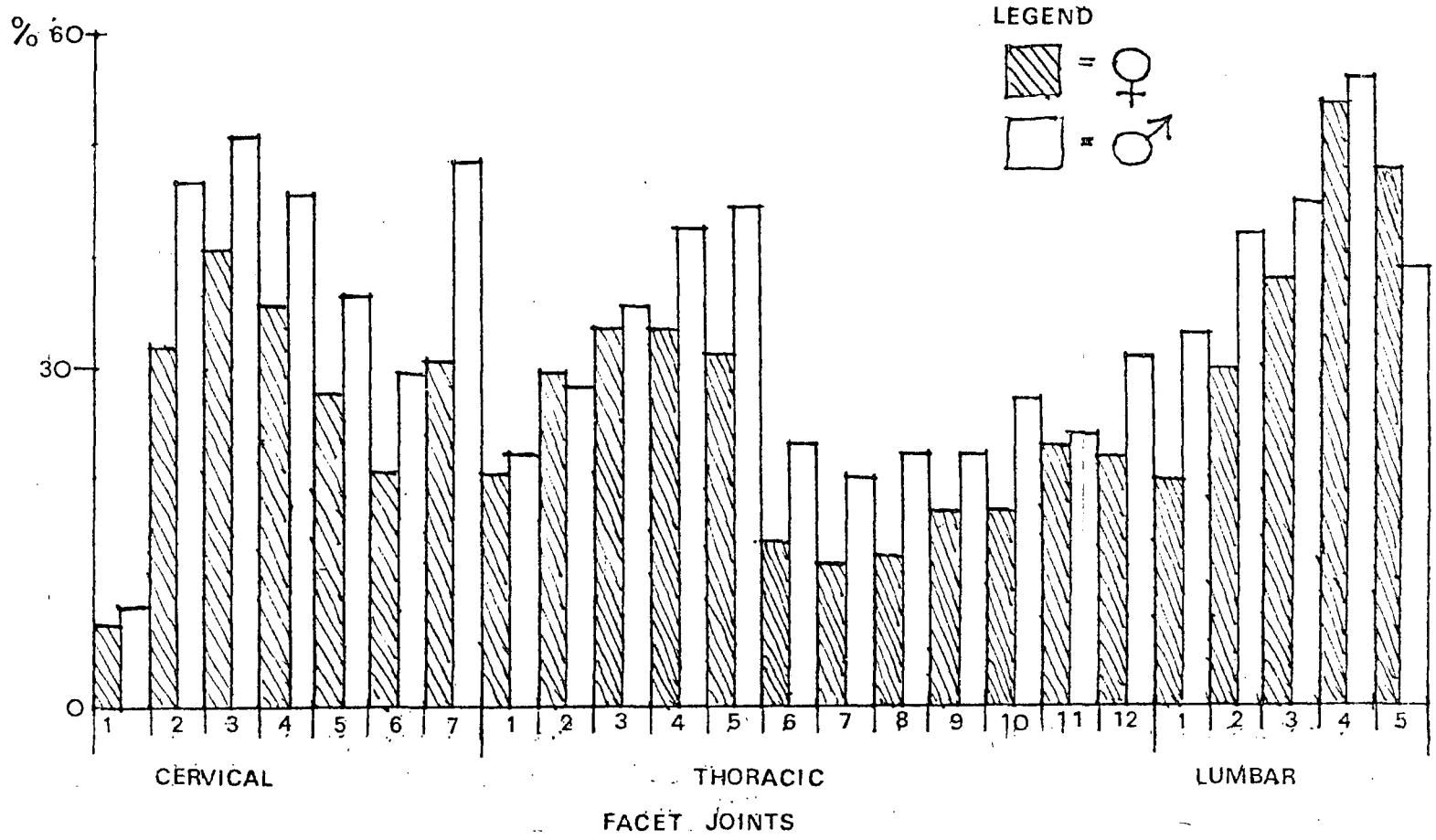
for both sexes. There is no preferential preservation of vertebrae between the sexes except for the atlas and axis where 8% and 6% more respectively were recovered from male remains. The % present of each vertebra are given in Table 7 in Appendix C.

8.2.2 Distribution of Degenerative Joint Disease throughout the Spine.

The distributions by joint throughout the spine of any degeneration of the facet and disc joints are illustrated in Fig.s 8.2 and 8.3 respectively. The order of greatest involvement in the population of the joints has been drawn up for each sex for facet and disc joints. The distribution in the spine is then compared for the sexes using the Spearman rank correlation statistic. Both the facet distribution and the disc distribution show a significant correlation between the sexes. However, applying the same test to a comparison of facets and discs showed no significant correlation between the two for either sex. These calculations are given in Tables 10 to 12 in Appendix C. Here, then is evidence of a difference in degeneration of facet and disc joints at the simplest level, that of the pattern of involvement through the spine. Different local factors, most likely biomechanical ones, are operating on the facet and disc joints.

The pattern throughout the spine for both facets and discs of distinctive outcrops and relative troughs in

FIG. 8·2 % FREQUENCY OF OSTEOARTHROSIS IN EACH FACET JOINT.



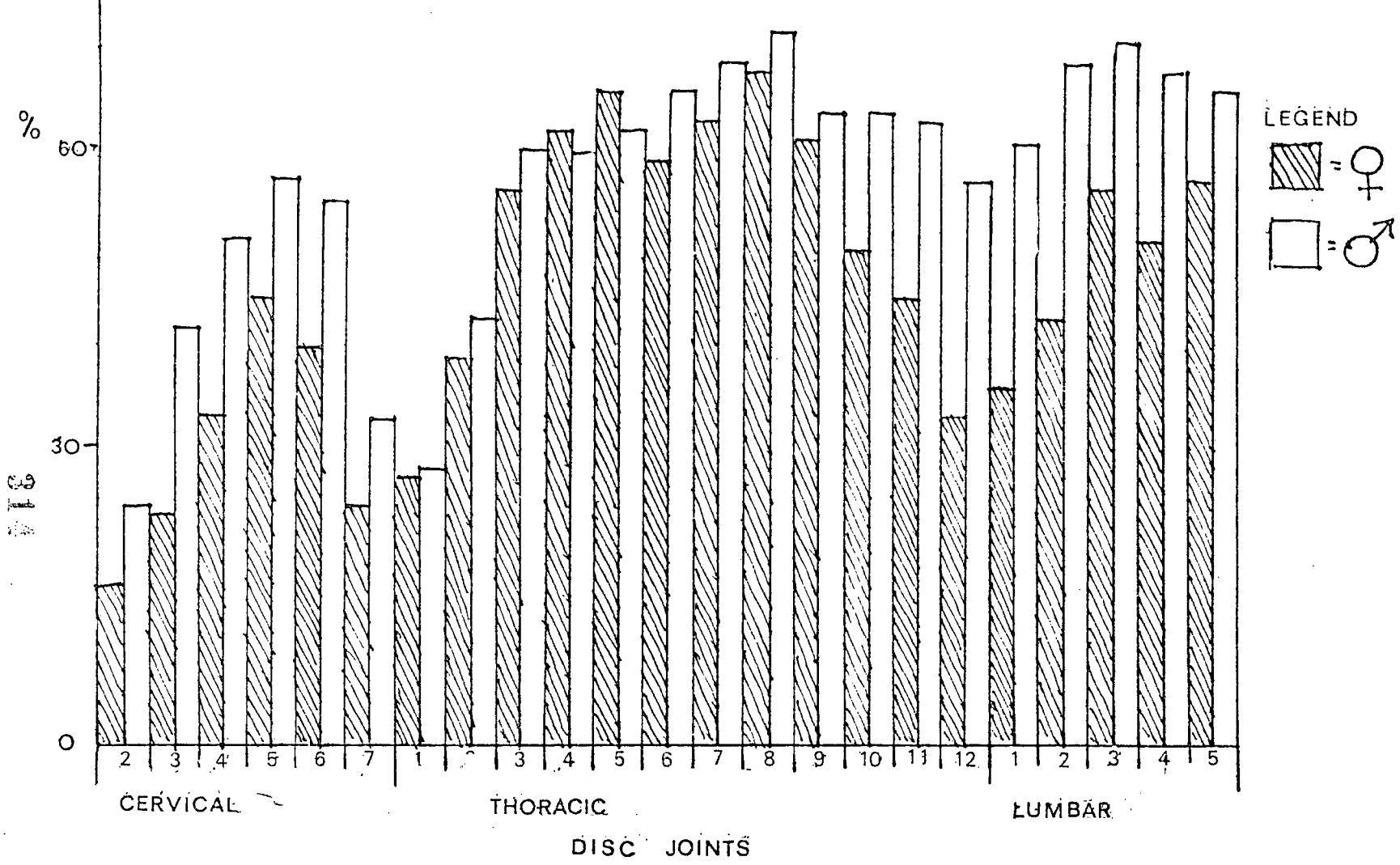


FIG. 8·3 % FREQUENCY OF OSTEOPHYTOSIS IN EACH DISC JOINT.

frequency of involvement corresponds with findings of other workers (see Table 8.3). The facet discs show peaks of frequency at C3, T5 and L4 for both sexes, plus a peak at C7 in males. The minimum points, or troughs are at C6 and T7. This corresponds very well with the findings of Shore, who has peaks at C3, C7, T4 and L2, except in the lumbar region where the peak in the Poundbury population is lower. Ingelmark et al.'s distributions of involvement of facets is also much the same with peaks at C3 and T4, but they do not find an outcrop at all in the lumbar region. The disc joint peaks in Poundbury are at C5, T8 and L3/5 with low points at C7/T1 and T12. The cervical and lumbar peaks correspond most exactly to Nathan's peaks, but the thoracic is higher at T8 than Nathan's finding at T10. A T8 peak is reported by both Shore and Ingelmark et al., but both of these have a slightly higher peak in the lumbar area. The cervical peaks of Shore and Ingelmark et al. are one joint higher and one joint lower respectively than Poundbury, but Shore's trough is at the same level as Poundbury, so the cervical outcrop as a whole is best seen as corresponding to Shore's.

Overall the pattern shows the highest levels of osteophytosis in the thoracic region, which has only a slightly greater involvement than the lumbar region. This agrees reasonably well with other reports where the lumbar is quite often the most involved. However, the facet joints of Poundbury show the greatest involvement in the lumbar region which is quite different from the report of Stewart on Americans, Eskimo and Pueblo where the mid-cervical and

upper thoracic are found to be greatly affected, but not the lumbar region. The suggestion by Lindblom that osteophytosis in the lumbar area could be higher in earlier populations than the modern low site at L5 is not supported here by the peak found from L3 to L5. A certain correspondence is found between the maximum disc joints involved and the minimum points of the facet joints affected. This relationship does not hold in reverse; the maximum facet joints involved do not correspond to the minimum disc joints.

The peaks and troughs found in distribution of involvement in Poundbury are summarized and compared with other workers in Table 8.3.

The frequency of involvement by superior and inferior portions of the joints is given in Tables 13 and 14 of the Appendix for facets and discs respectively. In the facet joints, the superior portion is more affected through the cervical region to the early thoracic. The middle thoracic is mainly much the same for both portions. The lower thoracic region again shows a greater contribution from the superior portion. This is reversed in the lumbar region where the inferior portion is more affected. None of the other workers have discussed facet joints so a comparison with other data is not possible.

In the disc joints, a less consistent pattern emerges with a trend of greater inferior involvement in the early cervical region. Thereafter in the lower cervical and

Table 8.3 The maximum and minimum joints of % involvement in Poundbury and other samples.

FACETS:

Worker	Sample	Max pt.s	Min pt.s
	Poundbury	C3 C7 male T5 L4	C6 T1 male T7
Shore		C3 C7 T4 L2	C6 T8
Ingelmark	Aebelholt	C3 T4	C6 T8 (all of L)

DISCS:

Worker	Sample	Max pt.s	Min pt.s
	Poundbury	C5 T8 L3/5	C7/T1 T12
Shore		C4 T8 L2	T1 T12
Ingelmark	Aebelholt	C6 T8 L2	T2 T12
Nathan	Todd Collection	C5 T10 L4	

thoracic regions the predominance of one portion over the other varies from joint to joint and varies between the sexes suggesting no consistency. In the lumbar region a predominance of the inferior surface over the superior is again observed. Both Nathan (1962) and Ingelmark et al. (1959) record a higher contribution from the inferior portion in the lumbar region but do not find the inferior surface predominating in the cervical region, rather the opposite in fact. The results here therefore support neither Nathan nor Ingelmark's suggestions of biomechanical effects except in the lumbar region where all three agree.

As regards the side of the joint involved, there is no particularly consistent pattern among the facet joints except in the early thoracic region where the right hand side predominates over the left. Again the other workers have not examined facet joints for comparison. By contrast, the characteristic predominance of the right hand side over the left is recorded in the disc joints of the thoracic spine. This is consistent with the findings of other workers (see Nathan, 1962). The frequencies of left and right involvement of facet and disc joints are given in Tables 15 and 16 of Appendix C.

8.2.3 Sex Differences in Frequency of Involvement and Severity of Each Joint.

Sex differences in any involvement at all of degeneration are assessed using the χ^2 statistic. The χ^2 and p values

are given for each joint, facets and discs in Table 17 of Appendix C. Those showing significant differences are presented in Tables 8.4 and 8.5, giving the % frequencies of involvement of each sex. Fewer facet joints show significant sex differences than discs. The most consistent pattern of sex differences is in the disc joints from T11 through to L4. In all cases of sex differences, it is the males who show a greater frequency of involvement than the females. The sex differences of the facet joints and C2, 7 and T5 correspond to the maximum points of involvement of the facets. The difference found at C7 would have been predicted as this represents a maximum point in males that is not equally marked in females. The lower facets showing sex differences do not correspond to maximum or minimum points. There is no relationship between the discs showing significant sex differences and those of greatest involvement overall.

The χ^2 and p values for each joint, facets and discs for sex differences in frequency of severity grade are given in Table 18 of Appendix C. Again those showing significant differences are presented here in Tables 8.6 and 8.7 giving the % in each severity grade. In all cases, the difference involves a higher proportion of males in the more severe grades. From these figures, the far greater sex differences in the disc joints is again found, particularly from the lower thoracic downwards.

Table 8.4 % Frequency of involvement of facet joints showing differences between the sexes ($p < 0{\cdot}01$)

vertebral facet joint	% of affected females	% of affected males
C2	32·2	46·8
7	30·5	48·9
T5	31·6	45·0
10	17·5	28·3
L1	21·0	33·7
2	31·0	42·7

Table 8.5 % Frequency of involvement of disc joints showing significant differenced between the sexes (p 0·01)

vertebral disc joint	% of affected females	% of affected males
C3	23·5	41·6
4	33·2	51·0
6	40·3	55·4
T11	45·0	63·0
12	33·1	56·8
L1	36·0	60·6
2	43·3	68·7
3	55·8	70·8
4	50·7	68·2

Table 8.6 % Frequency of severity grades in facet joints showing significant differences between the sexes.

	% of females					% of males				
Grade	0	1	2	3	4	0	1	2	3	4
vertebral facet joint										
C7	69·5	22·1	8·0	0·4	0	51·1	38·2	9·0	1·3	0·4

Table 8.7 % Frequency of severity grades in disc joints showing significant differences between the sexes.

	% of females					% of males				
Grade	0	1	2	3	4	0	1	2	3	4
vertebral disc joint										
C3	76·5	16·3	5·1	1·5	0·5	58·4	28·9	8·6	2·0	2·0
4	66·8	22·1	8·0	3·0	0	49·0	33·2	13·3	3·6	1·0
T5	34·0	56·9	8·0	0	1·1	38·3	43·4	11·7	2·6	4·1
8	31·8	47·7	14·2	5·1	1·1	27·9	35·5	21·3	6·6	8·7
9	38·5	43·6	15·1	0·6	2·2	35·7	31·3	17·0	5·5	10·4
10	50·3	38·2	9·2	1·7	0·6	36·3	36·3	16·2	4·5	6·7
11	55·0	36·7	8·3	0	0	37·0	35·9	16·8	6·5	3·8
12	66·9	27·6	3·7	1·8	0	43·2	37·9	15·4	3·6	0
L1	64·0	27·4	6·7	1·2	0·6	39·4	28·3	26·1	5·6	0·6
2	56·7	33·2	7·0	2·1	1·1	31·3	34·9	26·7	5·6	1·5
3	44·2	39·3	14·6	1·5	0·5	29·2	32·7	30·2	6·9	1·0
4	49·3	32·6	15·9	2·2	0	31·8	36·4	24·8	7·0	0

8.2.4 Individual Facet and Disc Joints and Dental Age.

The correlation coefficients and p values for each facet and disc joint degeneration with dental age are given in Appendix C, Table 23 for any involvement with dental age and Table 24 for severity with dental age. The six highest correlation coefficients for each set are presented in Table 8.8. The inclusion of severity grades in the calculations does not much alter which joints show the best correlation coefficients, but it does improve the correlation coefficient slightly. The important joints in age related degeneration are the lumbars and cervicals. The cervical joints are more dominant in the facet scores particularly in males, whereas the lumbar joints dominate in the disc correlations. The males show consistently high correlation coefficients throughout the top six, all except one over ·5. From these it would be predicted that good correlations with dental age might be found for males in the scores based on facet joints than females, particularly in the cervical area. Scores based on lumbar disc joints should prove highly correlated with dental age, particularly in females. Thoracic joints of either type are not as highly related to dental age, and must be more susceptible to other factors than the cervical and lumbar joints.

Table 8.8 The Highest Correlation Coefficients of the Disc and Facet Joints with Dental Age by Any Involvement and Severity.

	FACETS				DISCS			
	Females		Males		Females		Males	
Rank	Joint	Coeff.	Joint	Coeff.	Joint	Coeff.	Joint	Coeff.
Any involvement								
1	L4	•5325	C3	•6413	L3	•6237	L4	•5974
2	C3	•5075	C4	•5638	L4	•6207	L3	•5867
3	C2	•4863	C2	•5456	L5	•5920	L5	•5461
4	C4	•4857	C6	•5383	C5	•4762	C1	•5207
5	L5	•4675	L3	•5327	L2	•4757	T8	•5080
6	L2	•4589	L2	•5014	T6	•4615	L1	•4973
Severity								
1	L4	•5524	C3	•6752	L3	•6386	L4	•6176
2	L5	•5288	C4	•5816	L5	•6298	L3	•5886
3	C3	•5264	C2	•5775	L4	•6171	C1	•5481
4	C4	•4953	C6	•5405	C5	•4915	L1	•5478
5	C2	•4946	L3	•5339	L2	•4806	L2	•5160
6	L2	•4723	L4	•5131	T6	•4747	L5	•5229

8.3 Composite Scores.

8.3.1 Relationships between Composite Scores.

The correlation coefficients of the maximum score with the corresponding % score using Spearman's rank correlation statistic are given for each sex in Table 8.9. All are highly significant. Since both the increase in maximum grade and in the percentage of joints involved are mediated by age, the significance is not surprising. However, the coefficients are high, all $>\cdot 7$ and the majority $>\cdot 8$ implying severity and extent do increase very closely together. This suggests that the combined score of % x max would not be expected to provide a very much better correlation with age than either alone. As can be seen from the correlation coefficients of the whole series with age, it is by and large true that % x max does not make a great improvement over %, though some improvement over the simple severity grades is found. Particularly high correspondence between extent and severity is seen in the cervical region, especially amongst females. Males show a better correspondence between the two scores in lumbar facets, thoracic discs, thoracic combined and the column discs. In all the others the correspondence is closer in females, but the differences are often very slight.

The relationship between facets and discs is examined using Spearman's rank correlation statistic between the corresponding facet and disc scores of all three composite

Table 8.9 Correlation Between Maximum Severity and Extent (%) Spine Measures.

Spine Score	Max vs %	Females	Males
Facets	Column	•7806	•7375
	Cervical	•9200	•8658
	Thoracic	•8396	•8392
	Lumbar	•8281	•8602
Disc	Column	•7732	•8099
	Cervical	•9110	•8589
	Thoracic	•7521	•7776
	Lumbar	•8092	•7798
Combined	Column	•8446	•8390
	Cervical	•9276	•9065
	Thoracic	•7978	•8420
	Lumbar	•8652	•8684

p = 0•000 in all cases

Table 8.10 Correlation Between Facet and Disc Spine Measures.

Spine Score		Females	Males
MAX			
	Column	•5886	•6081
	Cervical	•5648	•5957
	Thoracic	•4152	•3970
	Lumbar	•5278	•5207
%			
	Column	•5526	•6301
	Cervical	•5178	•6395
	Thoracic	•3705	•4710
	Lumbar	•4914	•5267
% x MAX			
	Column	•6232	•6830
	Cervical	•5760	•6850
	Thoracic	•4171	•4907
	Lumbar	•5500	•5574

p = 0•000 in all cases

types i.e. max, %, % x max. The coefficients are given in Table 8.10. Again, all are significant but the coefficients are much lower than those seen in the preceding table on severity and extent. The highest coefficients are seen in males between cervical facets and discs % x max scores, the total column % x max scores, the cervical % scores and column % scores. The females highest coefficients are lower than any of these four male values but involve similar measures: - the column % x max scores, the column max scores, the cervical % x max scores and the cervical max scores. The lowest coefficients in both sexes involve thoracic joints. These results suggest additional information can be gained by combining facet and disc measures to enhance the correlation to age, of degeneration in both joint types is age-related. As seen from the highest correlation coefficients with age, the majority did involve combined scores.

Finally, the relationship between regions of the spine is examined using Spearman's rank correlation statistic for the closeness between the same composite scores of each region. The coefficients are given in Table 8.11. Again, all are significant. Males show higher coefficients than females in all cases except in the max cervical facet score's relationship to the same score in the thoracic and lumbar regions. The higher values in males would suggest a greater uniformity in degeneration throughout the column, as opposed to more localization in the females.

Table 8.11 Correlation Between Comparable Regional Spine Measures.

Spine Score		Female		Male	
		Thoracic	Lumbar	Thoracic	Lumbar
MAX Facets					
	Cervical	•4935	•6147	•4540	•5439
	Thoracic		•4357		•5237
Discs					
	Cervical	•5520	•5706	•5633	•6400
	Thoracic		•5223		•6497
% Facets					
	Cervical	•5280	•6178	•6414	•6428
	Thoracic		•4480		•6278
Discs					
	Cervical	•6421	•6418	•6794	•6278
	Thoracic		•6897		•7798
% x MAX Facets					
	Cervical	•5451	•6592	•5969	•6259
	Thoracic		•4620		•6427
Discs					
	Cervical	•6583	•6652	•6695	•7077
	Thoracic		•7082		•7404

p = 0•000 in all cases

8.3.2 Incidence of Degenerative Joint Disease and Dental Age.

The % frequency of individuals in each dental age group having some level of involvement i.e. at least one joint showing grade one degeneration, are shown in Fig. 8.4 for the facet and disc joints. The % affected in the regional and column measures in each age group are given in Table 25 of Appendix C.

In the first dental age group of 20 - 25, a high proportion of individuals are already affected. In the facet joints, 61% of females and 51% of males show some degeneration while in the disc joints, 67% of females and 63% of males are affected. About 3% of males never show any degeneration of the facet joints at all, whereas 100% females are involved by the 35 - 45 age group. Both sexes reach their maximum frequency of affected individuals by 35 - 45. The disc joints are affected in 100% of males by the 35 - 45 age group and in all females by the oldest dental age group of 45+. The χ^2 values for incidence in the first age category do not show a significant difference between the sexes for either facet or disc involvement.
(χ^2= 2·455 for facets; 0·549 for discs.)

In the regional figures, presented in Appendix C, about 10% of both sexes never show any involvement of the cervical or thoracic facet joints. All the population shows some involvement of the thoracic discs by 45+. Some males never have any involvement of the lumbar facets (6%) or disc

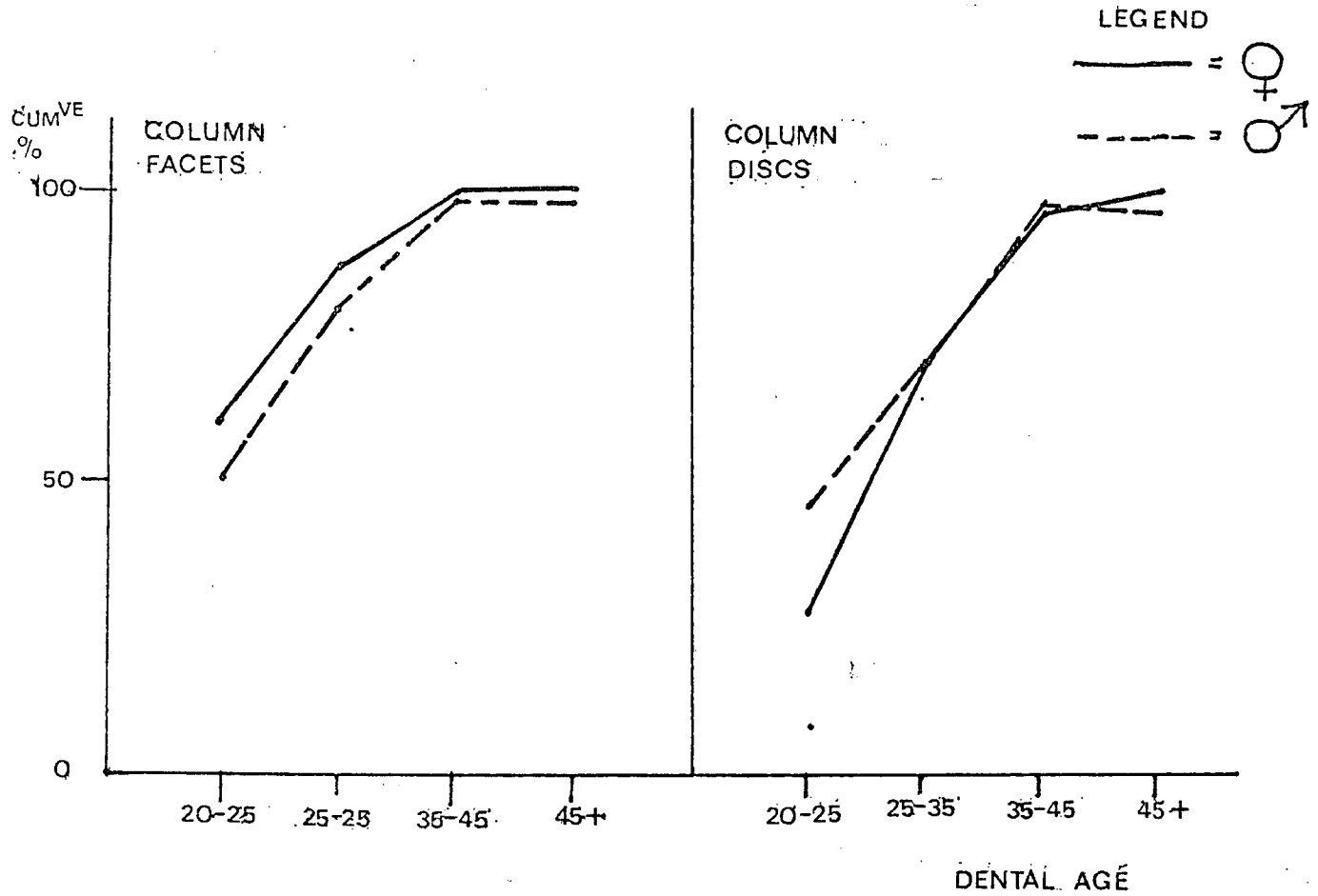


FIG. 8·4 CUMULATIVE % FREQUENCY OF INVOLVEMENT OF THE COLUMN FACET AND DISC JOINTS PLOTTED BY DENTAL AGE GROUP.

joints (4%).

The fact that over 80% of the population has some level of involvement in the facet joints and in the disc joints by the 25 - 35 dental age group illustrates the ubiquitous nature of degenerative joint disease. It also underlines the necessity for a more discriminating scale of measurement of degenerative joint disease than incidence. All incidence can tell the anthropologist is that a skeleton without any trace of degenerative joint disease must be under 35 years of age.

8.3.3 The Maximum Severity Measure and Dental Age.

The Mann-Whitney U test has been run for the distributions of the maximum severity spine scores for differences between the sexes in each dental age group. The U and p values are given in Table 26 of Appendix C. Since the division into subsamples by sex and dental age reduces the numbers in each subgroup, significance is accepted as $p < 0{\cdot}05$. No significant differences are found between the sexes in the first two dental age groups. In the 35 - 45 age group, males show a significantly higher distribution of scores than females in lumbar disc severity. This difference, however, is closed by 45+, indicating a greater progression of severity in females than in males between the two dental age groups 35 - 45 and 45+. The only other significant difference is found in dental age group 45+ in the cervical facet joints, where again males show a higher distribution of

severity scores than females. Presumably this represents the end product of a greater rate in the progression of severity in males throughout life in the cervical facet joints, reaching a significant difference by the oldest age group. The greater involvement of the cervical region in general, and particularly of the facet joints, in males has been noted already.

The cumulative % frequency of individuals with each maximum grade is plotted by dental age for the columnal and cervical facet and disc scores in Fig.s 8.5 and 8.6 respectively. The % of individuals in each dental age group for these and the rest of the regional scores are given in Tables 27 and 28 and graphs for the thoracic and lumbar regions in Fig. 1 and 2 of Appendix C.

Although no significant sex differences are found with the Mann-Whitney U statistic, differences can be seen between the two sexes from the graphs, perhaps better termed as trends. Dental age group 45+ distinguishes badly from group 35 - 45 in severity for females. This reflects the tendency for females not to show a very large proportion of individuals with severe grades in any dental age group. In the last two dental age groups, 75% of the female population is accounted for by grades 1 and 2 of severity in both facets and discs, whereas only 50% of the male population in the 45+ age group is accounted for by grades 1 and 2. A clearer demarcation is found in males in the cervical facets, but less so for females, a sex difference which is significant. The plots of the other

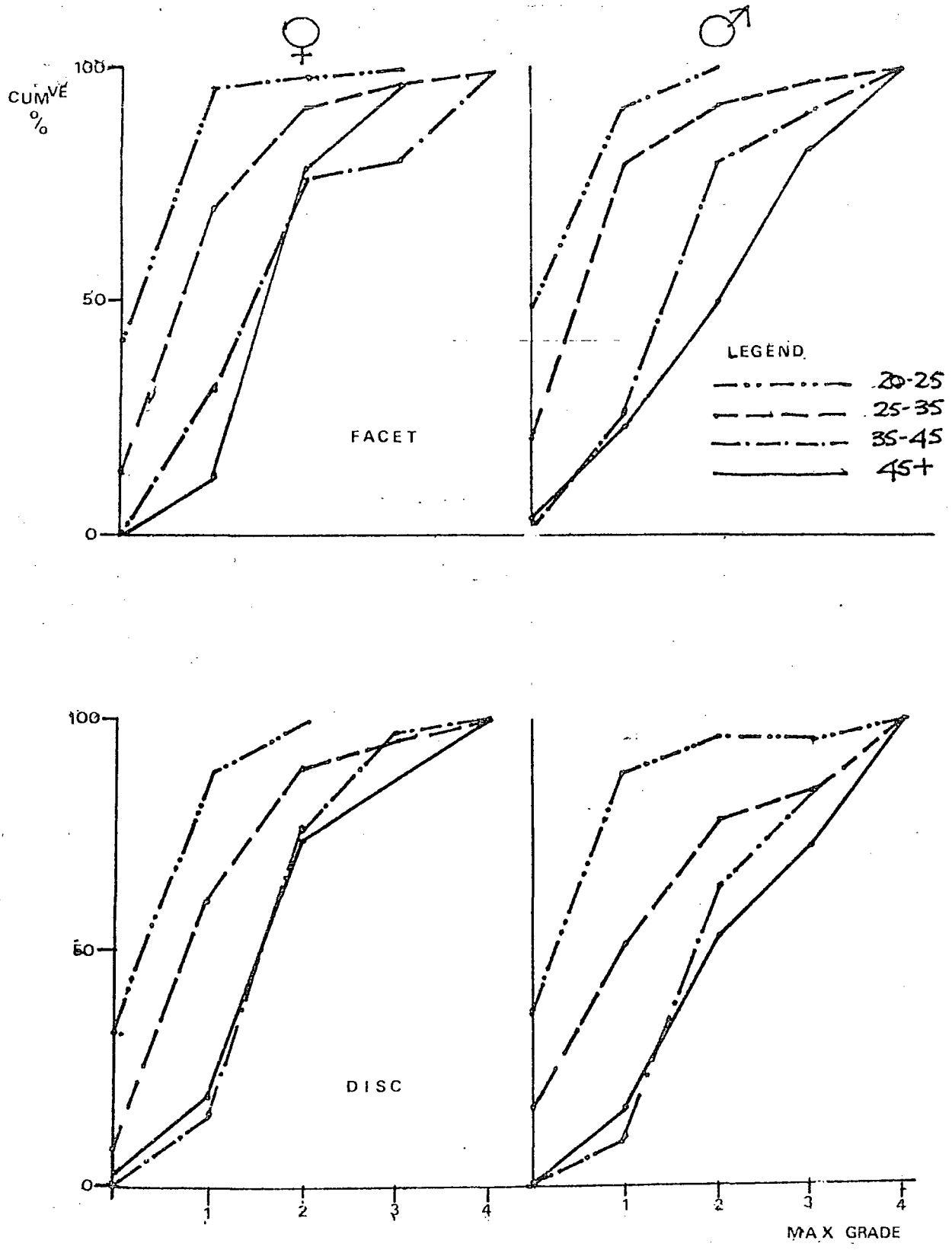


FIG. 8·5 CUMULATIVE % FREQUENCIES OF THE MAXIMUM GRADES OF THE COLUMN FACET AND DISC JOINTS PLOTTED BY DENTAL AGE GROUP.

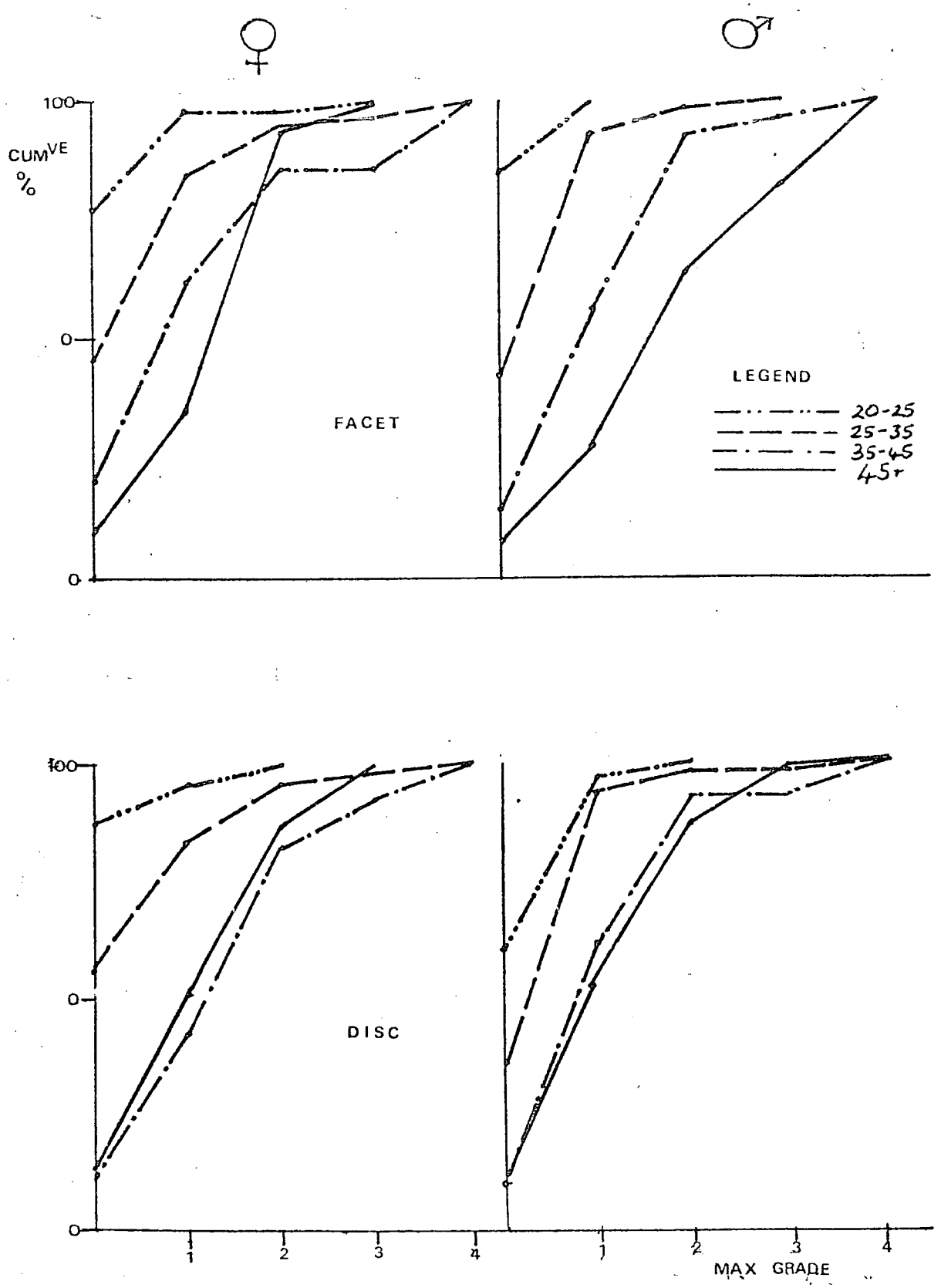


FIG. 8·6 CUMULATIVE % FREQUENCIES OF THE MAXIMUM GRADES OF THE CERVICAL FACET AND DISC JOINTS PLOTTED BY DENTAL AGE GROUP.

regional severity scores, in Appendix C, show very little distinction between the 35 - 45 and 45+ dental age groups.

It seems, therefore, that there is no reason to expect a sex difference in the age of onset of degenerative joint disease, nor does there appear to be any differences in the increase in severity with age, except in those cases outlined, particularly the cervical facets.

8.3.4 The Extent (%) Measure and Dental Age.

The results of the Mann-Whitney U test for sex differences of distribution in each dental age group are given in Table 29 of Appendix C. Far more significant differences are found between the sexes in the measures of the number of joints involved than was the case in the severity measures. In all instances, the difference represents a higher distribution of scores for males. The lumbar scores for discs and for the combined facets and discs show differences between the sexes in the 35 - 45 and in the 45+ dental age groups. In addition the significance of the difference increases between these two age groups. This then, describes a steadily greater progression in males than in females of the number of joints affected in the lumbar region, particularly of the disc joints. The combined cervical score also records an increasing difference between the sexes, resulting in a significant difference by the 45+ dental age group. The other cases showing significant differences are all in the 35 - 45 age group, but have decreased in

significance by the 45+ age group, in some cases, no longer representing a statistical sex difference. This describes a faster rate of increase in extent of degenerative joint disease in males until the 35 - 45 age group, at which point it appears as if a plateau is reached in extent of involvement, so the progression slows. The females continue to progress up to this plateau extent of involvement, thereby progressing in extent at a relatively faster rate than males in the later age group and decreasing the difference in extent of involvement between the two sexes. Significant differences are still found between the sexes in the oldest age group in the column disc scores and the cervical facet scores. The column facet, column combined, thoracic facets and thoracic combined are significant at 35 - 45, but no longer so by the 45+ age group.

In Fig.s 8.7 and 8.8 the cumulative % frequency is plotted by dental age group in the extent of degeneration for the column and lumbar combined scores respectively, since these two are of particular interest for aging. The tables of data and fig.s for the rest of the series are given in Table 30 and Fig.s 3 to 12 in Appendix C. A far clearer demarcation between the dental age groups is seen here, particularly between the 35 - 45 and 45+ dental age groups than found with the severity score.

In the column graphs, 70% of the female and male populations are accounted for in the 25 - 35 dental age group by combined % scores of 94 and 80 respectively,

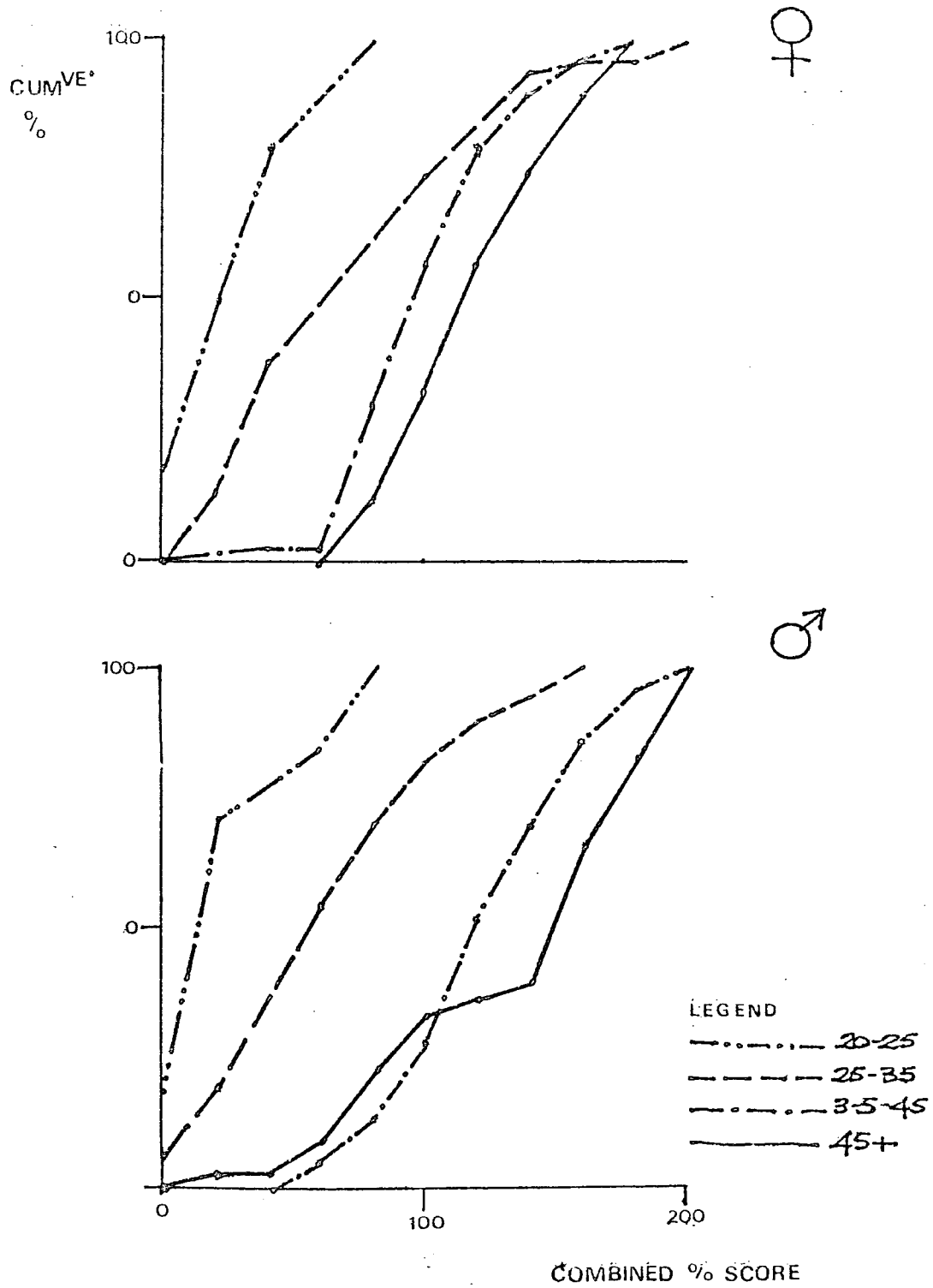


FIG. 8·7 CUMULATIVE % FREQUENCIES OF THE COMBINED EXTENT (%) SCORES WITHIN THE WHOLE SPINE PLOTTED BY DENTAL AGE GROUP.

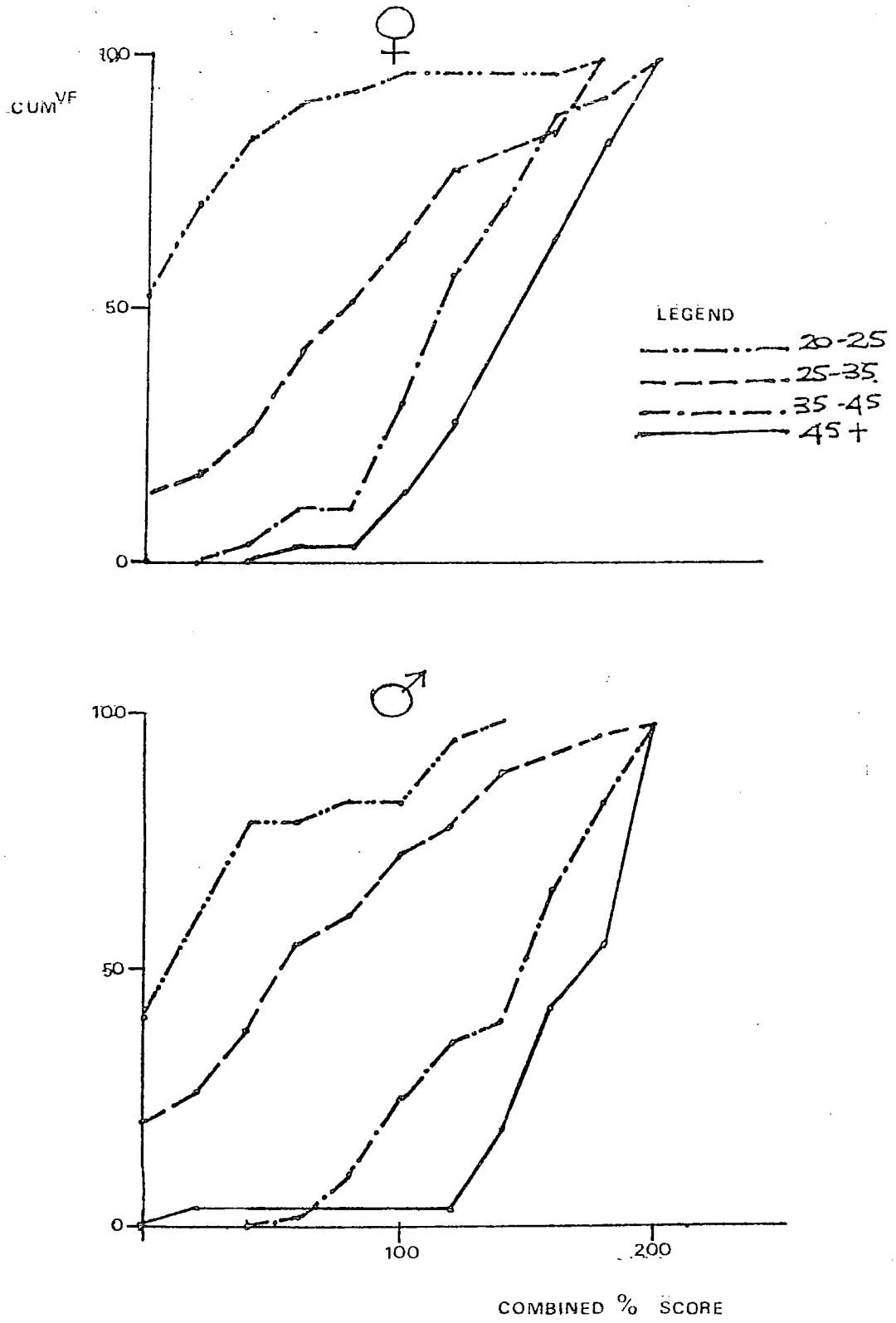


FIG. 8·8 CUMULATIVE % FREQUENCIES OF THE COMBINED EXTENT (%) SCORES WITHIN THE LUMBAR REGION PLOTTED BY DENTAL AGE GROUP.

whereas in the oldest age groups these scores only accounted for about a quarter of the population. 70% of females and males in the 35 - 45 age group are not accounted for until combined % scores of 112 and 140 each and the necessary scores for 70% of the 45+ dental age group are 134 and 164. The lumbar region is even better distinguished particularly in males where only 4% of the oldest dental age group are accounted for by the score (94) that accounts for 70% of the 25 - 35 age group. These numbers are presented in Table 8.12. Throughout, higher scores are given for males than females in the older dental age groups.

There is considerable overlap in the ranges of the scores in each dental age group. To present a clearer picture the mean score found in each dental age group is plotted for each of the combined % scores in Fig. 8.9. The increase in score with age can be clearly seen, and also the greater increase of males over females discussed above. The mean values, standard deviations and coefficients of variation are listed for each spine measure in Table 31 of Appendix C. The coefficients of variation are plotted in Fig.s 8.10 and 8.11. No particularly consistent pattern is visible between the sexes. Females show higher coefficients in the early dental age group, 20 - 25, for two of the disc scores, lumbar and cervical. The males show a slightly higher variation in thoracic discs. As stated earlier the thoracic disc scores record fused spines from other causes, such as DISH disease (Forestier and Lagier, 1971), which are often predominantly male pathologies. The highest variation is

Table 8.12 Values of Extent (%) Spine by which 70% of the Population is Accounted for in each Dental Age Group (Lumbar and Column).

Lumbar

Dental Age Group	Females	Males
25 - 35	110	94
35 - 45	140	164
45+	166	186

Column

25 - 35	94	80
35 - 45	112	140
45+	134	164

Table 8.13 Values of Extent (%) x Maximum Severity Spine Measures by which 70% of the Population is Accounted for in each Dental Age Group (Lumbar and Column)

Lumbar

Dental Age Group	Females	Males
25 - 35	160	176
35 - 45	256	320
45+	320	384

Column

25 - 35	176	144
35 - 45	272	336
45+	336	464

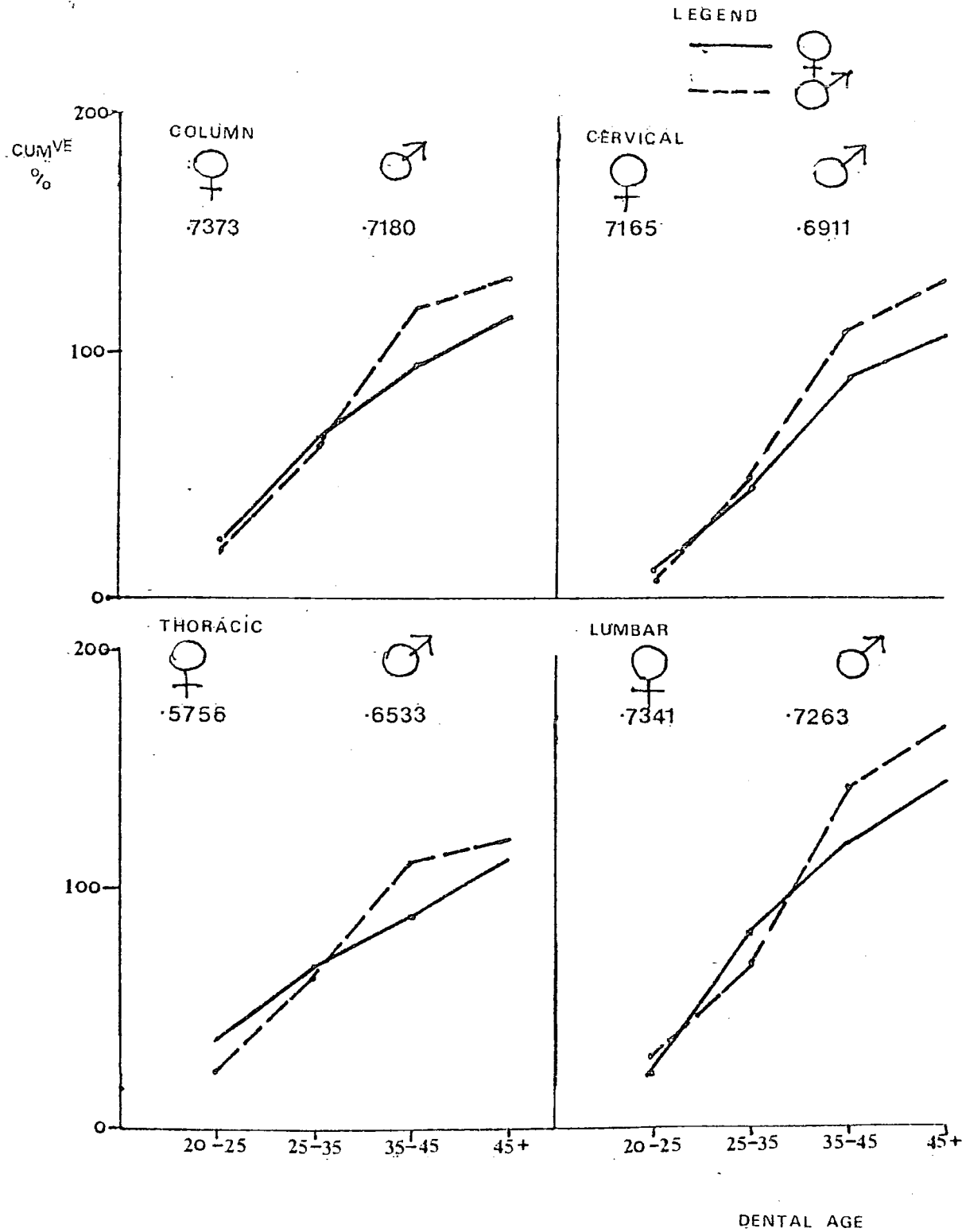


FIG. 8·9 MEAN VALUES OF THE COMBINED EXTENT (%) SCORES WITHIN THE WHOLE SPINE AND BY REGION AND IN EACH DENTAL AGE GROUP.

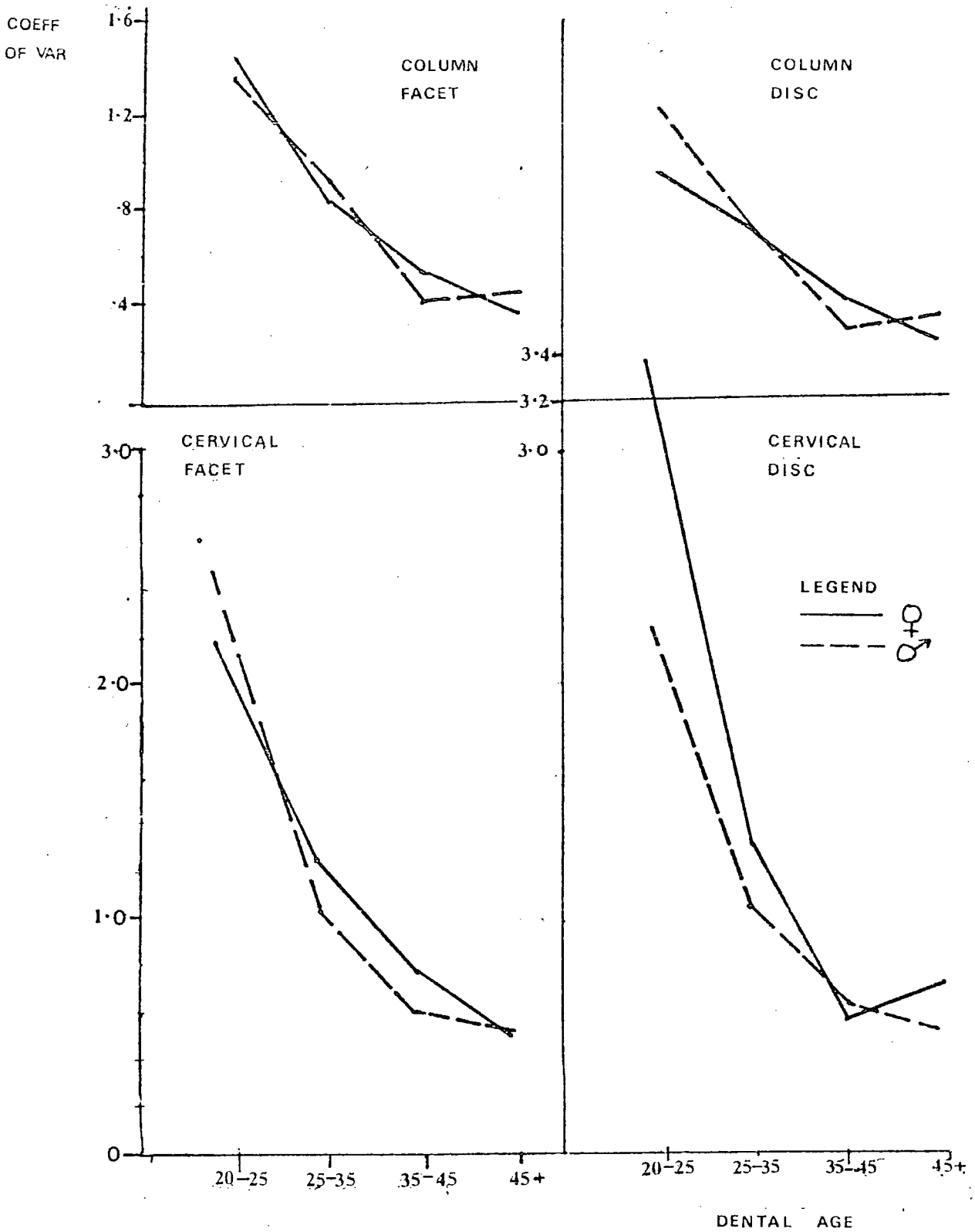
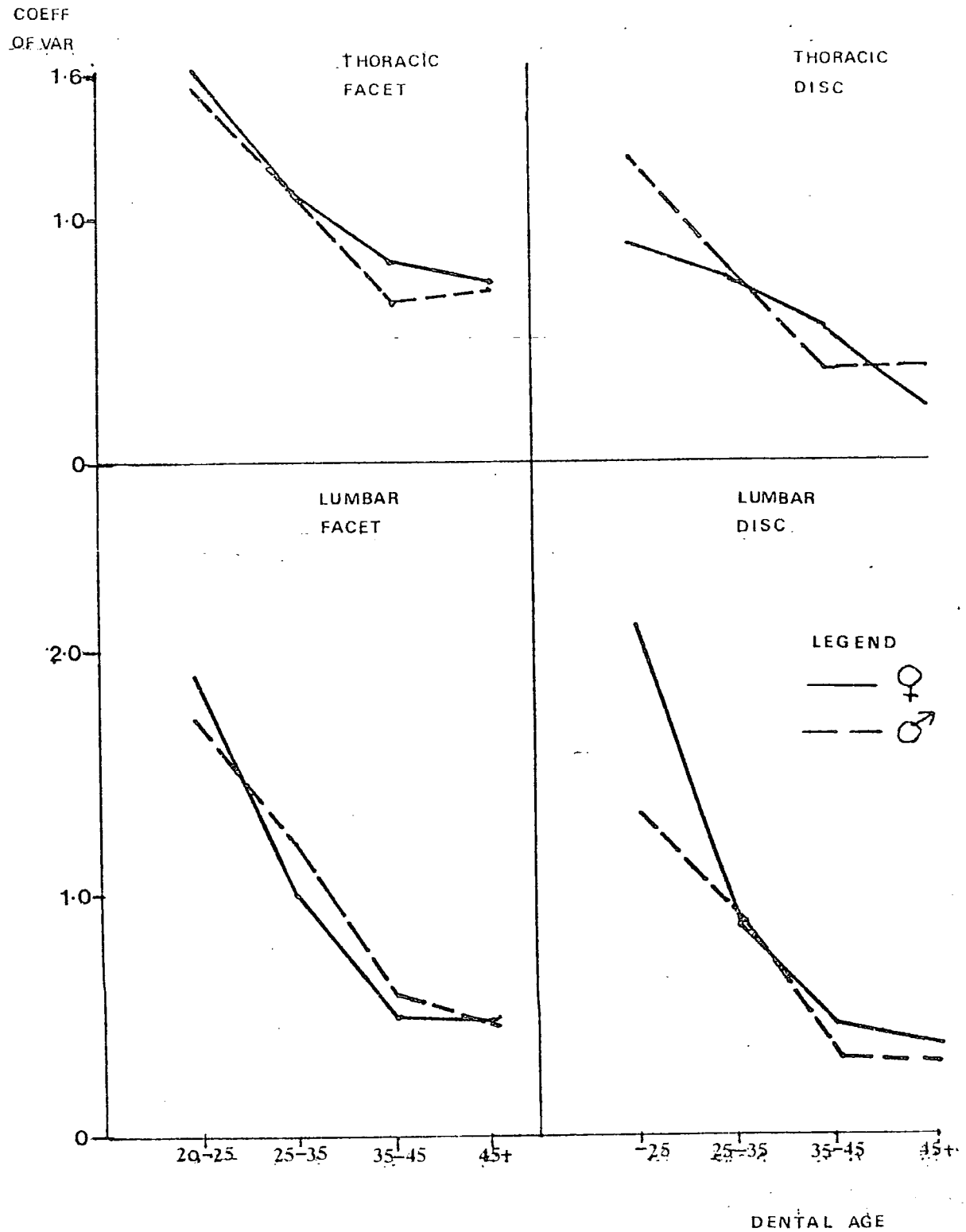


FIG. 8·10 COEFFICIENTS OF VARIATION OF THE FACET AND DISC EXTENT (E) SCORES WITHIN THE WHOLE SPINE AND BY REGION IN EACH DENTAL AGE GROUP.


COEFF
OF VAR
1·6
1·0
0
THORACIC
FACET
THORACIC
DISC
LUMBAR
FACET
LUMBAR
DISC
LEGEND
♀
♂
2·0
1·0
0
20–25
25–35
35–45
45+
–25
25–35
35–45
45+
DENTAL AGE

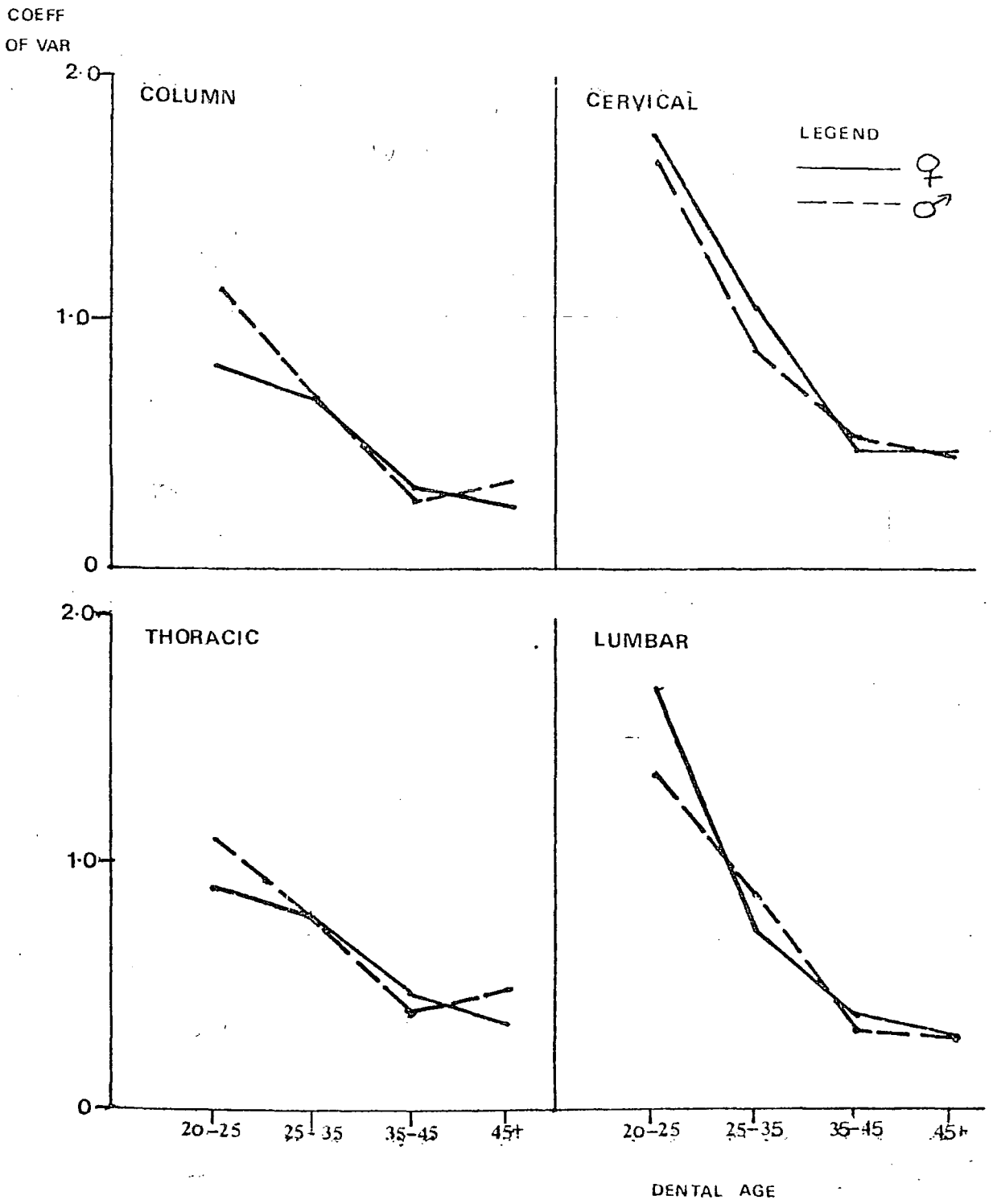


FIG. 8·11 COEFFICIENTS OF VARIATION OF THE COMBINED EXTENT (%) SCORES WITHIN THE WHOLE SPINE AND BY REGION IN EACH DENTAL AGE GROUP.

found in both sexes in the youngest dental age group in the cervical scores for both facets and discs. This is not, however, reflected in the combined score. The coefficients of variation decrease with dental age in all cases, largely as a function of the age-related increase in the mean. Since the mean is increasing with age without a proportional increase in standard deviation, it is therefore justified to claim a decrease in the relative variation about the mean with advancing age.

8.3.5 The Extent (%) x Maximum Severity Measure and Dental Age.

The results of the Mann-Whitney U tests for sex differences in each dental age group are given in Table 32 of Appendix C. Fewer significant values are found than for % scores, but more than for max scores. Only two describe a steadily greater rate in progression of this composite score of males over females. These are cervical facets and the combined cervical score. The cervical discs show a trend but do not quite achieve significance in the final dental age group. All the others show decreases in significance from the 35 - 45 to the 45+ dental age groups. Only the lumbar disc score is still significant by 45+. The column disc and combined scores, the thoracic combined score and the lumbar combined score are no longer significantly different in the 45+ dental age group, documenting a catch up process in females, as described in % scores, by either a continued steady rate while males slow down or an increased rate of progression.

The cumulative % frequencies are plotted for the column, lumbar and cervical combined scores by dental age in Fig.s 8.12, 8.13 and 8.14 respectively. The tables and fig.s for the rest of the series are presented in Table 33 and Fig.s 14 to 22 in Appendix C. In the column 70% of females and males in dental age group 25 - 35 are accounted for by scores of 176 and 144 respectively whereas this only accounts for about 20% of the population in the 45+ dental age group. 70% of the 35 - 45 dental age group are covered by a score of 272 in females and 336 in males. In the oldest dental age group a score of 336 in females and of 464 in males is needed to account for 70% of the individuals. The cervical region combined score distinguishes the dental age groups very well for males, however, since a small proportion never show any degeneration its use as a means of age assessment is reduced, as reflected in the correlation coefficient with dental age that was not as high as might have been predicted from the clarity of the graph. The 70% level spine scores for each dental age group are given for the three graphs in Table 8.13. Again, the higher male values are evident, a combination of course of this feature already described in severity and extent. In the combined column scores essentially all females are accounted for (90%+) by a score of 400 and all by 640 whereas some males show very high scores between 720 - 800. These very high values are probably largely a reflection of some of the thoracic fusions, but the cervical graph indicates some high values there also.

The mean values for the combined scores by dental age

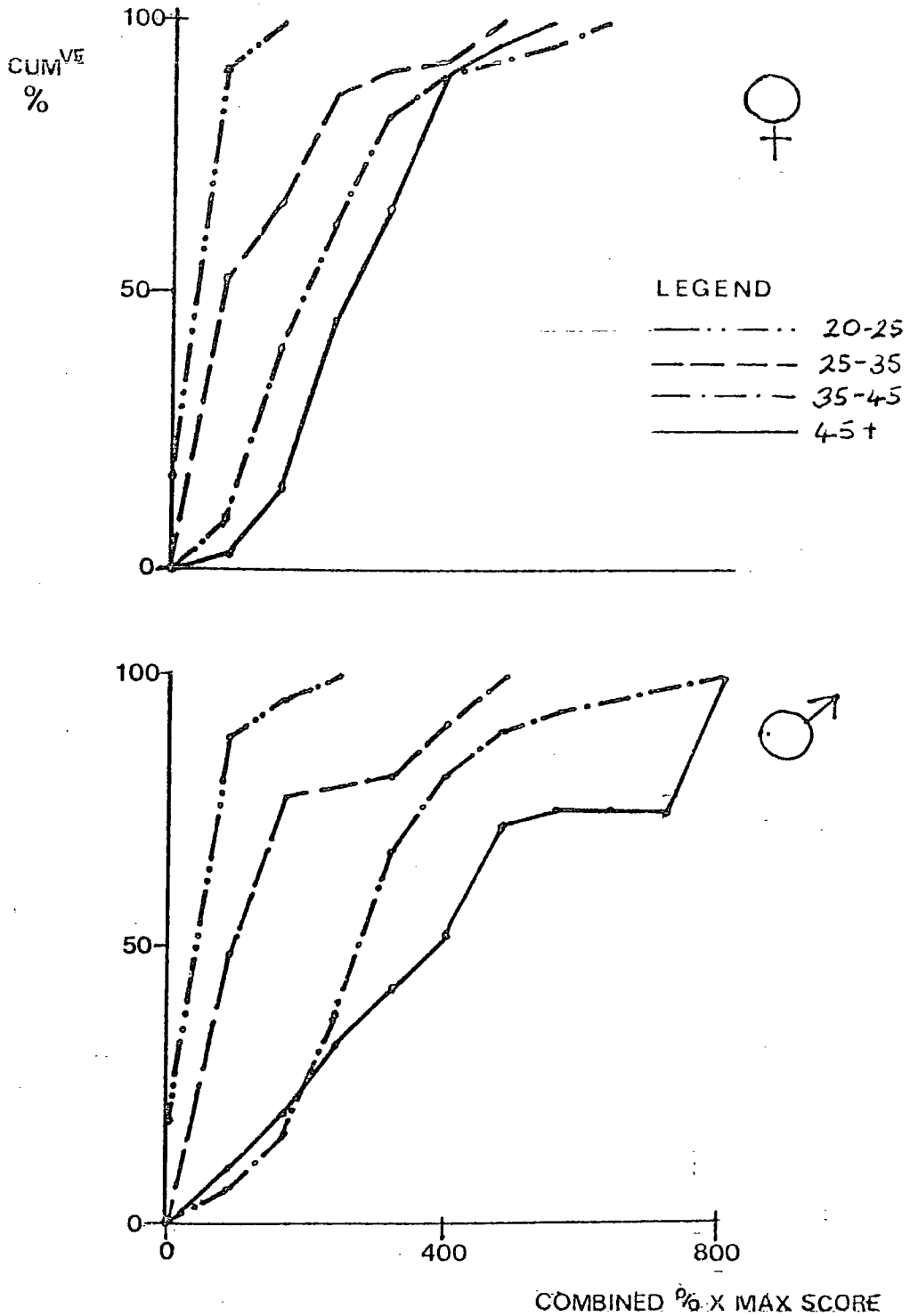


FIG. 8·12 CUMULATIVE % FREQUENCIES OF THE COMBINED EXTENT (%) X MAX. GRADE SCORES WITHIN THE WHOLE COLUMN BY DENTAL AGE GROUP.

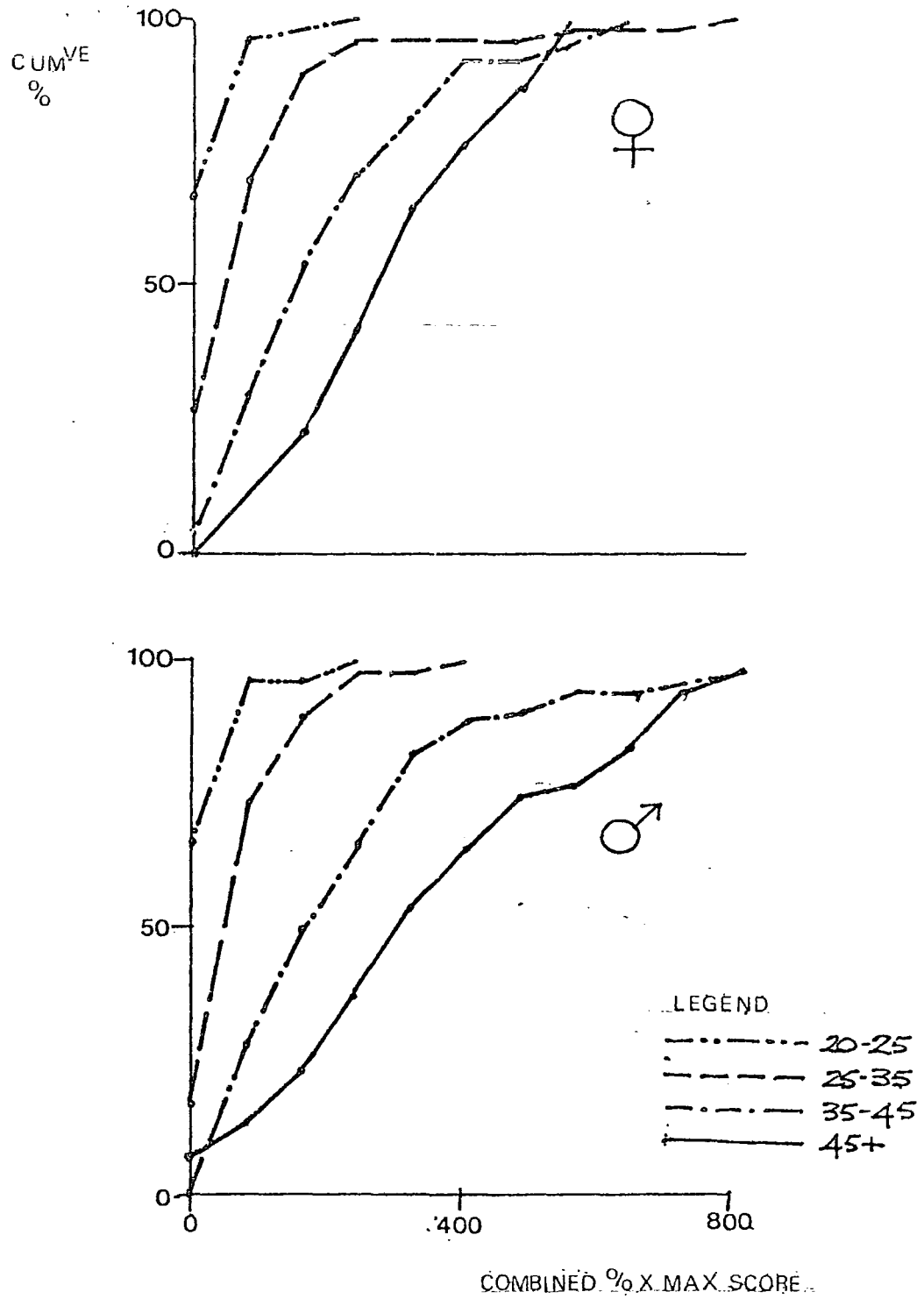


FIG. 8·13 CUMULATIVE % FREQUENCIES OF THE COMBINED EXTENT (%) X MAX. GRADE SCORES WITHIN THE CERVICAL REGION PLOTTED BY DENTAL AGE GROUP.

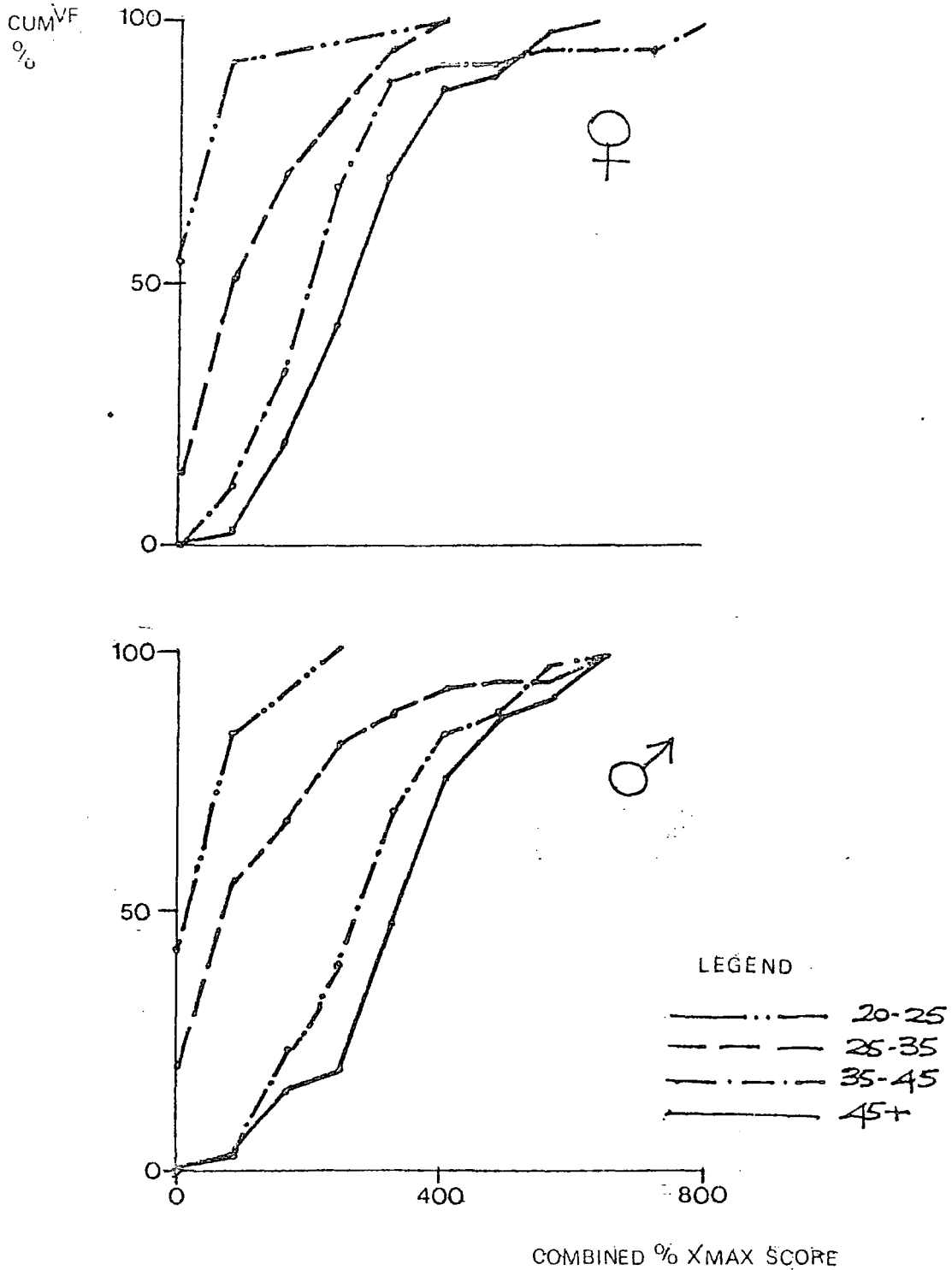


FIG. 8·14 CUMULATIVE % FREQUENCIES OF THE COMBINED EXTENT (%) X MAX. GRADE SCORES WITHIN THE LUMBAR REGION PLOTTED BY DENTAL AGE GROUP.

are illustrated in Fig. 8.15, with the rest in Fig. 23 of Appendix C. Here again is clearly visible the greater scores of males in the older dental age groups. The mean values, standard deviations and coefficients of variation are given for each of the spine scores in Table 34 of Appendix C. The coefficients of variation are plotted by dental age group in Fig.s 8.16 and 8.17. The scores for cervical facet joints in the youngest dental age group of 20 - 25 again show the highest variation. Females also have a high value in the lumbar disc scores in this dental age group. There is a slight tendency for males to have greater variation than females in the oldest dental age group of 45+, and also in the earliest dental age group of 20 - 25 except in the cases mentioned already.

8.4 Summary.

The overall pattern of involvement throughout the spine is similar to that found by other researchers. The two sexes are correlated in this. The facet and disc joints are not correlated with one another in the distribution of involvement. Scores of degeneration of facets and discs in each of the regions do not show particularly high correlations with one another. It appears that the two kinds of joint degenerate independently of one another. The patterns of involvement of the superior and inferior portions of the joint recorded by other researchers are not observed here.

The measures of maximum severity and of extent correlate

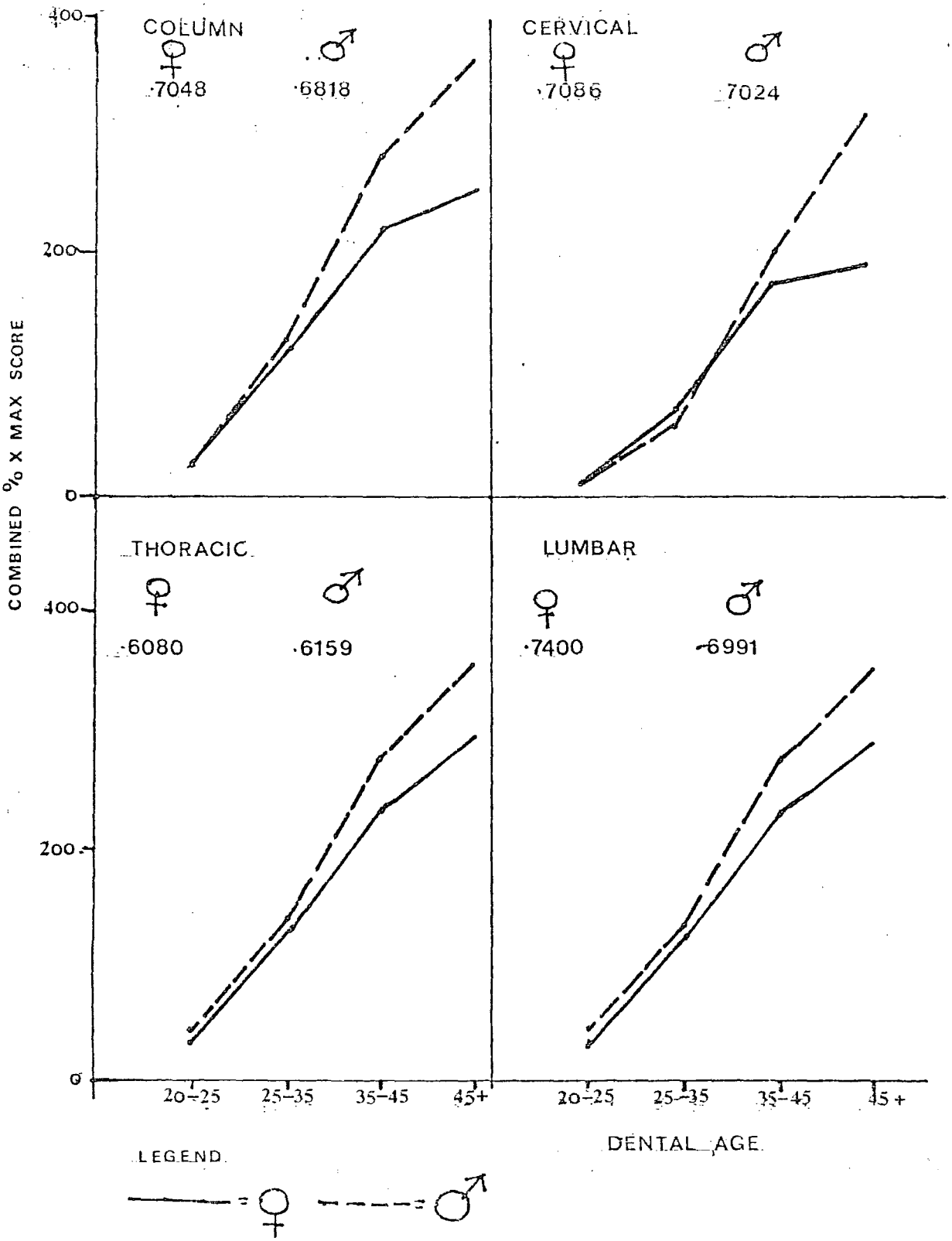


FIG. 8·15 MEAN VALUES OF THE COMBINED EXTENT (%) X MAX. GRADE SCORES WITHIN THE WHOLE COLUMN AND BY REGION IN EACH DENTAL AGE GROUP.

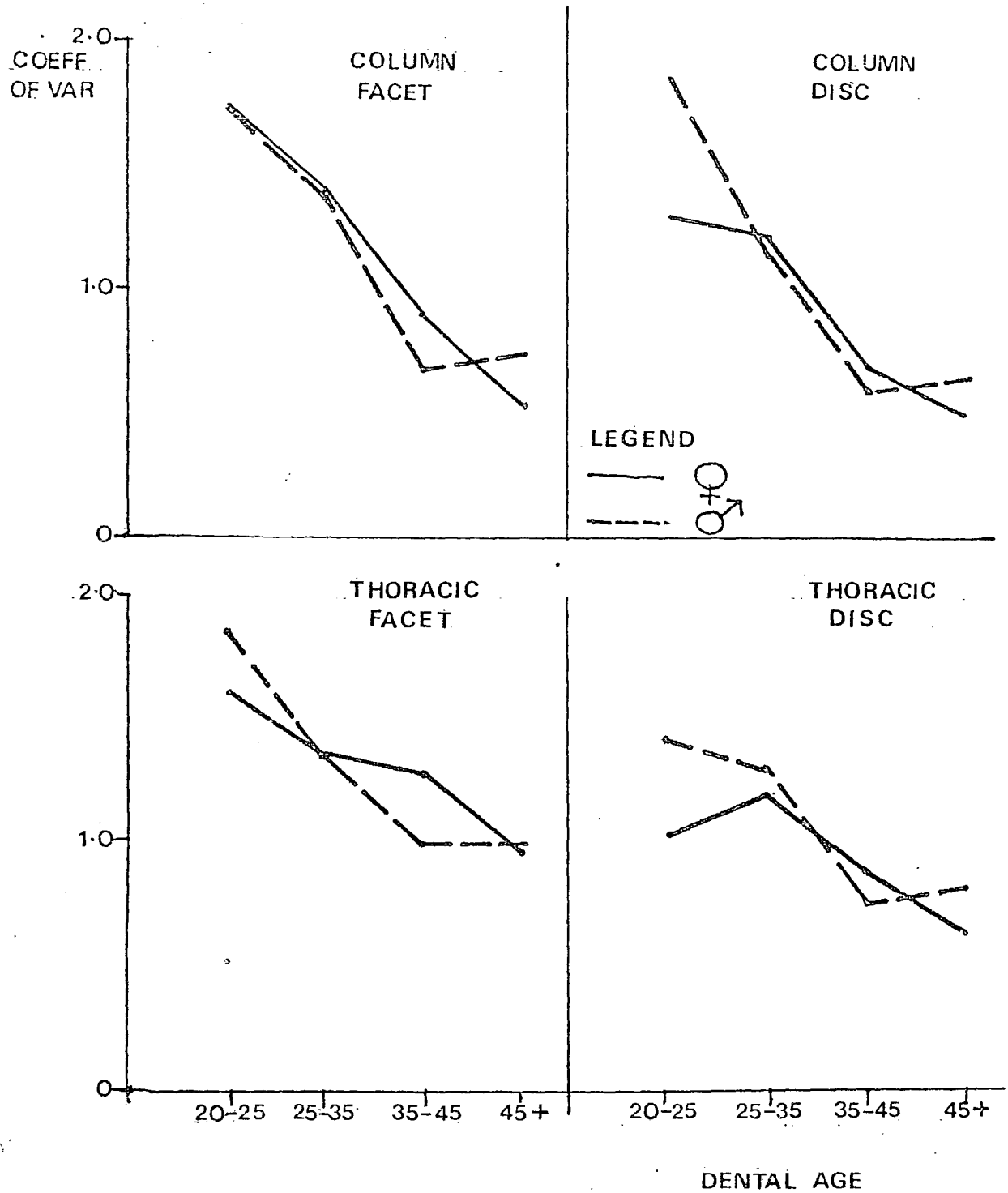
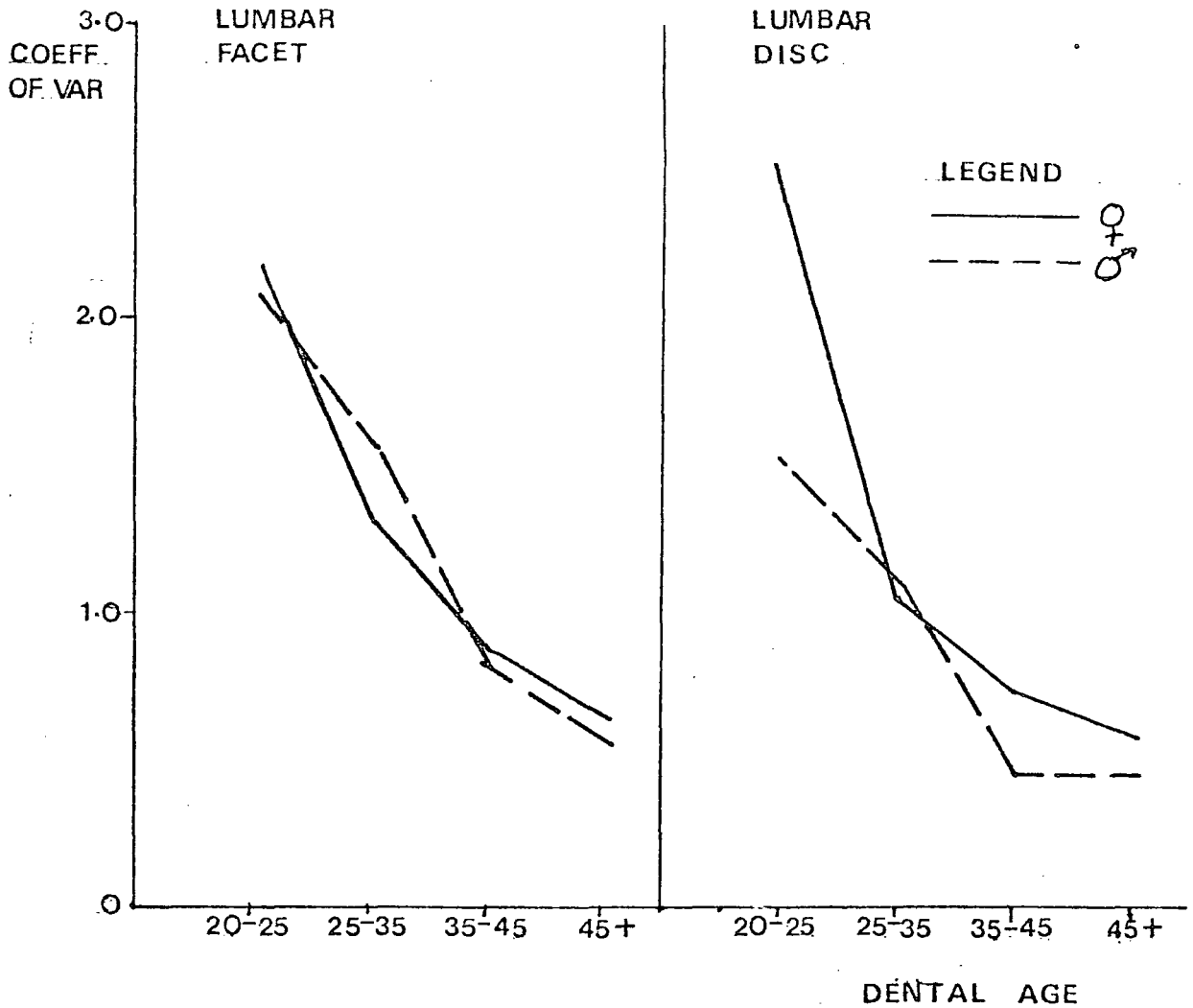
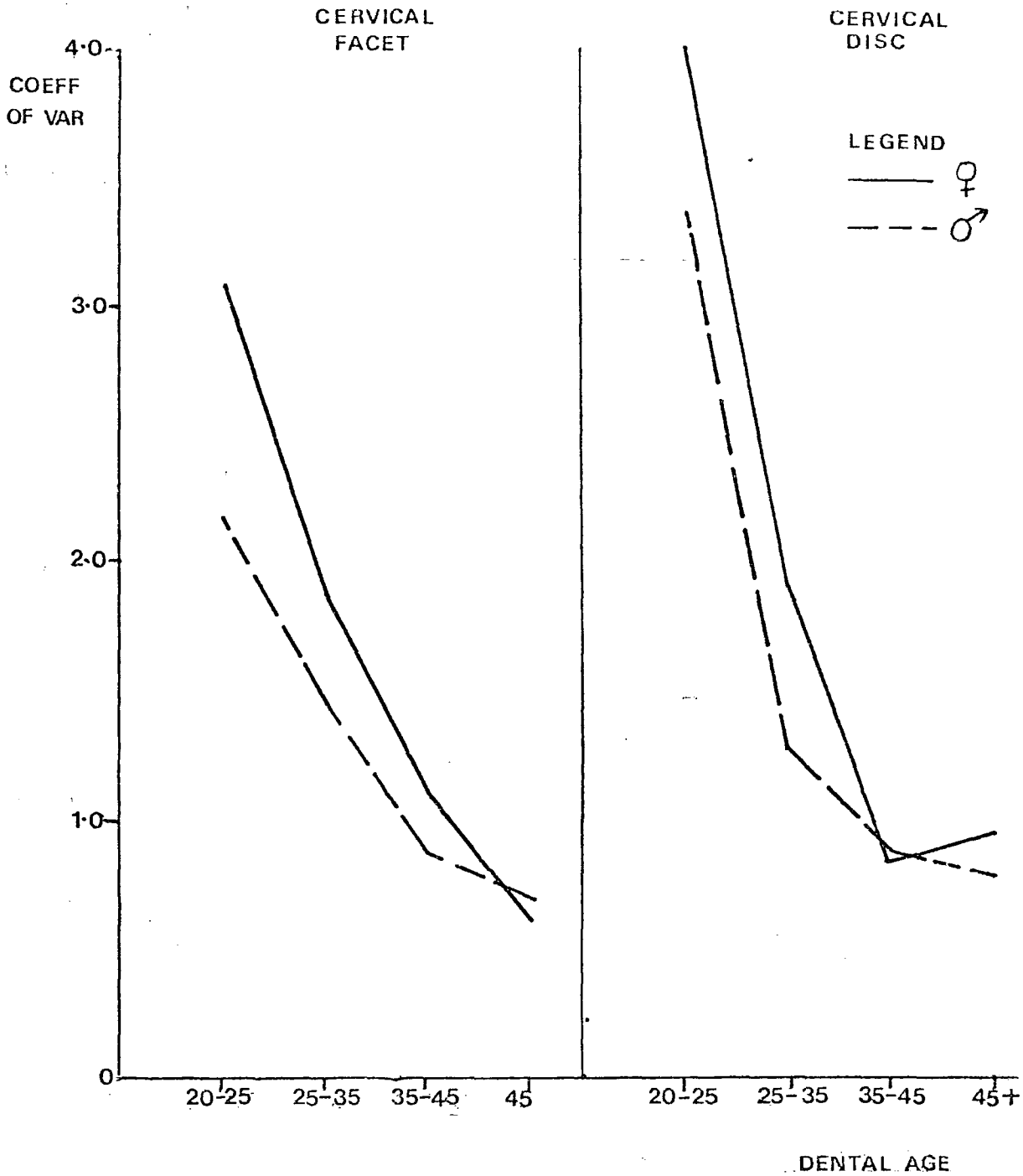


FIG. 8·16 COEFFICIENTS OF VARIATION OF THE FACET AND DISC EXTENT (%) X MAX. GRADE SCORES WITHIN THE WHOLE SPINE AND BY REGION IN EACH DENTAL AGE GROUP.


3·0
COEFF.
OF VAR
2·0
1·0
0
LUMBAR
FACET
LUMBAR
DISC
LEGEND
♀
♂
20-25
25-35
35-45
45+
20-25
25-35
35-45
45+
DENTAL AGE


CERVICAL FACET
CERVICAL DISC
4·0
COEFF OF VAR
3·0
2·0
1·0
0
LEGEND
♀
♂
20-25
25-35
35-45
45
20-25
25-35
35-45
45+
DENTAL AGE

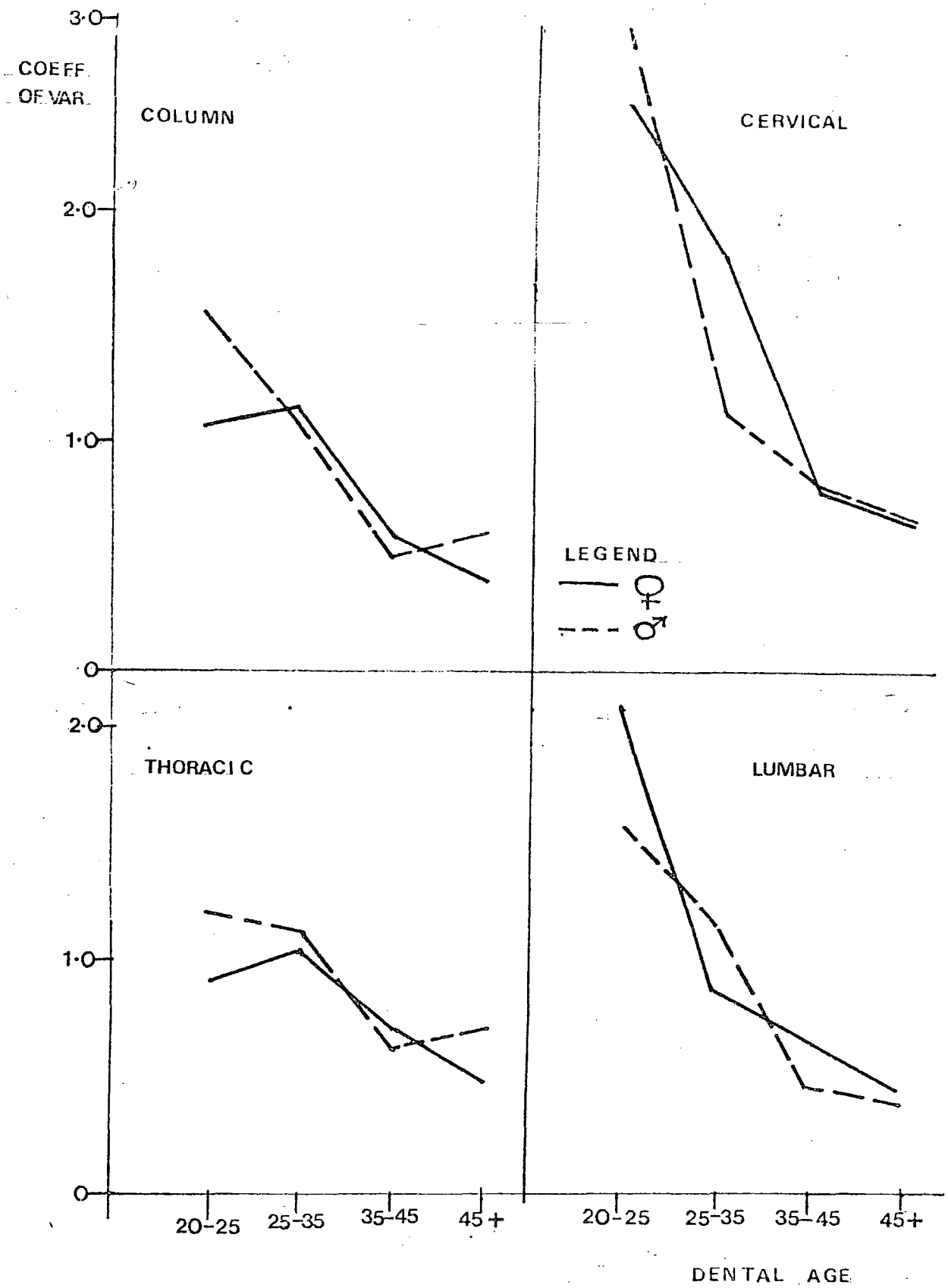


FIG. 8·17 COEFFICIENTS OF VARIATION OF THE COMBINED EXTENT (%) X MAX. GRADE SCORES WITHIN THE WHOLE SPINE AND BY REGION IN EACH DENTAL AGE GROUP.

very closely together, so the addition of the information of severity to the extent score does not improve the correlation to age, as it is redundant. This is convenient, as the measure of extent only demands the identification of presence of degenerative joint disease, rather than using a subjective method of severity assessment. An aging method based on extent therefore, would involve less inter-observer error than the use of severity.

Males show a greater rate in the progression of the extent of the degeneration. This has to be compensated for in making a conversion from the spine score to absolute age.

Degenerative joint disease is found to progress linearly with age in incidence, severity and extent. It therefore provides a potentially useful means of assessing age at death. The best measures for both females and males are the combined extent in the column and in the lumbar region. The lumbar spine is the best preserved region, so this measure is the most favoured for the reason of preservation and because fewer vertebrae are required to make the calculation.

Chapter 9. RESULTS: RELATIONSHIPS BETWEEN ALL MEASURES OF AGE-RELATED CHANGES.

9.1 Bone Structure and Degenerative Joint Disease of the Spine.

The correlation coefficients between all the series of degenerative joint disease measures and the microage estimates and the microparameters are given in Tables 1 to 6 of Appendix D. Those showing a significant correlation are represented in Fig.s 9.1 to 9.6 by the shaded areas.

Looking at the graphical representations of significant correlations, the shaded areas make a pattern for the males with a dominantly horizontal line to it, whereas in the female patterns the eye is drawn to the vertical dimension. This illustrates that males correlate with only a few of the bone structure measures, but across all the spine scores, whereas females have one regional measure in the spine correlating with a number of bone structure measures. KPCL and KAL feature repeatedly as significantly correlated in males. The recurrent high correlating values with the spine in females are TAB, TAA, ADAGE and ADPO.

Since the measures of spinal degenerative joint disease correlated well with dental age such that both increased

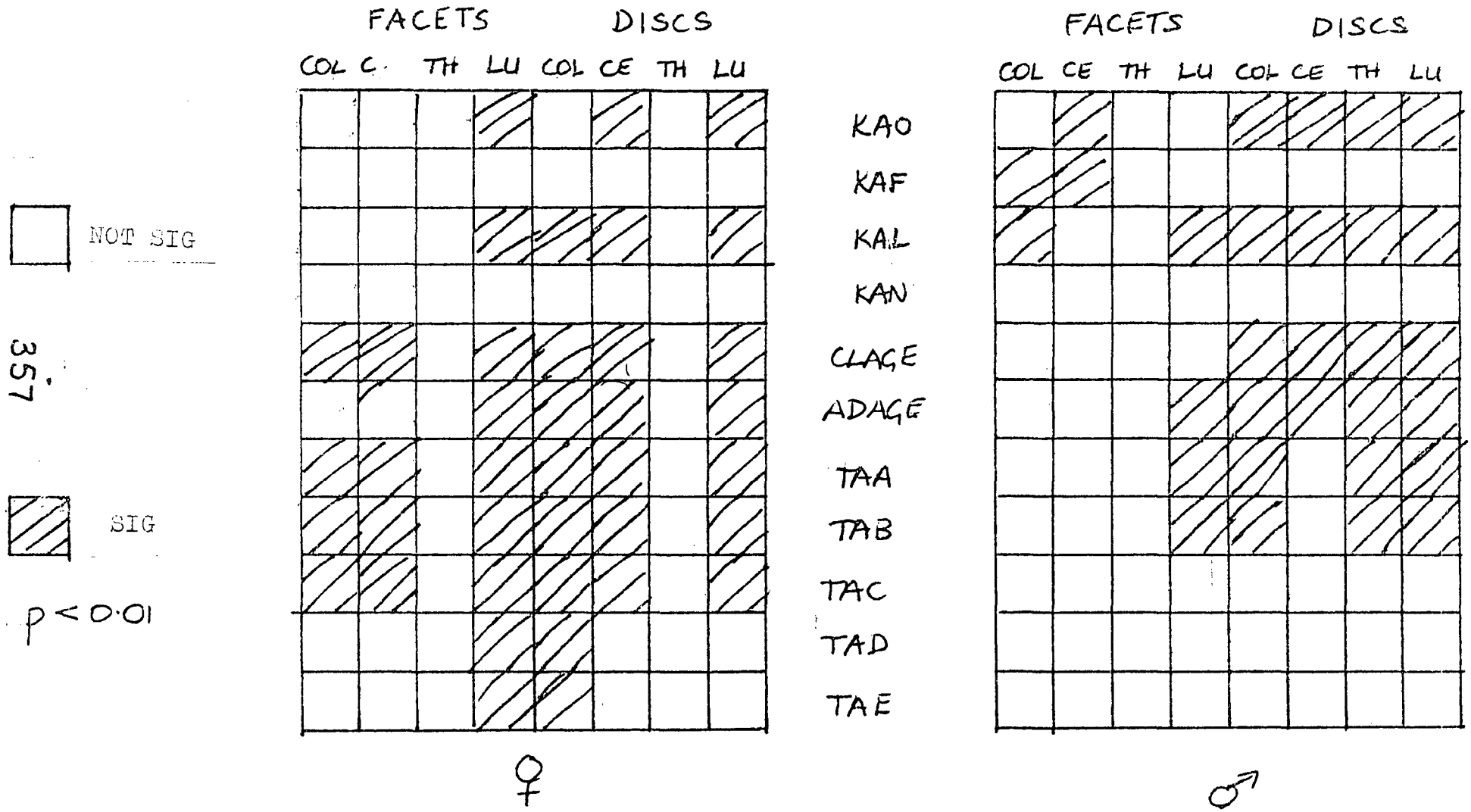


FIG. 9·1 CORRELATIONS BETWEEN THE MICROAGES AND THE SPINAL MAXIMUM GRADE SCORES.

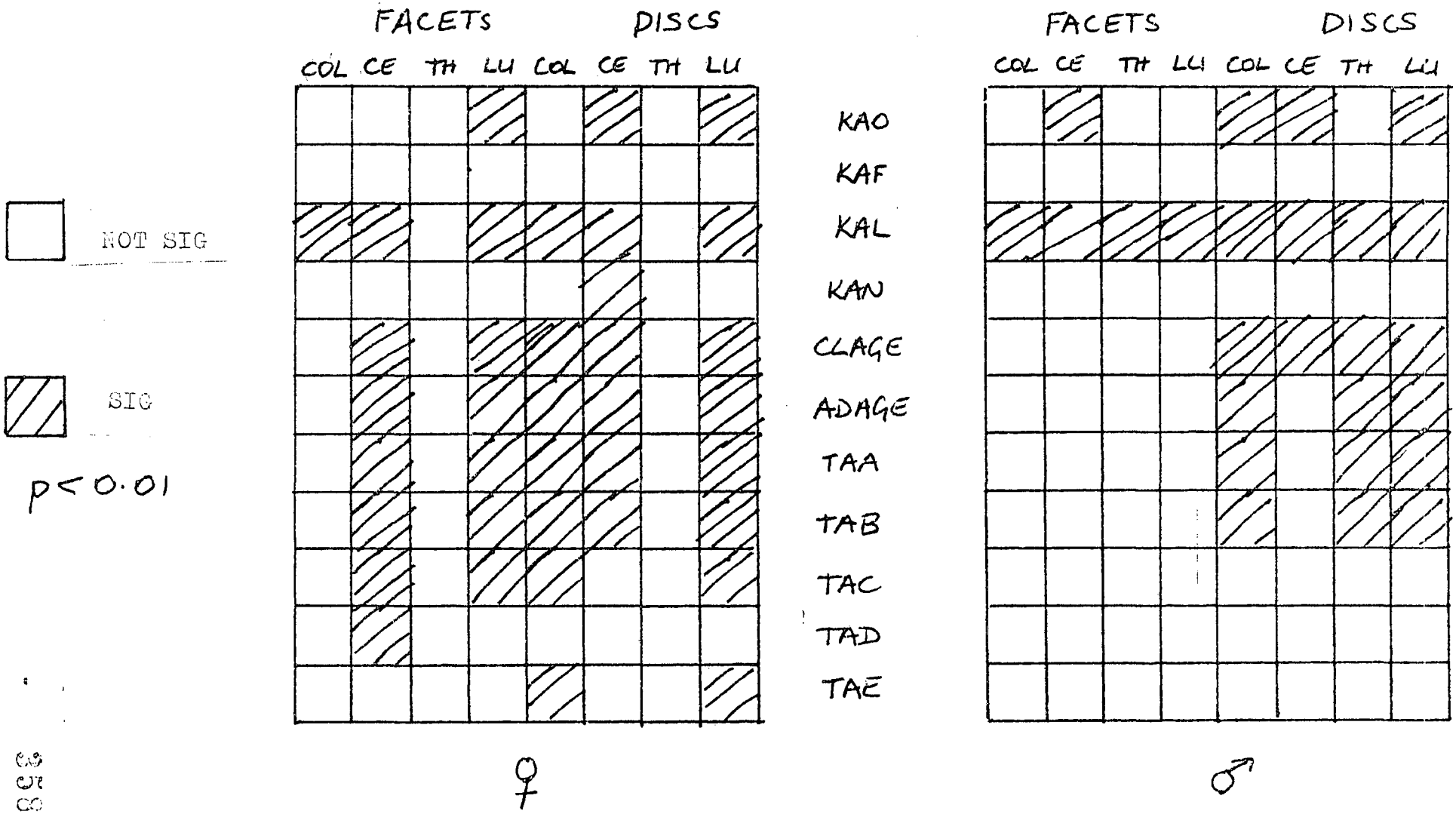


FIG. 9·2 CORRELATIONS BETWEEN THE MICROAGES AND THE SPINAL EXTENT (%) SCORES.

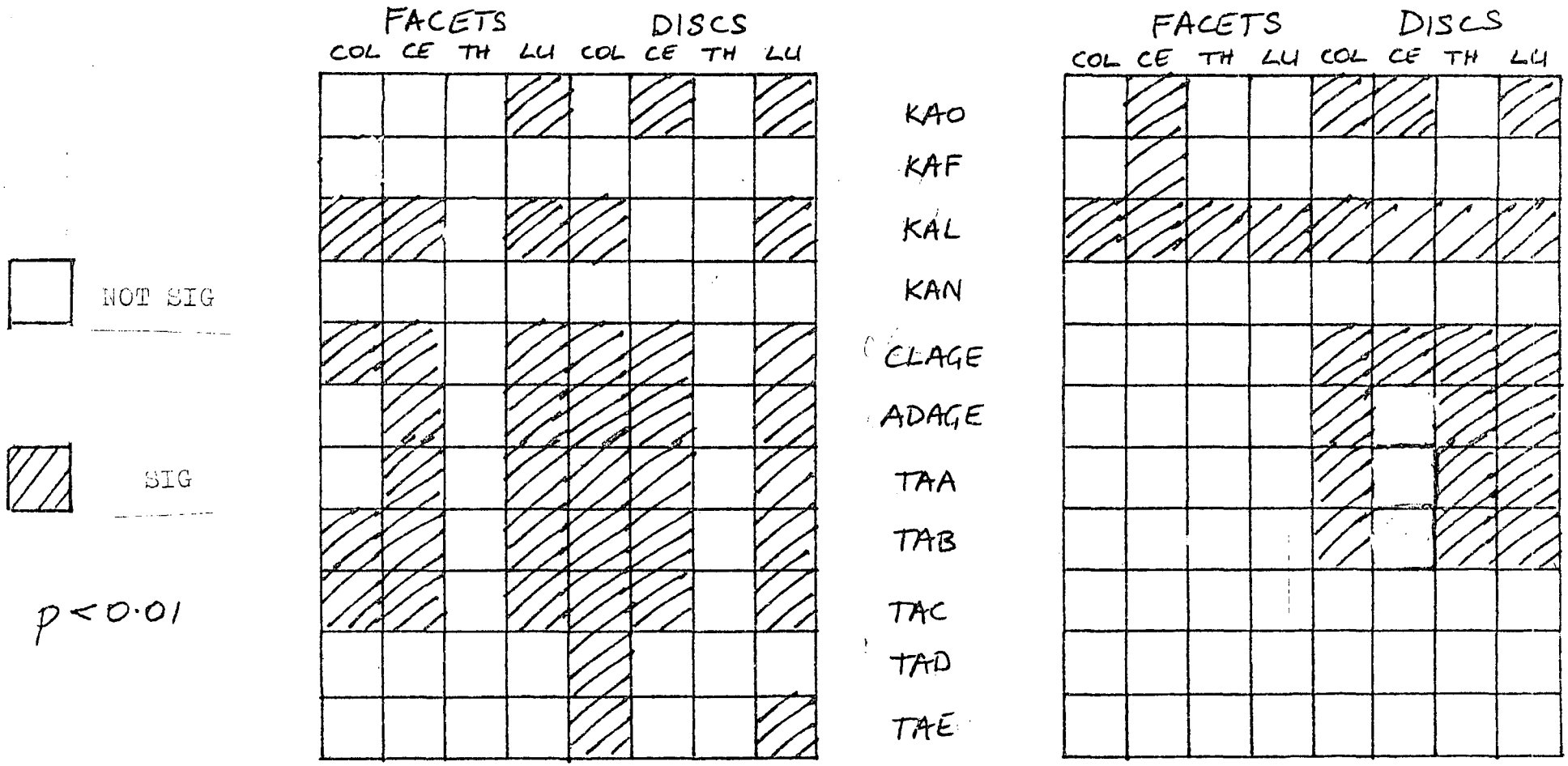


FIG. 9·3 CORRELATIONS BETWEEN THE MICROAGES AND THE SPINAL EXTENT (%) X MAX. GRADE SCORES.

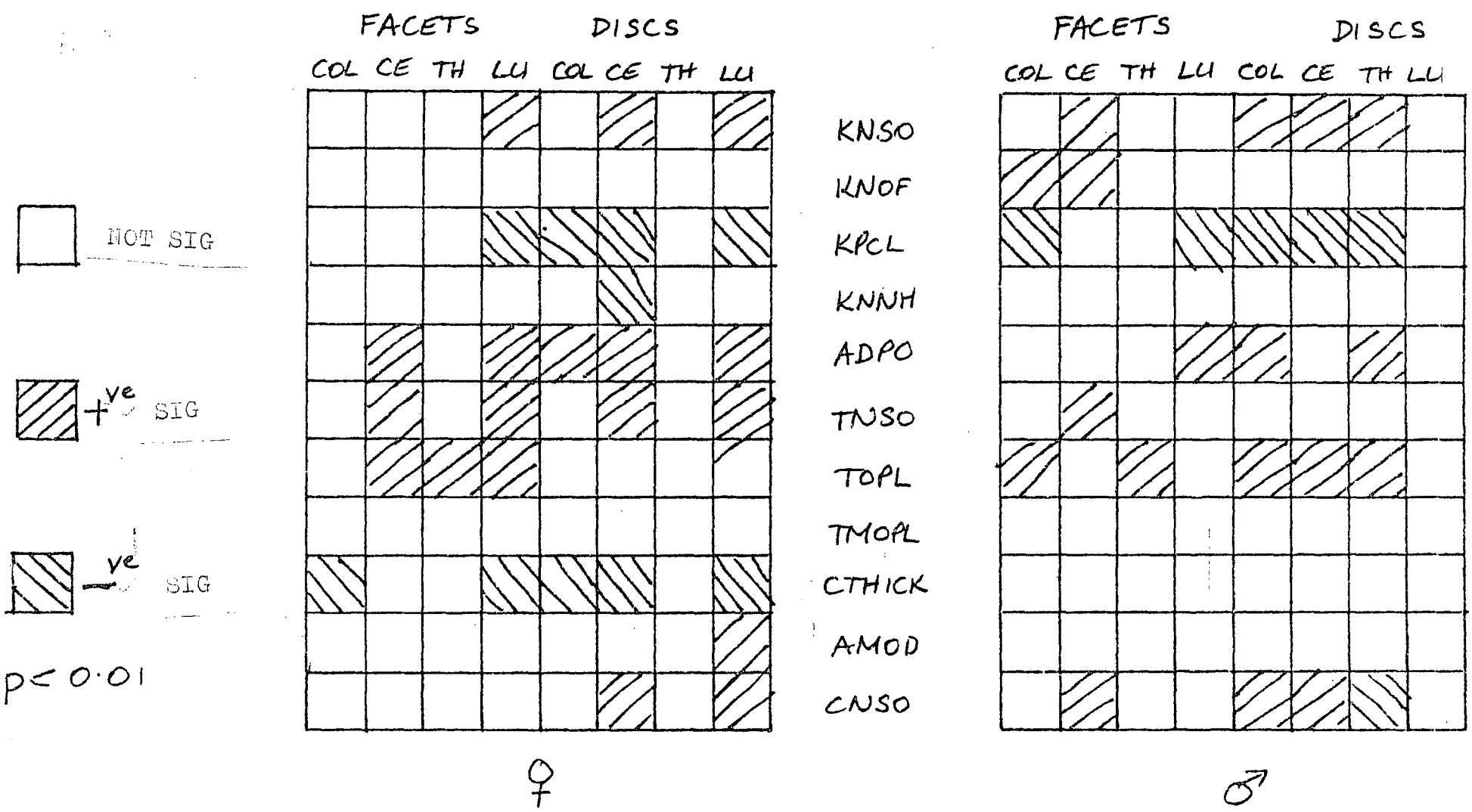


FIG. 9·4 CORRELATIONS BETWEEN THE MICROPARAMETERS AND THE SPINAL MAXIMUM GRADE SCORES.

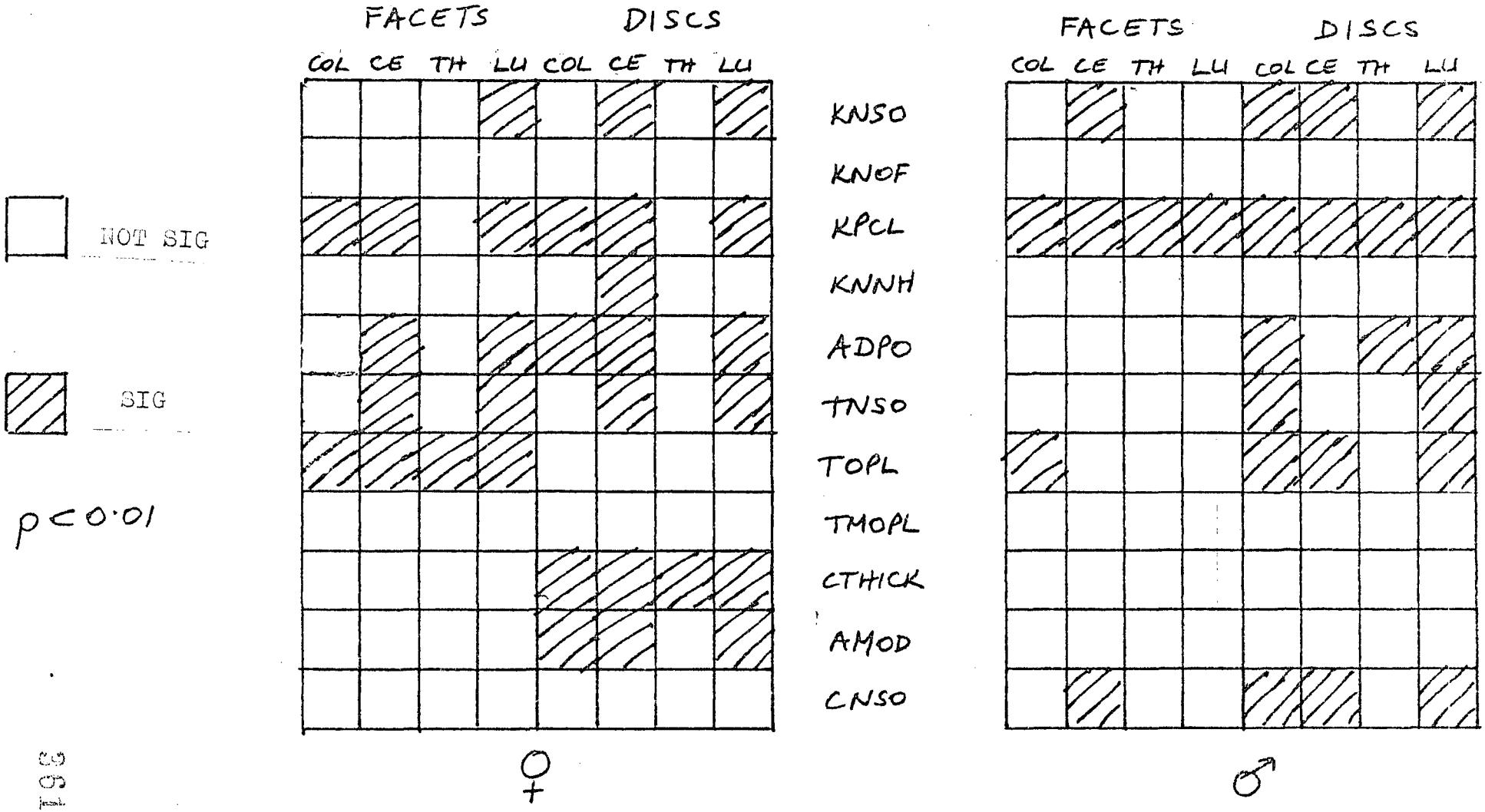


FIG. 9·5 CORRELATIONS BETWEEN THE MICROPARAMETERS AND THE SPINAL EXTENT (%) SCORES.

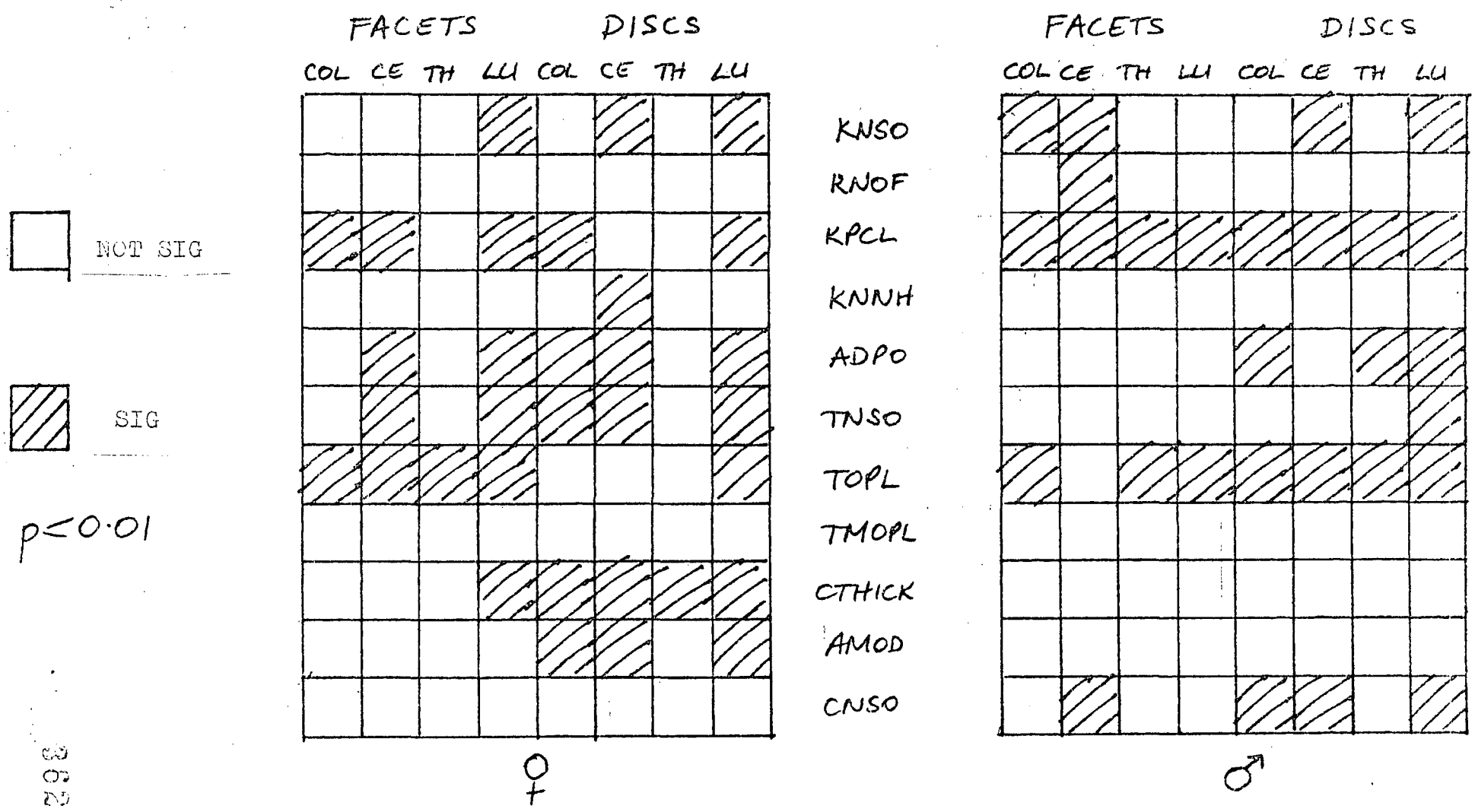


FIG. 9·6 CORRELATIONS BETWEEN THE MICROPARAMETERS AND THE SPINAL EXTENT (%) X MAX. GRADE SCORES.

linearly together, the bone structure measures would be expected to relate to the spine scores in the same way as they were seen to relate to dental age. The combined extent (J) measure for the whole spine have been classed into five groups of 0 to 4, 0 being a score of 0, the other four representing successive ranges of scores of 50, up to the maximum score of 200. The mean values for the microparameters in each spine measure category are given in Table 7 of Appendix D.

The internal parameters display the same pattern of change in relation to the spine measure catogories as to dental age, that is an increase in value followed by a decrease. The external parameter of cortical thickness follows this pattern in males, but in females shows an increase in the mean value in the last spine measure category. This is evidence that extreme cases of bone loss and degenerative joint disease are not found together. The overall loss in females is 12%, the same as found against dental age. The male loss is slightly less than by dental age, only 4%, but of much the same order. The internal parameters do not show this reversal trend against the final spine measure category since the extreme cases of bone resorption were excluded from internal examination through the practical difficulty of producing a thin section.

9.2 Factor Analysis.

To assimilate all the possible relationships between

all the measures of age-related changes, a general summary is produced by performing a factor analysis on the following measures:

dental attrition (M_1 M_2 M_3),

pubic symphysis metamorphosis (PS),

the extent (%) spine measures,

the extent (%) x maximum severity spine measures, and

the microparameters.

Dental attrition and pubic symphysis metamorphosis are treated as continuous variables for this purpose, which they are not, strictly speaking. The maximum severity spine measures, dental age and pubic symphysis age have been left out as they very obviously use ordinal rather than interval scales of measurement.

The resultant groupings of the variables by rotated factors are given for each sex in Tables 9.1 and 9.2. The full tables of the correlation coefficients of the variables with each of the seven factors are given in Tables 8 and 9 for females and males respectively in Appendix D. The group of microparameters are isolated from all the other measures in both sexes, reflecting their non-linear behaviour. Both sexes have one main core group of microparameters, which in females also includes KNOF. The scores of attrition and pubic symphysis metamorphosis are grouped together in males, but are in a separate category from any of the other scores. In females, the pubic symphysis score is separated from the attrition scores, not highly correlated with any factor but preferentially grouped with the first factor which contains

Table 9.1 Dental Attrition, Pubic Symphysis Metamorphosis, Bone Structure and Spine Scores grouped by Rotated factor analysis in Females.

Factor 1	Factor 2
Thoracic disc %	CNSO
Column disc %	TNSO
Thoracic disc % x max	KPCL
Column disc % x max	KNSO
Thoracic combined %	ADPO
Thoracic combined % x max	TOPL
Column combined %	KNOF
Lumbar disc %	KNNH
Cervical disc %	
Lumbar disc % x max	
Pubic Symphysis	

Factor 3	Factor 4
Cervical combined % x max	Lumbar combined %
Cervical facets % x max	Lumbar facets % x max
Cervical disc % x max	Lumbar combined % x max
Cervical combined %	Lumbar facets %
Cervical facets %	M_3
Column facets % x max	M_2
Column combined % x max	M_1

Factor 5	Factor 6
Thoracic facets %	TMOPL
Thoracic facets % x max	CTHICK
Column facets %	

Factor 7

AMOD

Table 9.2 Dental Attrition, Pubic Symphysis Metamorphosis, Bone Structure and Spine Scores grouped by Rotated Factor Analysis in Males.

Factor 1

Column disc % x max
Cervical disc % x max
Cervical combined % x max
Cervical disc %
Column disc %
Cervical combined %
Column combined % x max
Thoracic disc % x max
Thoracic disc %
Column combined %
Thoracic combined % x max
Cervical facets % x max
Lumbar disc % x max
Thoracic combined %
Lumbar disc %
Cervical facets %

Factor 2

Thoracic facets %
Lumbar facets % x max
Column facets %
Thoracic facets % x max
Column facets % x max
Lumbar facets %
Lumbar combined % x max
Lumbar combined %

Factor 3

TNSO
KNSO
ADPO
KPCL
TOPL
CNSO
KNNH

Factor 4

M_3
M_2
Pubic Symphysis
M_1

Factor 5

KNOF

Factor 6

AMOD
CTHICK

Factor 7

TMOPL

spine disc scores. The female attrition scores are grouped with a number of lumbar scores, reflecting the better correlation between dental age and some of the spine measures in females compared to males.

The first factor in both sexes involves a group of disc and combined scores. In females these are thoracic and lumbar scores, whereas in males all the disc scores and all but two of the combined scores are in this first group. The male facet scores and the two lumbar combined scores are grouped together by factor 2. By contrast in females, the facet scores and the remaining disc and combined scores are spread over three other factor groups, separated by region.

The factor analysis therefore restates in a brief, manageable form some of the relationships and differences already found from the bivariate analyses. Males show a greater integrity of the spine degeneration than females. Facets and discs appear to degenerate independently of one another. A number of spine scores are more closely related to the dental attrition scores in females than in males. The pubic symphysis score in females does not appear to be reliable. The microparameters follow a pattern of age-related change that is quite different from the other measures.

Chapter 10. DISCUSSION.

The results throw up a whole host of issues for discussion, but since the main interest here is to evaluate the assessment of age at death from skeletal material, the discussion will be directed towards this problem. First the age-related changes observed in Poundbury in the bone structure and in the progression of degenerative joint disease will be considered followed by a discussion of mortality patterns in Poundbury and other archaeological samples.

10.1 Age-related Changes in Bone Structure.

The Poundbury cortical sections were all examined from the periosteal border, a fact to be remembered in discussing the processes of internal remodelling described by the parameters studied. Internal remodelling shows a recurrent pattern in a number of the parameters, which depicts a change in the process at the 35 - 45 age group. The specific measures involved in this trend are the number of secondary osteons, the number of osteon fragments, the total osteon perimeter length, the % area of osteon bone and the percentage area of circumferential lamellar bone. These parameters show a steady increase with age up to 35 - 45, whereafter a decrease is found, except for percentage area osteon bone and total perimeter length in females which

continues to increase. The measure of the percentage area of circumferential lamellar bone follows the opposite pattern of decreasing, then increasing from 35 - 45.

The increase in osteon number, osteon fragment number, total perimeter length and percentage area of osteon bone all suggest a steady process of remodelling of the bone already laid down. The subsequent decrease in these measures at 35 - 45 onwards implies that new bone is being formed at the periosteal surface in the characteristic fashion of parallel-layered lamellae before being remodelled into Haversian systems. This would account for the lowered numbers of osteons per unit of area, of osteon fragments, of percentage area osteon and therefore the decrease in total perimeter length. It also explains the opposite measure of percentage area of circumferential lamellar bone which increases at this age.

The literature review of studies on bone dynamics presented in chapter 2 gives evidence to support such a hypothesis. A steady and low level of bone turnover is maintained throughout young adulthood, up to 35 - 45. This would produce a steady age-related increase of osteon number, fragment number, percentage area of osteon bone and total perimeter length, plus the associated decrease in percentage area of circumferential lamellar bone. At 45 - 50, an increase is observed in activity of both resorption and formation. This was studied at the internal level of bone cell dynamics (see Table 2.1) indicating an acceleration of bone turnover. The studies of external remodelling demonstrate that bone

is laid down periosteally at a steady rate throughout life after adolescence. There is no evidence of an acceleration of periosteal apposition of new bone at this 40 - 45 year period. However, this has only been studied in terms of diameter measures, not by analysis of the type of bone found at the periosteal surface. The results here suggest that the increase of internal turnover of both resorption and formation is not simply a remodelling of the extant Haversian systems, but a true increase in the formation of new bone in the childhood model of parallel-layered lamellae. This is seen to occur at the periosteal surface, the rest of the section not having been examined. The onset of the increased bone turnover appears earlier than in a modern sample.

The males of the population show a greater decrease in all the parameter measures after the 35 - 45 age group than females. Females, in fact, do not show a decrease at all in two of the parameters, those of percentage area osteon bone and total perimeter length. Although in older age groups, the rate of resorption increases dramatically over formation this mainly occurs endosteally, so would not affect the periosteal observations. The rate of formation, by contrast, remains constant after the initial increase. No sex differences have been recorded by other researchers, so there is no reason to expect a greater rate of formation in males, although this is not impossible. The greater decrease in the observed male values seems more likely to reflect a higher mean age in the 45+ age group amongst males compared to females.

The external process of remodelling displays an increase in cortical thickness up to the 25 - 35 age group, whereafter a decrease occurs in both sexes of 12% in females and 6% in males. The age of onset of cortical thickness loss is earlier than found in modern samples but comparable to that found in archaeological populations. The age-related loss would therefore support a model of nutrition mediated hormonal control over resorption rather than a post-menopausal action. However, if the sample is incorrectly aged the evidence from the archaeological populations is misleading. Instead, it is proposed to use the start of cortical loss to set the menopausal age. This indicates that the dental age categories are underaging the sample. This would be supported by the evidence from the internal parameters, which also appear to display the characteristic increase in turnover rate at an earlier age than a modern sample.

The percentage of loss of cortical thickness is lower in Poundbury than in a modern sample, and also lower than in other archaeological samples. This implies the mean age of death is lower in Poundbury than in a modern sample, since the greatest loss of cortical bone occurs during the fifties. A large sex difference is seen in the amount of bone lost. The maximum sex difference in the rate of bone loss is found in the forties and early fifties in modern samples. This, again, implies only a few individuals are surviving to sixty and beyond.

It is, of course, quite possible that the archaeological

samples display a quite different pattern of age-related loss, beginning earlier in life and progressing at a much slower rate. However, the modern pattern has been recorded over a great range of samples from very different environments and nutritional levels and found to hold good. Therefore, the age-related changes found in the modern populations can be drawn upon to set age levels in the archaeological sample. It is found that using the parameters in this way is more useful for age assessment than the current methods, which assume a single direction of age-related changes, an assumption that is not justified.

Although both the internal and external measures show a change of remodelling activity, the two do not appear to be interdependent, and are not considered to be so by other researchers. The actual age at which the alteration of remodelling activity is different in the two levels of observation, and a sex difference observed at the external level is not found at the internal level. In the internal remodelling process the increased turnover is in the late thirties and early forties. No corresponding change is seen at the external level. The escalation of resorptive activity seen internally occurs mainly at the endosteal margin and does appear to be related to the external observations, if not in fact representing the same process for the most part. Endosteal resorption is not reflected in the internal measures taken here, only in the external measure of cortical thickness. The internal measures therefore show very little correlation with the cortical thickness measure for either sex. Some

support is given here to the concept of separate processes of internal and external remodelling. The changes of increased resorption of later years may be more closely related between the two levels than earlier activities.

Measures of body size tend to show a smaller range of variation about the mean in females compared to males. This is interpreted as the result of genetic action operating more strongly in females to control size closely around an optimum whereas males are less strictly controlled and respond more to environmental agents of nutrition and climate. This is traditionally viewed as an evolutionary response to the different roles of the two sexes in reproduction. The survival of the mother is paramount for the survival of the offspring so a response to the environmental vagaries of food availability is not appropriate. Males, on the contrary, reproduce maximally through successful competition with other males, therefore size becomes an important factor even if at the expense of long-term survival. This argument has been extended to measures of cortical thickness which show a marked sexual dimorphism, with males having far thicker cortices at all ages.

The Poundbury population does not fit with the view of greater male size variation. The coefficients of variation of cortical thickness are higher in females from 25 - 35 onwards than in males. There are three possible interpretations of this surprising result. First, it could be that the theory is wrong, that females do not experience a greater genetic

control over cortical thickness and that if anything, the opposite is true. Since the evidence for an X-linked genetic control comes from studies where full information on age, sex and familial relationships is available, it is not likely that their results are irregular compared to Poundbury, where only a very low level of such information is present. It seems equally unlikely that the Poundbury population should represent an unusual exception to the rule. The second possibility is that the method of dental age assessment is less accurate for females than males and that a large proportion of females have been categorized into the wrong group. If such a jumble in the female age groups were to be the case, the expected age-related patterns of change in either bone structure or spinal joint degeneration would not be found. Since this is not the case, there is no reason to suppose a difference between the sexes in the applicability of the method of dental age assessment. Lastly, the Poundbury population may contain a more heterogeneous mix of females in terms of gentic origin than males. Again, there is no particular reason for this to be so. However, since details of the population background are the unknown variables of this sample, this line of argument seems the most plausible at present.

Measures of internal remodelling do show a greater coefficient of variation in males, mainly in the last age group. Rather than postulate a greater variation in the rate of age changes in males, it seems more likely to reflect a greater range of ages of males in the last age group, as

discussed above.

The one sex difference in variation that is not explicable in other terms is the tendency for females to show a greater correlation between the various parameters. However, this is only a slight tendency mainly concerning the correlation between osteon fragments and the other measures. It may be therefore, that variation between different parameters is greater in males than females. This is important only in using the methods of age assessment available where combinations of parameters could give a slightly different pattern in relation to age for the two sexes. Since the current methods are not found to be useful this is only a minor consideration.

10.2 Age-related Changes of Degenerative Joint Disease in the Spine.

Discussions of distributions, severity, extent and age relationships of degenerative joint disease of the spine usually draw upon biomechanical interpretations for particular population specific results. This is difficult to perform on the Poundbury sample as little is known about lifestyle and daily activities. Instead, the observed patterns of degenerative joint disease might give support to speculations of life in a Romano-British town. The cemetery is assumed to have served the town of Dorchester, which is thought to have been a market town. There are, therefore, few individuals in the sample who would have been involved

in heavy agricultural labour. The majority of the population were probably engaged in trade, service or administration, none of which make great mechanical demands on the joints. Certainly some individuals would have been engaged in the heavy work of building etc., but it is suggested these would represent a minority. The standard of living was probably very varied, but socioeconomic status would not affect the occurrence of degenerative joint disease in itself, only indirectly in relation to the extent of physical labour. If this is so, the incidence and severity of degenerative joint disease in Poundbury may be expected to show similar patterns to modern samples.

The distribution of incidence throughout the spine shows the usual pattern of three outcrops in both facet and disc joints. The outcrops are largely comparable to the modern samples rather than the anomalous archaeological sample from Aebelholt in Denmark. The lumbar peak covers the region of L3 to L5 lending no support to the hypothesis that the peak was higher in archaeological times and lower in modern times (Lindblom, 1951).

The proportion of the Poundbury sample showing some degeneration peaks at the 35 - 45 age group for both sexes in both facet and disc joints. The modern study of osteophytosis by Nathan (1962) states 100% of the population are affected by 30 - 39. There is no evidence of accelerated involvement in the Poundbury population therefore, compared to a modern sample. Since it is probable that the dental age

assessment underestimates the ages at death, it is likely that the peak of incidence in Poundbury is in fact later than Nathan's (1962) sample, at about 45 - 55. There are no sex differences in incidence measures in Poundbury as opposed to some studies of modern samples and archaeological ones where males have shown a faster rate of incidence than females (Lawrence et al., 1966; Sager, 1969). One modern sample found the faster rate of incidence in females (Roche, 1957) whereas Nathan (1962) and Ingelmark et al.(1959) found no differences. There is a great variation between populations, therefore, in the relation of the sexes in simple incidence of degenerative joint disease.

There is a greater degree of severity in males in modern English samples (Lawrence et al., 1966) but not in the American sample of Nathan (1962). All modern studies report a very low increase of severity with age up to 50 - 60, after which a marked increase occurs. The low proportion of individuals in Poundbury with grades 3 - 4 of severity reflects the combination of a similar progression of severity as in modern samples and a small proportion of individuals surviving over 55. Males show a greater proportion of severe grades than females, particularly in the disc joints. Although this could reflect a greater proportion of males surviving beyond 55 than females, as was discussed in the previous section, the sex difference in frequency of severe grades should only be found in the oldest age group if this were the full explanation for its occurrence. Since the difference in severity between the sexes is found also in earlier age

groups it can be said that a true difference exists. It is usually assumed that males are engaged in heavier labour than females and are of larger body build which together account for the greater severity of degeneration in the spinal joints.

It is not known what the divisions of labour between the sexes would have been in Romano-British Dorchester. The greater severity in males may reflect not simply a greater physical demand from labour activities but also an earlier age of undertaking physically demanding activities than females. Other unpublished data from the Poundbury population demonstrate that males suffer a far higher frequency of Schmorl's Nodes than females. These are depressions in the vertebral body surface thought to result from disc herniation, typically occurring in young adults. This finding describes a greater susceptibility in young males than females to Schmorl's Nodes in Poundbury, possibly evidence of greater physical demands on the spine at this age. However, without an analysis of younger individuals it is difficult to tell if there is a younger age of onset in males. Certainly, by 20 - 25, there is no difference between the two sexes in incidence or severity.

The possibility of different degeneration processes existing in the two sexes could also be considered. However, there has been no evidence put forward by any of the structural or biochemical studies on degenerative changes in either soft or hard tissues for any such sex differences.

The number of joints affected in the spine is seen to increase significantly with age in both modern and archaeological samples (Lawrence et al., 1966; Stewart, 1958; Ingelmark et al., 1959). In Poundbury, the extent of involvement gives the best correlations against age. In the column, in Poundbury, combining both disc and facet no-one in the first age group of 20 - 25 has more than a score of 80 from 200. In the oldest age group of 45+ only about 15% has a score less than 80 from 200. Males have higher values than females and again this is not explained purely by a greater proportion of older males, but represents a real sex difference of greater rate of spread through the spine in males. The difference between the sexes has to be considered when using the extent of degenerative joint disease to assess age. No figures of age relation are given for modern samples for comparison from which as indication of definite ages at which specific measures of extent may be found. The sex difference is not evident until the 35 - 45 age group, which may be underestimated by about ten years. The mean values are still quite low at this point so an acceleration of involvement seems to occur after this time. Without exact information on ages it is not possible to give a detailed conversion from measures of extent of spinal degenerative joint disease to absolute age estimate. It can be said that very few joints are involved under 30 and that any spines displaying more than 50% involvement of both facets and disc joints will be over 50. Further work with measures of the extent of degenerative joint disease should prove a fruitful area for improvements in aging techniques.

A number of previous researchers have suggested that the facet and disc joints experience degenerative joint disease independently of one another. This view is supported here from the different distributions of facet and disc involvement throughout the spine and the low correlations between measures of degeneration in facets and discs of the same region compared to disc or facet correlations with other disc or facet measures of different regions. This is seen to result from different localized stresses operating on the two kinds of joint. The role of the two kinds of joint are quite different in the maintenance of stability, the facet joint acting as anchors to some extent while the disc joint takes the brunt of vertical compressive forces. Equally, the relationship of the two kinds of joint to the spinal curvature is different. The detailed biomechanical descriptions of the different stresses experienced by the two kinds of joints is beyond the scope of this thesis.

The males display a greater integrity within the spinal column regions than females. No work has been found that comments on this aspect elsewhere, so whether it is a specific feature of the Poundbury population or general is unknown. Assuming it represents a general observation in a population experiencing low levels of physical demand, it suggests that demand operates more generally throughout the male spine but is more localized in females. Stresses which could be specific to females are those from pregnancy which alters the usual distribution of weight over the hips, affecting the curve of the spine. This would particularly

affect the stress on joints of the lower thoracic and lumbar regions. The cervical region is noticeably more involved in males than females which may add support to this explanation. This indicates a far greater rate of degenerative joint disease in males than females, which is partly compensated for in the lower spine from the effects of pregnancy.

Males tend to show a greater coefficient of variation in the last age group than females. Similarly to the discussion of the same result in internal remodelling parameters this is indicative of a higher proportion of males in the 45+ (or 55+) age group.

10.3 Degenerative Joint Disease of the Spine and Cortical Thickness.

The external measure of bone structure displays the same relationship to spinal degenerative joint disease as to dental age except in the last group of the most extensive degenerative cases. Individuals with extensive degenerative joint disease, although representing the oldest members of the population do not have the extreme loss of bone characteristic of old age.

The two processes of bone loss and degenerative joint disease proceed independently of one another but involve the opposite behaviour as regards quantity of bone. The resorptive activity in cortical bone reduces dramatically the

amount of bone present, whereas the changes of degenerative joint disease involve an increase in the amount of bone present in the joint, both as osteophytes and in sclerosis. An escalation of bone formation is not consistent with extreme loss elsewhere thought to be mediated by a systemic hormonal-calcium action. The vertebral bodies themselves are frequently one of the most seriously affected sites of osteoporosis. Aging individuals appear to suffer, therefore, from one or the other in the more extreme forms of 55+, but not both. This is observed by other authors also (see Dequeker, 1975). An assessment of age at death that incorporated a consideration of both degenerative joint disease and cortical thickness therefore has a better chance of identifying the oldest individuals.

10.4 Mortality at Poundbury.

Summaries of demographic aspects of skeletal samples are presented by Brothwell (1972b) for British populations and by Angel (1972) for populations from the eastern Mediterranean. The average adult ages of death through different time periods are given in Tables 10.1 and 10.2, adapted from the two author's papers. It is seen from both series of figures that until the 1900s the average age at death is given as below 40, and very often only around 30. Examination of mortality figures from third world populations, many of which experience appalling social conditions, show higher mean adult ages of death than the majority of these figures for archaeological samples (Boddington, pers. comm.). It has been

Table 10.1 Average Age at Death for Adult Males and Females in Britain through Different Time Periods.

Time Period	Females	Males
Neolothic	28•3	31•5
Bronze Age	29•9	31•3
Iron Age	29•9	31•3
Romano-British	31•9	34•8
Dark Ages	31•3	33•7
Saxons Towns	29•9	36•0
Country	33•1	34•7
Medieval Towns	30•1	35•3
Country	31•3	35•3

(adapted from Brothwell, 1972b.)

Table 10.3 The % Frequency of Individuals found in the 50+ Age Category in Britain through Different Time Periods.

Time Period	(50+) %
Neolithic	5•3
Bronze Age	3•3
Iron Age	2•7
Romano-British	10•4
Dark Ages	4•5
Saxons	7•9
Medieval	9•9

(adapted from Brothwell, 1972b.)

Table 10.2 Average Age at Death of Adult Males and Females in the Eastern Mediterranean through Different Time Periods.

Time Period	Females	Males
1928-1929 : AD 1900	54·3	56·1
Romantic : AD 1800	37·3	40·2
Moslem : AD 1400	27·8	33·9
Byzantine : AD 600	31·1	37·7
Imperial Rome : AD 120	34·3	40·2
Hellenistic : 300 BC	36·6	42·6
Classic : 650 BC	34·6	44·5
Early Iron Age : 1150 BC	30·4	38·8
Late Bronze Age : 1500 BC	32·0	39·3
Middle Bronze Age : 2000 BC	31·0	36·7
Early Bronze Age : 3000 BC	29·6	33·5
Late Neolithic : 4000 BC	28·2	35·7
Middle Neolithic : 5000 BC	-	-
Early Neolithic : 6800 BC	29·8	33·6
Mesolothic : 9000 BC	24·9	32·0
Upper Palaeolithic : 30000 BC	28·7	33·3

(adapted from Angel, 1972)

Table 10.4 Age Distribution of the Remains from Maiden Castle, Dorset.

Age Group	Females	Males
20 - 25	41	25
25 - 35	41	64
35 - 45	14	7
45+	4	4

(adapted from Wheeler, 1943)

suggested increasingly that archaeological material characteristically is underaged and that life may not have been so nasty, brutish and short as popularly imagined.

The results from Poundbury presented here involve a consideration of a range of aging parameters and the mean age of death is indicated as 40 - 45 with only a small proportion of individuals surviving beyond sixty. A greater proportion of males are found in the older age groups than females. Compared to the ages presented by Brothwell and Angel, this is a higher mean age at death than any of the other archaeological samples. The greater proportion of males in the older age groups is consistent with the higher mean age of death in males compared to females in both the British and Mediterranean series. The percentage of individuals over 50 in British archaeological samples is given in Table 10.3, taken from Brothwell (1972b). The Romano-British period has a higher proportion of individuals over 50 than in the other time periods, but even so the results from Poundbury suggest the proportion over 50 would be at least 20% rather than the 10% indicated by Brothwell. It is concluded here, however, that very few survive beyond 60 years of age.

Table 10.4 summarizes the skeletal report on Maiden Castle, Dorset by Morant and Goodman, in Wheeler (1943). Maiden Castle could well have been the forerunner to Dorchester as one of the major administrative centres of the region under the Durotriges. The Maiden Castle remains indicate a greater proportion of females than males in the

older age categories in contrast to the other British samples and Poundbury. Maiden Castle is thought to have been a site of major conflict, which may account for the earlier age of death of many of the males through warfare. Again the mean age of death (25 - 35) is low compared to the equivalent Poundbury figure.

The mean ages at death given for the British and Mediterranean series and for Maiden Castle, considered separately, are comparable to the mean age at death indicated for Poundbury by age assessment from dental attrition. The examination undertaken here of the microparameters and degenerative joint disease complements the use of dental attrition giving a broader foundation on which to build the population mortality profile. It is suggested that dental age underestimates the absolute age at death but is a reliable indicator at Poundbury of relative age relationships between individuals. The mortality profile from the pubic symphysis age indicates an overestimation of age of the female population relative to the males. In addition age assessment from the pubic symphysis metamorphosis is not found to be consistent with other age-related changes and is therefore considered less reliable for age determination than dental attrition.

It is suggested that previous studies of age at death in archaeological samples have consistently underaged the individuals through the use of only one aging parameter. Maiden Castle, for example, was aged largely from cranial suture closure (Wheeler, 1943) and dental attrition is often

used on British samples. The use of microparameters can complement dental attrition to callibrate the 35 age point in the dental attrition scale. Degenerative joint disease is also a good indicator of age, particularly beyond 30, but similarly needs to be callibrated against some independent measure such as the microparameters. This type of callibration of measures against one another should help avoid the evident danger of underestimating age at death.

CONCLUSIONS

The comparison of different measures of age-related change has proved useful in describing the mortality pattern of the Romano-British population of Poundbury. In addition, it has been possible to evaluate the potential of degenerative joint disease of the spine and remodelling of cortical bone structure for age assessment.

Spinal degenerative joint disease shows a linear progression with age. Differences are seen between the sexes in the rate of the degeneration as regards severity and extent. Males tend to progress at a greater rate than females after about 40. Males show a greater coherence throughout the spine of degenerative changes. In both sexes the facet and disc joints undergo degeneration independently of one another.

The methods of age assessment from bone structure are inadequate as they depend on a regression equation when the parameters' relationship to age is non-linear. The raw parameter measures can be used in connection with other aging criteria to callibrate the rates of change. Methods could be devised using the measures of bone structure if the population were first divided into two broad age groups of pre-45 and post-45, assigned on the basis of other criteria. Two regressions could then be utilized for each of the

directions of age-related change observed in the measures.

Individuals do not develop both extreme bone loss and extensive degenerative joint disease as the processes involved in each are incompatible.

There is no reason to accept the argument for accelerated loss of bone or internal remodelling in Poundbury. The loss of bone is nowhere near as great as in modern samples. The incidence, severity and extent of degenerative joint disease in Poundbury is found to be of a comparable level to that of modern samples, describing an urban lifestyle with only low levels of physical demand.

The Poundbury population is finally depicted as having a mean age of death at 40 - 45, with a higher proportion of males in the 55+ ages than females. This represents a higher mean age of death than often estimated for pre-industrial populations and illustrates the danger of underestimation of age from the traditional aging methods.

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APPENDIX A

Dental Attrition and Pubic Symphysis.

Table 1a. Frequencies of the dental age categories by sex.

Dental age	females			males		
	Frequncy	%	Cum %	Frequency	%	Cum %
15 - 20	5	2	2	4	2	2
20 - 25	76	31	33	44	20	22
25 - 35	83	33	66	72	32	54
35 - 45	37	15	81	63	28	82
45+	47	19	100	41	18	100
	valid number of cases = 248			valid number of cases = 224		

Table 1b. Frequencies of the pubic symphysis age categories by sex.

Pubic Symphysis Age	females			males		
	Frequency	%	Cum %	Frequency	%	Cum %
15 - 20	2	1	1	7	4	4
20 - 25	4	3	4	11	6	10
25 - 35	10	7	11	75	43	53
35 - 45	51	36	47	41	24	77
45+	75	53	100	40	23	100
	valid number of cases = 142			valid number of cases = 174		

Table 2 Sex differences in the distributions of age at death by dental age and pubic symphysis age, using the Mann-Whitney U test.

Age	U	p
Dental Age	23282·0	0·0067
Pubic Symphysis Age	6416·5	0·0000

Table 3 Spearman's nonparametric rank correlation test between dental age and pubic symphysis age for each sex.

sex	correlation coefficient	p
females	·5982	·000
males	·7070	·000

Table 4 % Frequency of the attrition scores of each of the Molars by sex.

Molar	Grade	females % frequency	males % frequency
M_1	10	0	0
	20	0	2
	25	8	3
	30	12	8
	35	22	14
	40	21	16
	45	10	10
	50	11	18
	55	12	24
	60	3	5
M_2	10	1	0
	20	25	16
	25	19	11
	30	12	8
	35	19	18
	40	8	11
	45	6	10
	50	5	11
	55	5	11
	60	1	3
M_3	10	18	10
	20	51	29
	25	14	13
	30	3	10
	35	2	8
	40	3	12
	45	0	3
	50	1	3
	55	4	8
	60	1	3

Table 5 Sex differences in distribution of dental attrition and pubic symphysis metamorphosis scores, using the Mann-Whitney U test.

Molar	U	p
M_1	14058·5	·0000
M_2	12575·5	·0000
M_3	3486·5	·0000
PS	10357·0	·0045

Table 6 : Frequencies of the differences between the 3 molar attrition scores by sex.

Molars compared	score	females	males
$M_1 - M_2$	-11		
	-10	1	1
	- 9	1	1
	- 2	0	0
	- 1	1	4
	0	6	11
	1	20	20
	9	16	16
	10	36	33
	11	11	6
	19	7	5
	20	2	4
	21	0	0
	29	0	1
	30		
	31		
$M_2 - M_3$	-11		
	-10		1
	- 9		1
	- 2		0
	- 1	1	2
	0	13	15
	1	26	25
	9	3	8
	10	34	39
	11	15	6
	19	3	2
	20	4	1
	21		
	29		
	30		
	31		
$M_1 - M_3$	-11		1
	-10		1
	- 9		1
	- 1		1
	0	2	6
	1	6	6
	2	0	0
	9	0	1
	10	14	17
	11	25	21
	19	8	8
	20	27	28
	21	10	6
	29	0	1
	30	7	3
	31	1	0

Table 7 The sex differences in the distribution of difference between molar attrition scores using the Mann-Whitney U test.

Molars compared	U	p
$M_1 - M_2$	13800·5	0·0485
$M_2 - M_3$	4459·0	0·0963
$M_1 - M_3$	4062·5	0·0453

Table 8 : Frequency of the pubic symphysis scores.

pubic symphysis score	females	males
1	1	1
2	2	1
3	2	2
4	2	0
5	2	1
6	1	1
7	0	2
8	1	1
9	3	2
10	4	2
11	10	5
12	21	10
13	19	26
14	15	24
15	18	23

Table 9 : Distributions of Dental Attrition scores by pubic symphysis age group.

Pubic symphysis age group		20 - 25		25 - 35		35 - 45		45+	
		female	male	female	male	female	male	female	male
M_1	20	25			2				
	25	25	11	25		3			
	30	25		25	7	8			
	35		22	37	13	30		10	
	40	25	56	13	19	27	6	10	7
	45		11		17	11	12	20	
	50				22	5	24	24	28
	55				14	11	53	24	50
	60				4	5	6	10	14
M_2	20	75	20	75	6	20			
	25		30	25	13	20		11	
	30	25	20		15	15		11	
	35		20		17	17	6	11	8
	40		10		19	12	19	23	8
	45				10	6	19	8	15
	50				13	6	12	15	31
	55				8	3	31	15	38
	60						13	4	
M_3	10	67		50		14			
	15								
	20	33		50	50	36	30	27	
	25		33		13	21	10	40	
	30				20	7	10	7	10
	35		17		7	7	20	7	10
	40				17	7	10		20
	45				7		10		10
	50					7	10		20
	55				3		30	13	20
	60						3	6	10

Table 10 The median values of dental attrition scores in each pubic symphysis age group.

	Pubic symphysis age group							
	20 - 25		25 - 35		35 - 45		45+	
molar	female	male	female	male	female	male	female	male
M_1	25/30	40	30/35	45	40	55	50	55
M_2	20	25/30	20	35	30	50	40	50
M_3	10	20/25	10/20	30	20/25	40/45	25	45/50

Table 11 The median values of pubic symphysis metamorphosis scores in each dental age group.

	Dental age group							
	20 - 25		25 - 35		35 - 45		45+	
	female	male	female	male	female	male	female	male
pubic symphysis score	10	9	12	12	13/14	13	15	15

Table 12 Spearman's Rank Correlation Test between Dental Attrition and Pubic Symphysis Age.

	Pubic Symphysis Age	
Molar	Females coefficient/p	Males coefficient/p
M_1	·6138 ·000	·6436 ·000
M_2	·6362 ·000	·6827 ·000
M_3	·5398 ·000	·6918 ·000

Table 13 Spearman's Rank Correlation Test between Pubic Symphysis Metamorphosis and Dental Age.

	Dental Age	
	Females coefficient/p	Males coefficient/p
Pubic Symphysis	·6330 ·000	·7155 ·000

Table 14 % Distributions of Pubic Symphysis Metamorphosis Scores by Dental Age Group.

Pubic Symphysis Score	Dental Age Group 20 - 25		25 - 35		35 - 45		45+	
	female	male	female	male	female	male	female	male
1	3•2	6						
2	10	6		2•3				
3	10	19						
4	10							
5	6	6	3•2					
6				2•3				
7		6		5				
8			3•2	5				
9	10	12		2•3				
10	10	6	6	5				
11	13	12	13	12	11	6	10	
12	19	19	35	19	17	6	10	10
13	10	6	10	30	22	33	24	5
14			12	14	39	31	5	14
15			13	5	11	25	52	71

Table 15 Spearman's nonparametric rank correlation test between scores of Dental Attrition and Pubic Symphysis Metamorphosis.

variables	Females coefficient/p value			Males coefficient/p value		
	M_1	M_2	M_3	M_1	M_2	M_3
M_2	•8906 •000			•8944 •000		
M_3	•7169 •000	•7901 •000		•8110 •000	•9222 •000	
PS	•6651 •000	•6675 •000	•6518 •000	•6735 •000	•7177 •000	•6436 •000

APPENDIX B

Bone Structure.

Table 1 : Distribution of the Microages in 5 year Age Groups.

	Females	Males	Females	Males
	CLAGE		ADAGE	
-5 - 0	0	0	1	0
5	0	0	1	2
10	1	0	3	0
15	0	0	2	1
20	1	1	1	4
25	1	1	2	2
30	3	1	2	2
35	3	2	4	5
40	3	2	3	2
45	5	4	8	6
50	5	6	10	14
55	10	11	23	23
60	19	9	15	18
65	10	12	13	12
70	13	18	8	5
75	10	11	3	2
80	4	9	0	2
85	5	5	0	0
90	0	3	0	0
95	1	1	0	1
100	1	2		
105	1	1		
110	1	0		
115	1	0		
	KAO		KAF	
0 - 5	0	0	0	0
10	2	0	4	6
15	1	0	3	0
20	1	3	3	0
25	3	4	12	3
30	4	2	3	6
35	5	7	10	9
40	13	8	9	5
45	15	17	7	11
50	10	9	6	3
55	14	10	10	15
60	8	6	7	9
65	7	11	10	9
70	5	8	3	3
75	2	3	1	6
80	4	4	10	15
85	2	2		
90	1	1		
95	1	2		
100	0	1		
105	0	0		
110	1	0		
115	0	0		

Table 1 contd.

	Females	Males	Females	Males
	KAO			
120	0	0		
125	1	0		
130	0	1		
135	1	0		
	KAL		KAN	
0 - 5	4	0	0	0
10	17	11	15	20
15	4	5	22	13
20	4	7	7	7
25	5	5	7	7
30	9	3	0	7
35	8	2	15	27
40	12	10	0	0
45	4	10	4	7
50	7	5	4	0
55	7	10	7	0
60	7	8	19	13
65	7	13		
70	0	7		
75	4	2		
80	3	3		
	TAA		TAB	
0 - 5	0	0	0	0
10	1	0	0	0
15	2	2	0	0
20	1	0	1	2
25	2	0	2	1
30	2	2	2	0
35	2	4	2	3
40	1	2	2	3
45	3	3	2	2
50	3	3	3	5
55	3	3	3	2
60	9	10	4	4
65	18	17	10	16
70	19	23	19	24
75	15	16	20	16
80	9	8	13	14
85	6	4	10	4
90	3	2	5	3
95		2	0	1
100		1	0	0
105				1

Table 1 contd.

	Females	Males
	TAC	
-85 - -80	1	
-60 - -55		1
-40 - -35		1
-35 - -30	1	1
-25	1	1
-20	1	0
-15	2	2
-10	2	1
-5	2	2
0	4	2
5	2	3
10	4	5
15	4	8
20	9	7
25	7	11
30	13	17
35	9	16
40	16	7
45	11	9
50	9	1
55	1	5
60	1	1
65	0	0
70	1	1
75	0	1
80		
85		

	Females	Males
	TAD	
-190	1	
-120		1
-100 - -95	1	0
-90	0	1
-85	1	1
-80	0	1
-75	0	0
-70	1	2
-65	1	1
-60	0	0
-55	4	1
-50	1	0
-45	1	2
-40	4	3
-35	3	5
-30	1	3
-25	5	7
-20	8	5
-15	6	6
-10	6	13
-5	9	6
0	6	4
5	7	8
10	8	6
15	4	6
20	7	6
25	6	4
30	4	2
35	1	1
40	1	2
45	1	2
50	0	1
55	1	1
60		
65		

Table 1 contd.

TAB	Females	Males
-20	1	0
-15	0	0
-10	0	0
-5	0	0
0	1	1
5	1	2
10	1	1
15	2	7
20	7	4
25	9	5
30	11	13
35	12	13
40	12	16
45	12	9
50	12	8
55	5	7
60	3	7
65	3	3
70	3	2
75	1	0
80	1	1
85	1	1
90	0	0
95	0	0
100	0	1

Table 2 Sex differences in distributions of the Microages, using the Mann-Whitney U test.

Microage	U	p
CLAGE	9965·5	0·0376
ADAGE	9462·5	0·8646
KAO	9340·0	0·2147
KAF	1824·0	0·0613
KAL	1828·5	0·0339
KAN	188·0	0·7017
TAA	9462·5	0·8646
TAB	8740·5	0·2491
TAC	8405·5	0·3318
TAD	8950·0	0·9956
TAE	8641·0	0·7008

Table 3 Sex differences in distributions of the Microparameters, using the Mann-Whitney U test

Microparameter	U	p
KNSO	9340·0	0·2147
KNOF	1826·0	0·0625
KPCL	1815·5	0·0294
KNNH	192·0	0·7815
ADPO	9462·5	0·8646
TNSO	9135·5	0·6492
TOPL	8771·0	0·6912
TMOPL	9085·5	0·9012
CTHICK	11366·0	0·0000
AMOD	11571·5	0·9250

Table 4 : Distributions of the Microparameters.

KNSO	Females	Males
10 - 20	1	
20 - 30	2	
40	0	
50	2	2
60	3	2
70	3	4
80	5	4
90	3	9
100	22	17
110	16	13
120	14	14
130	10	9
140	7	10
150	6	6
160	4	5
170	0	3
180	1	0
190		0
200		1

KNOF	Females	Males
0 - 10	3	6
20	15	3
30	15	12
40	15	18
50	18	18
60	18	18
70	5	8
80	8	11
90	2	3
100	2	3

KPCL		
0 - 10	16	26
20	20	25
30	21	15
40	14	8
50	4	10
60	9	2
70	3	8
80	9	2
90	1	2
100	3	3

KNNH		
0	19	13
10	30	33
20	15	20
30	19	13
40	4	13
50	7	0
60	7	7

ADPO		
0 - 10	3	2
20	5	1
30	3	5
40	6	8
50	10	8
60	33	37
70	28	30
80	11	7
90		2
100		1

TNSO		
1	2	1
2	3	2
3	3	3
4	7	7
5	14	20
6	38	38
7	18	16
8	12	9
9	1	4
10	3	0

Table 4 contd.

TOPL	Females	Males
2	1	1
3	3	2
4	4	6
5	4	3
6	7	5
7	10	9
8	13	13
9	16	21
10	22	20
11	11	10
12	6	5
13	1	2
14	1	2

TMOPL	Females	Males
0 - ·5	0	1
1·0	9	7
1·5	63	62
2·0	27	25
2·5	1	5
3·0	0	0
3·5	1	1
4·0		

CTHICK		
1·0 - 1·5	1	0
2·0	7	2
2·5	16	3
3·0	30	8
3·5	28	24
4·0	13	38
4·5	5	18
5·0		6
5·5		1
6·0		0
6·5		

AMOD		
30 - 35	2	0
40	12	18
45	37	26
50	29	36
55	13	11
60	3	7
65	2	1
70	1	0
75	0	1
80	1	0
85	1	0

CNSO		
0 - 2	1	0
4	1	1
6	3	2
8	5	5
10	10	8
12	27	24
14	29	22
16	15	22
18	7	9
20	1	4
22	0	2
24	1	1
26		

Table 5 Correlations Between the Microages.

Female

ADAGE	•6887 •000									
KAO	•7566 •000	•5738 •000								
KAP	•6061 •000	•6742 •000	•6424 •000							
KAL	•7342 •000	•7750 •000	•7419 •000	•6859 •000						
KAN	•7426 •000	•7493 •000	•6484 •000	•7649 •000	•7911 •000					
TAA	•6887 •000	1•000 •000	•5738 •000	•6742 •000	•7750 •000	•7493 •000				
TAB	•6913 •000	•9888 •000	•5693 •000	•6913 •000	•7882 •000	•7535 •000	•9888 •000			
TAC	•5568 •000	•6437 •000	•5282 •000	•5516 •000	•6810 •000	•5146 •004	•6437 •000	•6443 •000		
TAD	•3610 •000	•4009 •000	•3513 •000	•3658 •001	•5196 •000	•4980 •007	•4009 •000	•3961 •000	•9316 •000	
TAE	•4034 •000	•5864 •000	•1983 •009	•3560 •001	•5169 •000	•5834 •001	•5864 •000	•5830 •000	•7366 •000	•7537 •000
	CLAGE	ADAGE	KAO	KAP	KAL	KAN	TAA	TAB	TAC	TAD

Table 5 contd.

Male

ADAGE	•4851 •000									
KAO	•7963 •000	•5444 •000								
KAF	•1022 •217	•1859 •069	•2951 •008							
KAL	•5676 •000	•7216 •000	•5913 •000	•3392 •012						
KAN	•8521 •000	•7695 •000	•8187 •000	•5663 •072	•8964 •000					
TAA	•4851 •000	1•000 •000	•5444 •000	•1859 •069	•7216 •000	•7695 •000				
TAB	•4793 •000	•9858 •000	•5339 •000	•1840 •071	•6950 •000	•7744 •000	•9858 •000			
TAC	•3679 •000	•5231 •000	•4004 •000	•0893 •240	•6351 •000	•6341 •007	•5231 •000	•5426 •000		
TAD	•1605 •041	•3034 •000	•2160 •008	-•0303 •406	•4972 •000	•3938 •082	•3034 •000	•3160 •000	•9199 •000	
TAE	•0416 •327	•4593 •000	•0601 •253	-•0767 •273	•5406 •000	•3070 •143	•4593 •000	•4666 •000	•6750 •000	•7289 •000
	CLAGE	ADAGE	KAO	KAF	KAL	KAN	TAA	TAB	TAC	TAD

Table 6 Correlations Between the Microparameters.

Female coefficients/p values

Variables	KNSO	KNOF	KPCL	KNNH	ADPO	TNSO	TOPL	TMOPL	CTHICK	AMOD
KNOF	·6424 ·000									
KPCL	-·7466 ·000	-·6851 ·000								
KNNH	-·6410 ·000	-·7459 ·000	·8037 ·000							
ADPO	·5738 ·000	·6738 ·000	-·7813 ·000	-·7864 ·000						
TNSO	·7478 ·000	·6533 ·000	-·7054 ·000	-·6533 ·000	·5784 ·000					
TOPL	·6033 ·000	·6287 ·000	-·6246 ·000	-·6365 ·000	·5695 ·000	·6322 ·000				
TMOPL	-·1090 ·099	-·0142 ·454	·0774 ·259	-·1366 ·253	·1266 ·067	-·2151 ·005	·3450 ·000			
CTHICK	-·0439 ·297	-·1635 ·091	·1246 ·143	-·0231 ·455	-·1296 ·C61	-·0713 ·197	-·1563 ·032	-·1318 ·060		
AMOD	-·0592 ·247	-·0593 ·322	-·0422 ·364	-·0165 ·468	·2422 ·002	-·1046 ·114	-·0035 ·484	·0660 ·227	-·2308 ·001	
CNSO	-·9329 ·000	·6690 ·000	-·7852 ·000	-·7860 ·000	·5930 ·000	·7803 ·000	·6044 ·000	-·0922 ·139	-·0076 ·460	-·0335 ·335

Table 6 contd.

Male — coefficients/p value

Variables	KNSO	KNOF	KPCL	KNNH	ADPO	TNSO	TOPL	TMOPL	CTHICK	AMOD
KNOF	·2974 ·008									
KPCL	-·5931 ·000	-·3374 ·013								
KNNH	-·7609 ·001	-·5663 ·072	·9097 ·000							
ADPO	·5444 ·000	·1904 ·064	-·7216 ·000	-·7351 ·001						
TNSO	·8172 ·000	·2615 ·018	-·5566 ·000	-·5921 ·010	·5847 ·000					
TOPL	·6776 ·000	·3758 ·001	-·4616 ·000	-·6511 ·006	·6000 ·000	·6596 ·000				
TMOPL	-·0515 ·282	·1013 ·211	·1561 ·121	·0112 ·485	·1433 ·053	-·1690 ·029	·2892 ·000			
CTHICK	·0669 ·220	·0038 ·488	-·0303 ·408	·2169 ·219	·0221 ·401	·0122 ·445	-·0041 ·482	·0051 ·477		
AMOD	-·2368 ·004	-·2065 ·055	-·0853 ·266	-·3995 ·088	·0528 ·281	-·1488 ·052	·0436 ·318	·1872 ·020	-·2214 ·004	
CNSO	·9512 ·000	·2529 ·020	-·5857 ·000	-·7973 ·000	·5246 ·000	·7791 ·000	·6174 ·000	-·0554 ·267	·0430 ·299	-·2039 ·008

Table 7 Correlations between the Microages and Dental Age and Pubic Symphysis Age.

	Dental Age		Pubic Symphysis Age	
Microages	Females	Males	Females	Males
CLAGE	•4098 •000	•2349 •009	•2492 •025	•1128 •169
ADAGE	•4617 •000	•2113 •021	•3577 •005	•0961 •213
KAO	•3782 •000	•2070 •021	•2089 •065	•0772 •261
KAF	•3109 •011	•1692 •133	•1506 •246	•2091 •107
KAL	•4055 •000	•4426 •001	•3087 •076	•1193 •251
KAN	•3036 •070	•4148 •102	•4148 •088	•2168 •320
TAA	•4617 •000	•2113 •021	•3577 •005	•0961 •213
TAB	•4921 •000	•2071 •023	•3905 •002	•1104 •180
TAC	•3448 •000	•0982 •179	•3484 •007	-•0335 •391
TAD	•2172 •011	•0092 •466	•2803 •024	-•1105 •183
TAE	•2647 •003	-•0430 •344	•2542 •037	-•0594 •314

Table 8 Mann-Whitney U test for Sex Differences in Distribution of Microages in each Dental Age Group.

Microage U/p	Dental Age 20 - 25	25 - 35	35 - 45	45+
CLAGE	223•5 0•1009	610•0 0•6947	322•5 0•8060	220•0 0•7892
ADAGE	200• 0•2608	511•5 0•6902	241•5 0•8129	119•2 0•0499
KAO	223•0 0•1570	535•0 0•7723	274•5 0•9832	194•0 0•9132
KAF	25•0 0•3484	123•0 0•2204	35•5 0•7584	45•5 0•5006
KAL	117•5 0•7633	176•5 0•7660	34•5 0•8877	25•5 0•4945
KAN	24•0 0•8205	16•0 0•5690		
TAA	200•0 0•2608	511•5 0•6902	241•5 0•8129	119•5 0•0499
TAB	212•0 0•3874	468•5 0•3417	231•0 0•6364	94•0 0•0074
TAC	152•0 0•0798	422•0 0•1753	150 0•0394	141•0 0•2709
TAD	146•0 0•0586	456•0 0•3639	170 0•1114	164•0 0•6703
TAE	139•0 0•0400	513•0 0•8745	203•0 0•5230	137•0 0•2231

Table 9 Means, Standard Deviations and Coefficients of Variation of the Microages by dental age group.

Micro-age	Dental Age Group	Females			Males		
		Mean	S.D.	Coeff of V	Mean	S.D.	Coeff of V
CLAGE	20 - 25	48	19	•386	54	14	•260
	25 - 35	60	16	•263	60	16	•263
	35 - 45	68	12	•179	68	15	•220
	45+	64	15	•234	64	20	•308
ADAGE	20 - 25	35	22	•629	43	15	•336
	25 - 35	49	13	•270	47	17	•361
	35 - 45	57	8	•133	55	14	•256
	45+	57	8	•141	50	14	•291
KAO	20 - 25	38	19	•499	44	14	•315
	25 - 35	51	18	•348	49	19	•387
	35 - 45	58	21	•363	59	22	•374
	45+	53	13	•239	51	19	•369
KAF	20 - 25	36	20	•552	47	14	•301
	25 - 35	40	21	•541	46	21	•460
	35 - 45	59	15	•255	57	19	•340
	45+	49	12	•245	53	16	•296
KAL	20 - 25	25	21	•834	24	16	•642
	25 - 35	33	20	•619	35	26	•725
	35 - 45	50	17	•351	52	12	•225
	45+	44	16	•370	49	20	•401
TAA	20 - 25	47	22	•473	56	15	•266
	25 - 35	63	14	•219	60	17	•290
	35 - 45	70	8	•110	68	14	•212
	45+	70	8	•117	62	15	•236
TAB	20 - 25	52	21	•402	59	15	•253
	25 - 35	66	13	•197	62	16	•259
	35 - 45	73	8	•102	69	13	•193
	45+	74	8	•106	65	14	•211
TAC	20 - 25	10	28	2•800	26	14	•559
	25 - 35	24	20	•846	18	21	1•142
	35 - 45	38	12	•317	30	14	•474
	45+	29	12	•401	20	23	1•183
TAD	20 - 25	-22	44	-2•033	3	26	9•630
	25 - 35	-7	31	-4•315	-14	30	-2•208
	35 - 45	13	24	1•820	1	20	15•420
	45+	-5	21	-4•702	-13	35	-2•783
TAE	20 - 25	28	22	•779	43	16	•381
	25 - 35	36	15	•425	36	14	•386
	35 - 45	45	19	•427	40	15	•365
	45+	41	14	•338	36	22	•623

Table 10 Correlation Between the Microages and Dental Attrition Scores and Pubic Symphysis Metamorphosis.

	Dental Attrition						Pubic Symphysis	
	M_1		M_2		M_3			
microage	Females	Males	Females	Males	Females	Males	Females	Males
CLAGE	·4668	·2796	·4290	·3468	·3139	·2343	·2218	·1209
	·000	·006	·000	·001	·023	·051	·040	·152
ADAGE	·4413	·2955	·3931	·3720	·2980	·2958	·3443	·0991
	·000	·006	·000	·001	·029	·021	·006	·205
KAO	·4359	·2249	·3363	·4033	·3569	·2664	·2066	·0738
	·000	·026	·001	·000	·010	·032	·065	·270
KAF	·3201	·3664	·1664	·2811	·1417	·0262	·2469	·1686
	·014	·020	·143	·066	·276	·455	·122	·159
KAL	·4115	·3923	·3025	·4665	·0736	·1977	·1910	·0917
	·001	·007	·012	·002	·363	·162	·186	·303
KAN	·4292	·2733	·2935	·3809	·0592	·1983	·2873	·3115
	·014	·256	·077	·139	·435	·335	·171	·248
TAA	·4413	·2955	·3931	·3720	·2980	·2958	·3442	·0991
	·000	·006	·000	·001	·029	·021	·006	·205
TAB	·4511	·2717	·4063	·3516	·2937	·2540	·3655	·1178
	·000	·010	·000	·001	·031	·041	·004	·164
TAC	·4627	·0617	·4277	·1966	·3698	-·1058	·1990	-·0204
	·000	·306	·000	·051	·010	·252	·081	·433
TAD	·3795	·0072	·3641	·0742	·4063	-·2318	·1240	-·0838
	·000	·476	·000	·271	·005	·061	·193	·247
TAE	·3165	-·0283	·3241	·0053	·3884	-·2169	·1732	-·0534
	·001	·409	·001	·483	·007	·074	·112	·331

Table 11 Correlations Between the Microparameters and Dental Age and Pubic Symphysis Age.

	Dental Age		Pubic Symphysis Age	
Microparameter	Female	Male	Female	Male
KNSO	•3782 •000	•2070 •021	•2039 •065	•0772 •261
KNOF	•3099 •011	•1733 •127	•1506 •246	•2091 •107
KPCL	-•4125 •000	-•4426 •001	-•3087 •076	-•1193 •251
KNNH	-•3524 •042	-•4294 •094	-•4374 •078	-•2168 •320
ADPO	•4617 •000	•2113 •021	•3577 •005	•0961 •213
TNSO	•4127 •000	•2499 •008	•2900 •019	•0534 •330
TOPL	•3681 •000	•2540 •008	•2261 •057	•2177 •036
TMOPL	•0583 •273	•1162 •136	-•0339 •408	•1186 •164
CTHICK	-•1849 •006	-•1000 •109	-•1246 •110	-•1738 •033
AMOD	•1194 •091	-•0890 •188	•0784 •272	•0898 •222
CNSO	•3127 •000	•2422 •005	•1260 •155	•0569 •310

Table 12 Means, Standard Deviations and Coefficients of Variation of the Microparameters by Dental Age Group.

Micro-parameter	Dental Age Group	Females			Males		
		Mean	S.D.	Coeff of V	Mean	S.D.	Coeff of V
KNSO	20 - 25	88	33	·380	100	22	·225
	25 - 35	109	25	·233	105	29	·278
	35 - 45	118	27	·226	121	28	·232
	45+	113	17	·154	109	29	·263
KNOF	20 - 25	33	20	·592	44	13	·294
	25 - 35	38	23	·601	44	25	·559
	35 - 45	58	19	·338	57	23	·398
	45+	46	11	·244	49	15	·313
KPCL	20 - 25	48	29	·606	41	19	·468
	25 - 35	34	21	·623	33	26	·777
	35 - 45	17	12	·740	15	8	·545
	45+	21	12	·584	18	16	·895
ADPO	20 - 25	40	22	·550	49	15	·302
	25 - 35	55	13	·246	52	17	·326
	35 - 45	62	8	·122	60	14	·235
	45+	62	8	·129	54	14	·264
TNSO	20 - 25	5	2	·417	5	1	·263
	25 - 35	6	2	·256	6	1	·266
	35 - 45	7	1	·175	6	1	·183
	45+	6	1	·152	6	2	·313
TMOPL	20 - 25	1·5	0·4	·303	1·2	0·4	·300
	25 - 35	1·4	0·3	·210	1·5	0·3	·222
	35 - 45	1·3	0·2	·174	1·4	0·2	·169
	45+	1·5	0·3	·190	1·5	0·3	·193
TOPL	20 - 25	6·5	2·7	·424	6·7	2·1	·322
	25 - 35	8·2	2·4	·286	7·7	2·3	·304
	35 - 45	8·8	2·0	·227	8·8	2·2	·254
	45+	9·1	1·7	·184	8·0	2·9	·368
CTHICK	20 - 25	3·1	0·5	·158	3·7	0·7	·203
	25 - 35	3·1	0·7	·219	3·8	0·6	·169
	35 - 45	3·1	0·6	·210	3·7	0·6	·158
	45+	2·7	0·7	·252	3·6	0·6	·162
AMOD	20 - 25	46	5	·114	46	5	·100
	25 - 35	45	7	·158	44	6	·137
	35 - 45	48	5	·096	47	8	·383
	45+	49	10	·198	45	6	·145
CNSO	20 - 25	10	4	·372	10	3	·329
	25 - 35	13	3	·232	13	3	·267
	35 - 45	14	3	·213	15	3	·229
	45+	13	2	·174	14	3	·252

Table 13 Mann-Whitney U Test for Sex Differences in Distribution of Microparameters in each Dental Age Group.

micro-parameter	Dental Age 20 - 25	25 - 35	35 - 45	45+
U/p				
KNSO	223·	535·0	274·5	194·0
	0·1570	0·7723	0·9832	0·9132
KNOF	25·0	125·0	36·5	45·5
	0·3484	0·2453	0·8261	0·5006
KPCL	111·5	176·5	34·5	25·5
	0·6075	0·7660	0·8877	0·4945
KNNH	23·0	16·0	-	-
	0·7336	0·5690	-	-
ADPO	200·0	511·5	241·5	119·5
	0·2608	0·6902	0·8129	0·0499
TNSO	211·5	456·5	212·0	170·0
	0·3725	0·3553	0·4226	0·5797
TOPL	215·0	450·5	234·0	140·5
	0·7715	0·3265	0·9253	0·2640
TMOPL	171·0	475·0	188·5	174·5
	0·0966	0·5079	0·2407	0·9057
CTHICK	310·0	632·0	311·5	242·5
	0·0004	0·0000	0·0001	0·0000
AMOD	294·5	621·5	291·0	166·5
	0·7446	0·7917	0·4119	0·1161
CNSO	287·0	685·5	358·0	279·0
	0·2181	0·7197	0·5366	0·2763

Table 14 : Distribution of Microages by Dental Age Group.

	Dental Age Group							
	20 - 25		25 - 35		35 - 45		45+	
CLAGE	Female	Male	Female	Male	Female	Male	Female	Male
0 - 10	3							
20	2	6	2					
30	13		5					5
40	18	13	2	13			5	
50	15	12	17	14		9	9	19
60	28	32	23	16	24	22	31	19
70	8	31	30	27	38	32	28	19
80	3	6	16	23	28	18	13	14
90	10			7	5	10	5	14
100			5			6	4	5
110						3	5	5
120						5		
ADAGE								
0 - 10	22	7		3				
20	11		3	7		4		11
30	6	7	8	6		3		
40	11	7	15	16		7		
50	19	43	17	10	17	7	19	39
60	23	36	37	19	50	40	38	33
70	5		17	16	27	32	8	11
80	3		3	3	6	7	5	6
90								
100								
KAO								
0 - 10	8							
20	8	6	3					6
30	19	13	5	10	5			11
40	24	18	20	23	6	14	14	5
50	17	38	19	16	31	24	27	28
60	13	12	22	3	21	28	36	11
70	3	13	23	29	16	10	14	22
80	8	6	2	13	10	3	4	11
90			3		6	7		6
100						11	5	
110			3					
120								
130			5	3				
140								

Table 14 contd.

	Dental Age Group							
	20 - 25		25 - 35		35 - 45		45+	
KAF	Female	Male	Female	Male	Female	Male	Female	Male
0 - 10	11		5	12				
20	17							
30	5		32			8		18
40	23	50	16	17		15	30	
50	22			30	33	15	20	9
60	5	25	11	23	34	8	30	46
70	11	25	15			16	20	18
80	6		11	18	33	38		9
KAL								
0 - 10	43	22	18	24				
20	3	34	18	11				13
30	18	11	5	18	17		25	
40	15	11	27		17	25	25	25
50	7	22	9	6	17	25	13	
60	7		14	17	17	8	25	25
70	3			12	17	42		37
80	4		9	12	17		12	
KAN								
10	23	25	10	25				
20	46	25	20	25				
30			20	25				
40		50	30		100		100	100
50	8		10					
60	23		10	100	100			
TAA								
0 - 10	3							
20	11	7		3				
30	17			3				
40	5	7	9	10		4		6
50	11	7	11	13		10		5
60	14	22	14	6	6	7	10	28
70	22	57	35	33	55	33	38	33
80	14		22	29	22	25	42	17
90	3		9		17	17	10	11
100				3		4		
110								

Table 14 contd.

	Dental Age Group							
	20 - 25		25 - 35		35 - 45		45+	
TAB	Female	Male	Female	Male	Female	Male	Female	Male
0 - 10								
20	6	7						
30	16			3				
40	14		3	10		4		
50	6	14	14	16		10		11
60	11	7	9	3		4	5	6
70	25	57	31	33	39	28	28	55
80	16	14	32	32	39	33	38	17
90	6		11	3	22	17	29	11
100						4		
TAC								
-80	3							
-70								
-60								
-50				3				
-40								
-30	3							6
-20	5		3					6
-10	12		3					6
0	14		8	10				
10	9	15	9	24		4	14	
20	14	16	17	10	6	17	5	11
30	11	23	11	23	18	40	33	30
40	18	38	23	20	23	14	29	29
50	11		23	7	35	14	19	6
60		8	3	3	18	7		6
70						4		
TAD								
-60	9		6	7				18
-50	11		5					
-40	6	8	9				10	
-30	8	8	6	23			4	
-20	12	8	5	10	6	14	5	6
-10	11		12	10	12	15	19	23
0	9	15	8	20	23	25	24	18
10	5	16	12	10	6	14	14	11
20	15	23	11	10	12	14	10	6
30	11	15	20	7	17	7	9	12
40			3		12	7	5	6
50	3	8	3	3	6	4		
60					6			

Table 14 contd.

	Dental Age Group							
	20 - 25		25 - 35		35 - 45		45+	
TAB	Female	Male	Female	Male	Female	Male	Female	Male
-20	6							
-10								
0	3							6
10	8		3					6
20	23		3	20	6	4	14	12
30	11	31	34	13	23	26	5	5
40	15	23	23	37	12	26	24	42
50	23	15	20	13	30	14	38	11
60	5	16	8	14	5	18	9	12
70	3	7	6	3	18	11	10	
80	3	8	3					
90					6			
100								6

Table 15 : Distribution of Microparameters by Dental Age Group.

	Dental Age Group							
	20 - 25		25 - 35		35 - 45		45+	
KNSO	Female	Male	Female	Male	Female	Male	Female	Male
0 - 10								
20	3							
30	5							
40								
50	8			3				6
60	6	6	6	7				
70	8	7		3	5			11
80	8	6	5	6				
90	8	12	11	16	6	10	5	5
100	24	7	11	13	15	18	27	28
110	6	31	14	7	16	10	9	
120	10	12	22	3	21	21	27	11
130	3	13	12	19	11	17	18	6
140	3	6	11	10	10		5	22
150	8		2	13	5	3	4	5
160			3		6	11	5	6
KNOF								
0 - 10	17		5	12				
20	11		27					
30	5	25	31	6		15	10	18
40	34	25		35	17	23	30	9
50	16	25	5	23	33	8	40	28
60	11	25	21	6	17	16	10	27
70	6						10	9
80			6	6	16	23		9
90				6	17	7		
100			5	6		8		
KPCL								
0 - 10	11		9	29	33	42	25	50
20	10	22	18	18	34	33	25	13
30	15	11	32		16	25	25	25
40	18	11	5	18	17		25	
50		34	9	11				12
60	10		18	18				
70	4	22	4					
80	21		5	6				
90	4							
100	7							

Table 15 contd.

	Dental Age Group							
	20 - 25		25 - 35		35 - 45		45+	
KNNH	Female	Male	Female	Male	Female	Male	Female	Male
0 - 10	31	50	50	25	100	100	100	100
20	15		20	50				
30	16		30	25				
40	7	25						
50	16							
60	15	25						
ADPO								
0 - 10	11	7		3				
20	20			3				
30	5		9	7		4		11
40	8	14	11	16		10		
50	14	15	11	6			10	17
60	25	64	26	26	56	32	33	39
70	11		34	36	22	29	47	22
80	6		9		22	21	10	11
90				3		4		
TNSO								
0								
1	8							
2	11	7		3				6
3	11		3	3				11
4	8	21	17	13		4		
5	28	29	11	27	6	3	19	22
6	17	29	37	27	50	63	48	22
7	17	14	11	13	22	11	19	17
8			17	13	16	8	14	22
9						11		
10			3		6			
TMOPL								
•5		7						
1•0	9	14	9	7	12	4	5	6
1•5	62	58	60	56	76	71	52	53
2•0	23	21	31	30	12	25	43	35
2•5	3			7				6
3•0	3							
3•5								

Table 15 contd.

	Dental Age Group							
	20 - 25		25 - 35		35 - 45		45+	
TOPL	Female	Male	Female	Male	Female	Male	Female	Male
0 - 1								
2	6							6
3	11			7				3
4	12	23	3	3		7	3	6
5	2	8	11					6
6	15		6	13	12	7		
7	11	15	11	7	12	7	10	11
8	6	23	6	17	11	4	23	18
9	17	16	26	26	12	25	15	18
10	14	15	14	14	29	25	28	6
11	3		14	10	18	11	14	23
12	3		6	3		10	5	
13			3			4	5	6
14					6			
CTHICK								
0 - ·5								
1·0								
1·5			2				5	
2·0	2	4	8	2		2	12	
2·5	10	4	8	2	29		20	6
3·0	34	12	21	5	25	6	31	7
3·5	38	16	35	19	21	23	17	34
4·0	14	28	19	44	14	42	13	34
4·5	2	32	7	15	11	25	2	19
5·0		4		13				
5·5						2		
AMOD								
30 - 35			5			6	5	
40	15	6	18	33	5	13	13	29
45	36	50	37	17	24	22	27	33
50	36	32	21	43	42	31	23	14
55	10	6	10	4	29	16	14	19
60		6	4			9	9	5
65	3		3	3			4	
70			2					
80						3	5	

Table 15 contd.

	Dental Age Group							
	20 - 25		25 - 35		35 - 45		45+	
CNSO	Female	Male	Female	Male	Female	Male	Female	Male
0 - 2	2							
4	5	6	2					
6	10			6				4
8	13	11	7	7	4			8
10	15	22	9	9	4	9	12	
12	27	17	20	19	21	21	30	30
14	16	27	33	18	38	25	31	16
16	7	11	22	29	21	21	19	19
18	5	6	7	12	8	12	4	19
20						9	4	
22								4
24					4	3		

Table 16 Correlations of Microparameters with Dental Age between each Dental Age Category.

micro-parameter	Dental Age Pairs									
	20-25 to 25-35		25-35 to 35-45		35-45 to 45+		20-25 to 35-45		25-35 to 45+	
	female	male	female	male	female	male	female	male	female	male
KNSO	•2808	•1170	•1725	•1861	-•1068	-•1078	•4735	•2871	•0751	•0839
	•002	•140	•055	•047	•210	•189	•000	•009	•259	•243
KNOF	•0972	-•0840	•4223	•2519	-•1899	-•1732	•4855	•2353	•2942	•1527
	•238	•291	•003	•064	•171	•164	•001	•129	•033	•180
KPCL	-•2399	-•1406	-•4846	-•2897	•0725	•1476	-•5921	-•6359	-•3331	-•2263
	•022	•160	•001	•043	•374	•227	•000	•000	•020	•103
KNNH	-•2890	-•2618	-•3963	-•5251	-	-	-•3648	-•6599	-•2015	-•4201
	•068	•139	•101	•142	-	-	•091	•077	•265	•203
ADPO	•2918	•1588	•2282	•2535	-•1149	-•1261	•4986	•4163	•1344	•1781
	•001	•074	•018	•012	•197	•155	•000	•000	•127	•070
TNSO	•3044	•1587	•1955	•3291	-•1856	-•1082	•5499	•4411	•0451	•1727
	•001	•076	•038	•002	•083	•194	•000	•000	•351	•078
TOPL	•2540	•2082	•0938	•2249	•0050	-•0306	•3893	•3414	•0768	•2099
	•005	•030	•200	•024	•486	•404	•000	•004	•259	•043
TMOPL	•1060	•2269	-•1427	-•2327	•3203	•1876	-•0817	•1218	•1087	•0266
	•142	•020	•099	•020	•009	•066	•231	•173	•180	•415
CTHICK	•2647	-•1394	•2303	-•2459	•0365	-•4024	•2837	-•1425	•0054	-•4288
	•000	•050	•003	•002	•359	•000	•000	•072	•475	•000
AMOD	-•1224	-•1321	•1940	•1907	-•1635	-•0268	•1219	•0786	•0789	•1100
	•093	•101	•026	•038	•098	•410	•117	•253	•235	•177
CNSO	•3315	•1678	•1542	•1106	-•0657	-•1332	•4746	•2980	•0550	•0239
	•000	•047	•056	•144	•286	•112	•000	•004	•296	•415

Table 17 % Gain and Loss in Microparameters (mean values) with Dental Age.

micro-parameter	Age-related % Change			Age-related % Change		
	Females			Males		
KNSO	20 - 25 to 35 - 45		+33%	20 - 25 to 35 - 45		+21%
		to 45+	- 5%		to 45+	-10%
KNOF	20 - 25 to 35 - 45		+74%	20 - 25 to 35 - 45		+30%
		to 45+	-26%		to 45+	-15%
KPCL	20 - 25 to 35 - 45		-65%	20 - 25 to 35 - 45		-64%
		to 45+	+25%		to 45+	+23%
ADPO	20 - 25 to 45+		+56%	20 - 25 to 35 - 45		+24%
					to 45+	-10%
TNSO	20 - 25 to 35 - 45		+48%	20 - 25 to 35 - 45		+26%
		to 45+	- 6%		to 45+	-10%
TOPL	20 - 25 to 45+		+41%	20 - 25 to 35 - 45		+63%
					to 45+	- 9%
TNOPL	20 - 25 to 35 - 45		-11%	20 - 25 to 25 - 35		+20%
		to 45+	+16%	25 - 35 to 35 - 45		- 7%
				35 - 45 to 45+		+ 8%
AMOD	20 - 25 to 25 - 35		- 1%	20 - 25 to 25 - 35		- 5%
	25 - 35 to 45+		+ 8%	25 - 35 to 35 - 45		+ 6%
				35 - 45 to 45+		- 5%
CNSO	20 - 25 to 35 - 45		+34%	20 - 25 to 35 - 45		+25%
		to 45+	- 6%		to 45+	- 5%
CTHICK	20 - 25 to 25 - 35		+0·8%	20 - 25 to 25 - 35		+ 3%
		to 45+	-12%		to 45+	- 6%

Table 18 Normalized Measure of Cortical Thickness against Dental Attrition and Pubic Symphysis Scores.

	Females	Males
M_1	-·0776 ·192	-·0988 ·153
M_2	-·0016 ·493	-·1005 ·153
M_3	-·0923 ·241	-·0555 ·323
PS	-·1348 ·095	-·2088 ·016

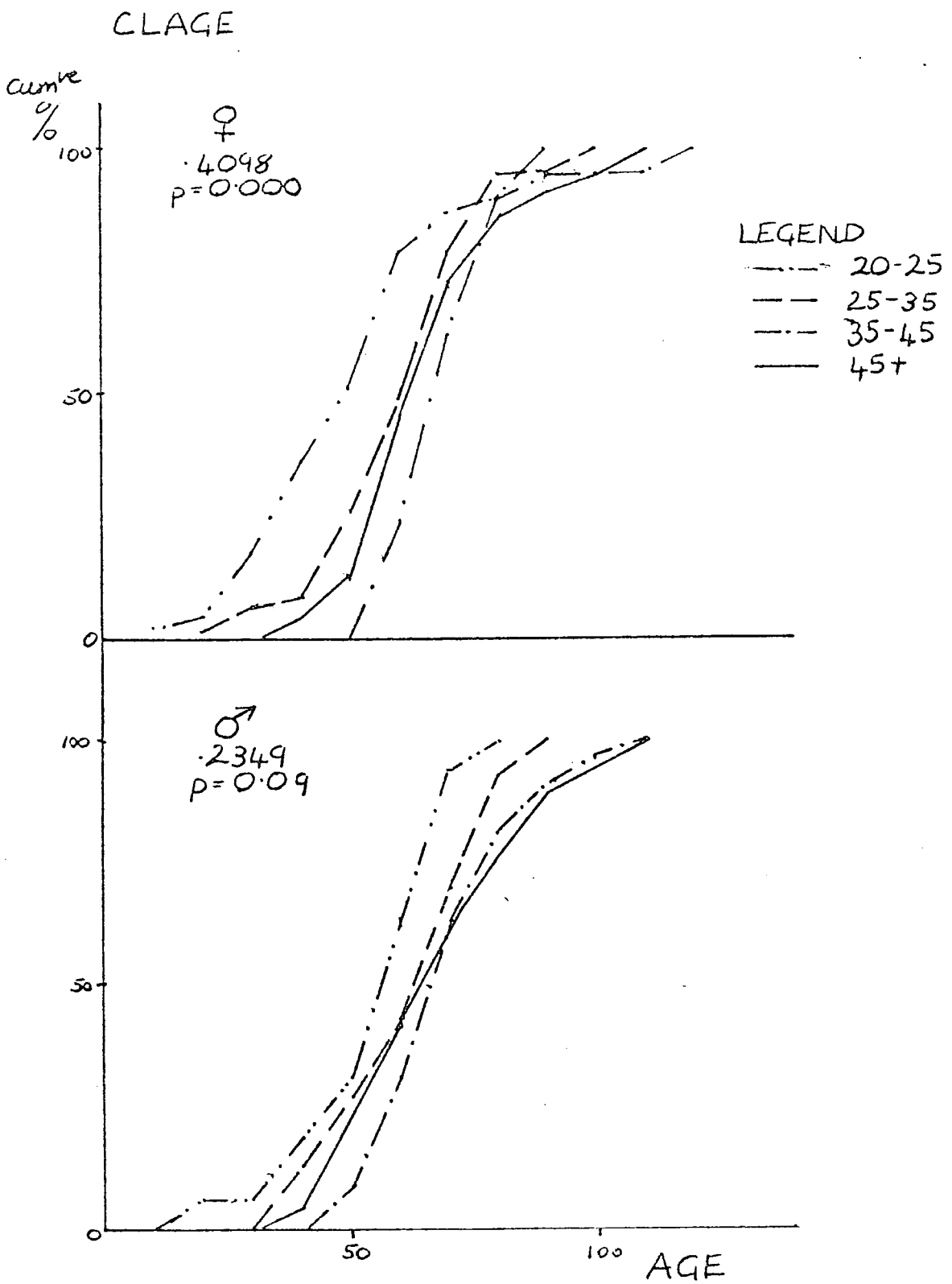


FIG. 1. CUMULATIVE % FREQUENCIES OF CLAGE IN 10 YEAR INTERVALS PLOTTED BY DENTAL AGE GROUP.

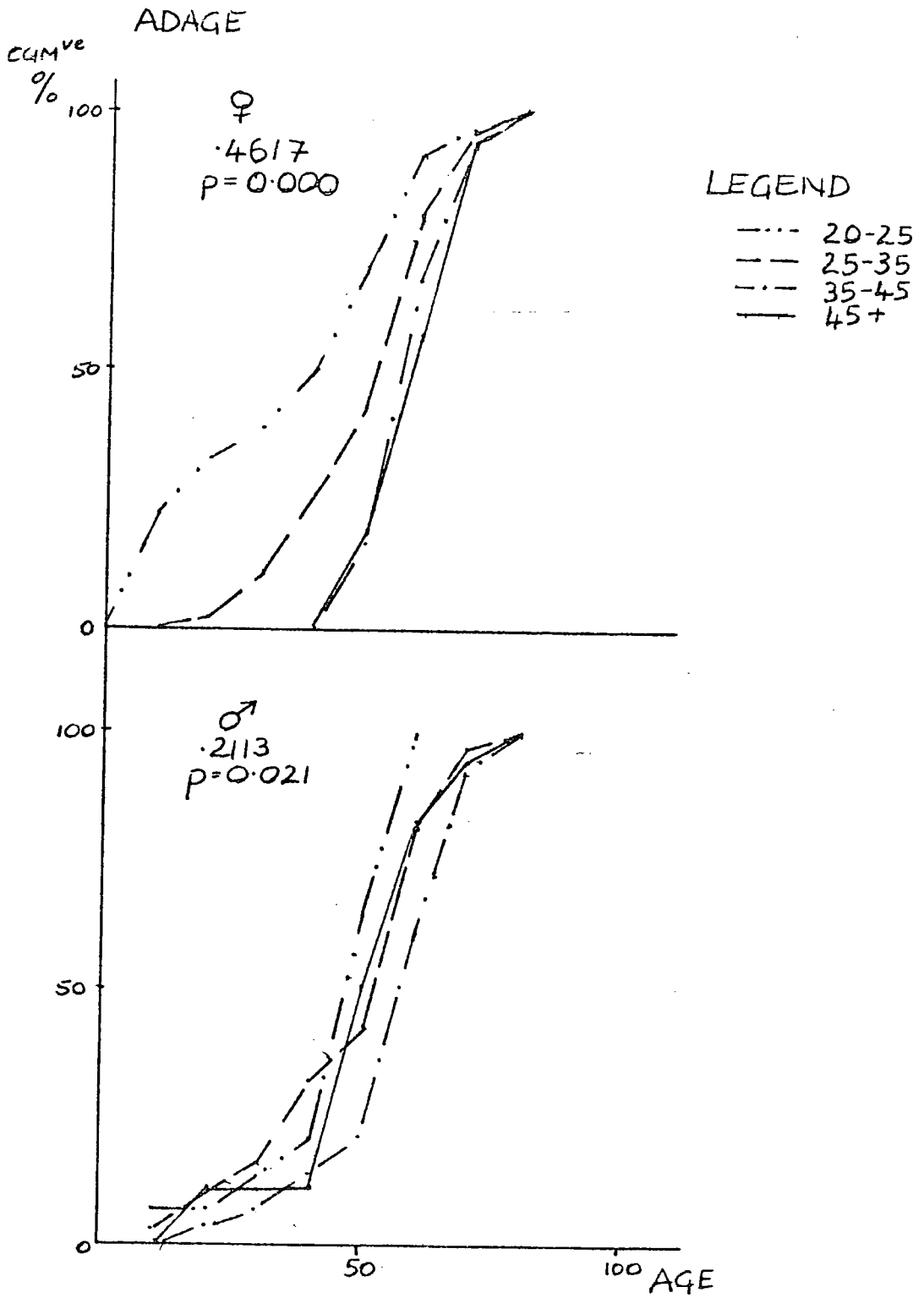


FIG. 2. CUMULATIVE % FREQUENCIES OF ADAGE IN 10 YEAR INTERVALS PLOTTED BY DENTAL AGE GROUP.

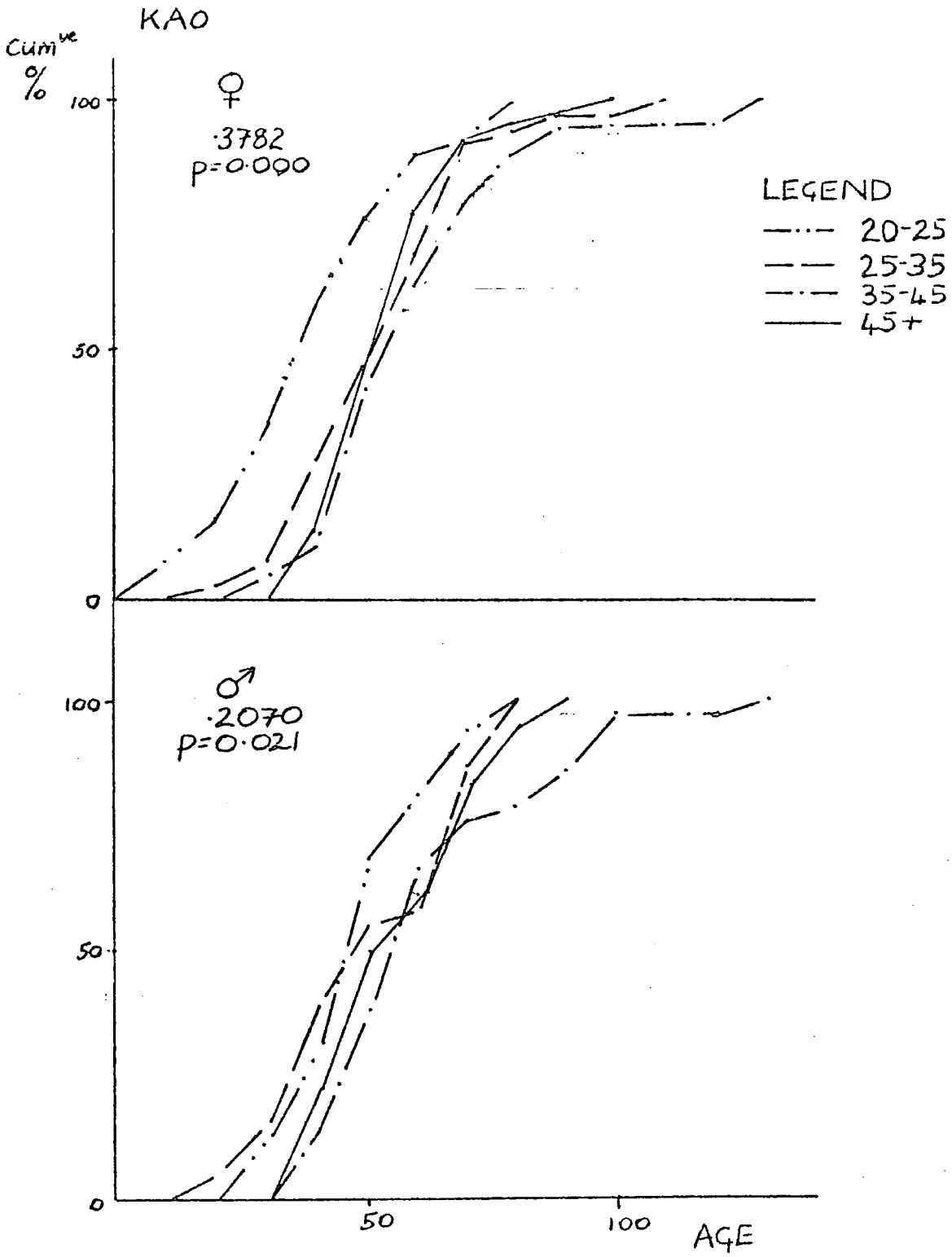


FIG. 3. CUMULATIVE % FREQUENCIES OF KAO IN 10 YEAR INTERVALS PLOTTED BY DENTAL AGE GROUP.

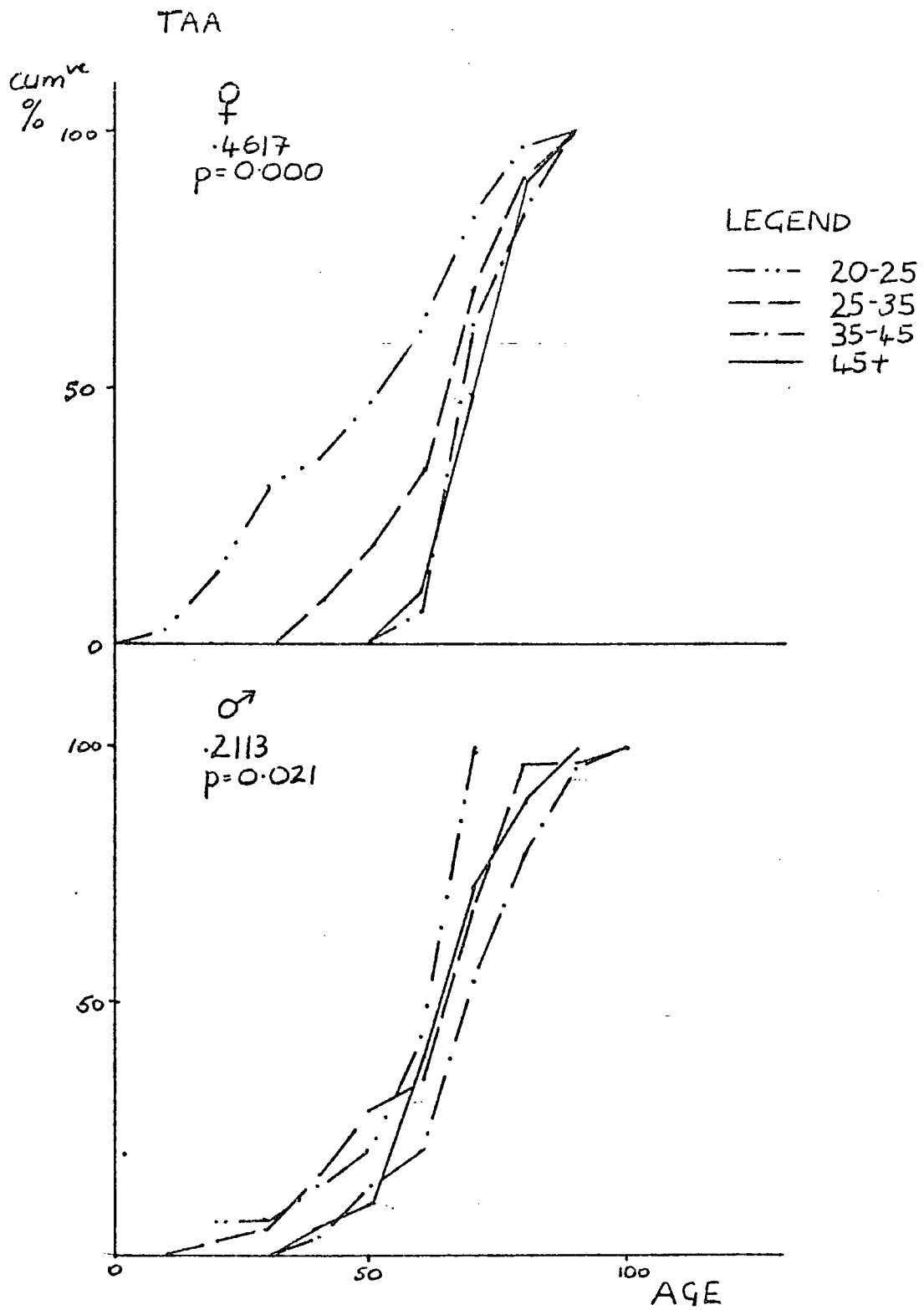


FIG. 4. CUMULATIVE % FREQUENCIES OF TAA IN 10 YEAR INTERVALS PLOTTED BY DENTAL AGE GROUP.

APPENDIX C

Degenerative Joint Disease of the Spine.

Table 1 Correlation Between Maximum Severity Spine Measures and Dental Age and Pubic Symphysis Age.

	Dental Age		Pubic Symphysis Age	
Spine Score	Females	Males	Females	Males
Facets Column	·6457	·6128	·4676	·4844
Cervical	·5979	·6634	·3690	·5264
Thoracic	·4576	·5063	·3627	·3009
Lumbar	·6439	·5311	·5415	·4974
Discs Column	·6200	·5360	·5412	·5051
Cervical	·6126	·5649	·3274	·4700
Thoracic	·4530	·4895	·4552	·4596
Lumbar	·6388	·5535	·5496	·4898
Combined Column	·7124	·6395	·5670	·5567
Cervical	·6930	·6868	·4087	·5624
Thoracic	·5699	·5886	·4807	·4802
Lumbar	·7390	·6508	·6340	·5787

p = 0·000 in all cases

Table 2 Correlation Between % Extent Spine Measures and Dental Age and Pubic Symphysis Age.

Spine Score		Dental Age		Pubic Symphysis Age	
		Females	Males	Females	Males
Facets	Column	·6564	·6783	·4423	·5111
	Cervical	·6127	·6856	·3473	·4860
	Thoracic	·4509	·5566	·3941	·4263
	Lumbar	·6295	·5734	·4408	·4548
Discs	Column	·6309	·6275	·4932	·5691
	Cervical	·6212	·5678	·3823	·5383
	Thoracic	·4828	·5772	·4850	·5431
	Lumbar	·6602	·6826	·5113	·5528
Combined	Column	·7373	·7180	·5209	·6083
	Cervical	·7165	·6911	·4275	·5587
	Thoracic	·5756	·6533	·5096	·5938
	Lumbar	·7341	·7263	·5430	·6006

P = 0·000 in all cases

Table 3 Correlation Between % Extent x Maximum Severity Spine Measures and Dental Age and Pubic Symphysis Age.

	Dental Age		Pubic Symphysis Age	
Spine Score	Females	Males	Females	Males
Facets Column	•6897	•6918	•4804	•5397
Cervical	•6149	•7002	•3637	•5215
Thoracic	•4702	•5675	•3909	•4064
Lumbar	•6608	•5786	•4878	•4688
Discs Column	•6617	•5985	•5367	•5732
Cervical	•6241	•5770	•3576	•5156
Thoracic	•5176	•5319	•4986	•5300
Lumbar	•6747	•6345	•5526	•5198
Combined Column	•7408	•6818	•5616	•6066
Cervical	•7086	•7024	•4249	•5729
Thoracic	•6080	•6159	•5216	•5590
Lumbar	•7400	•6991	•5966	•5783

p = 0•000 in all cases

Table 4 Correlation Between Maximum Severity Spine Measures and Dental Attrition and Pubic Symphysis Metamorphosis.

	M_1		M_2		P.S.	
Spine Score	Females	Males	Females	Males	Females	Males
Facets Column	•5684	•6487	•5778	•6181	•4551	•4975
Cervical	•5039	•6601	•5047	•6176	•4049	•5435
Thoracic	•4491	•5440	•4767	•4938	•3366	•3204
Lumbar	•5973	•5265	•5615	•5261	•5075	•5043
Discs Column	•5959	•5486	•6202	•5633	•4593	•5559
Cervical	•5958	•5438	•5829	•5479	•2696	•4959
Thoracic	•4652	•5712	•4624	•5517	•4297	•5094
Lumbar	•6471	•5938	•6881	•6351	•5062	•5151
Combined Column	•6661	•6688	•6927	•6710	•5051	•5974
Cervical	•6510	•6580	•6590	•6447	•4005	•5880
Thoracic	•5587	•6691	•5711	•6293	•4306	•5253
Lumbar	•7122	•6628	•7428	•6828	•5741	•5940

$p = 0 \cdot 000$ in all cases

Table 5 Correlation Between % Extent Spine Measures and Dental Attrition and Pubic Symphysis Metamorphosis Scores

Spine Score	M_1		M_2		P.S.	
	Females	Males	Females	Males	Females	Males
Facets Column	•5775	•6843	•5882	•6499	•4332	•5374
Cervical	•5226	•6793	•5116	•6330	•3654	•5145
Thoracic	•4153	•5964	•4530	•5335	•3664	•4414
Lumbar	•5575	•5541	•5511	•5479	•4368	•4775
Discs Column	•6189	•6462	•6431	•6577	•4743	•6176
Cervical	•5964	•5574	•5922	•5473	•3454	•5490
Thoracic	•4461	•6241	•4810	•6282	•4850	•5857
Lumbar	•6784	•6907	•7215	•6915	•4855	•6096
Combined Column	•7275	•7324	•7532	•7198	•5073	•6504
Cervical	•6837	•6823	•6754	•6589	•4142	•5819
Thoracic	•5752	•7089	•6081	•6734	•4876	•6225
Lumbar	•7153	•7192	•7554	•7109	•5190	•6418

$p = 0 \cdot 000$ in all cases

Table 6 Correlation Between % Extent x Maximum Severity Spine Measures and Dental Attrition and Pubic Symphysis Metamorphosis Scores.

	M_1		M_2		P.S.	
Spine Score	Females	Males	Females	Males	Females	Males
Facets Column	·6076	·7098	·6170	·6738	·4689	·5604
Cervical	·5249	·6865	·5152	·6444	·3936	·5453
Thoracic	·4463	·6106	·4779	·5438	·3664	·4252
Lumbar	·5894	·5653	·5718	·5542	·4818	·4847
Discs Column	·6479	·6253	·6745	·6335	·4959	·6252
Cervical	·6032	·5588	·5968	·5478	·3130	·5373
Thoracic	·4882	·6050	·5111	·5978	·4958	·5750
Lumbar	·6942	·6650	·7318	·6838	·5143	·5590
Combined Column	·7297	·7093	·7616	·6934	·5242	·6472
Cervical	·6728	·6726	·6673	·6535	·4101	·5969
Thoracic	·6068	·6902	·6290	·6512	·4970	·5992
Lumbar	·7240	·7068	·7673	·7163	·5635	·6082

$p = 0{\cdot}000$ in all cases

Table 7 The % Frequency of Presence of the Vertebrae by Sex.

Vertebra (osteological unit)	% present females	% present males
C1	57	65
2	60	66
3	56	60
4	59	61
5	59	60
6	62	62
7	63	65
T1	67	67
2	68	68
3	68	66
4	68	68
5	67	67
6	67	68
7	65	68
8	66	69
9	63	69
10	65	69
11	66	70
12	67	70
L1	69	73
2	73	76
3	76	77
4	77	79
5	77	78
6	3	5
SAC	75	75

Table 8 % of each joint affected by osteoarthosis by sex.

Vertebral facet joint (anatomical unit)	% affected of females	% affected of males
C1	7	9
2	32	47
3	41	51
4	36	46
5	28	37
6	21	30
7	31	49
T1	21	23
2	30	29
3	34	36
4	34	43
5	32	45
6	15	24
7	13	21
8	14	23
9	18	23
10	18	28
11	24	25
12	23	32
L1	21	34
2	31	43
3	39	46
4	55	57
5	49	40

Table 9 % of each joint affected by osteophytosis by sex.

Vertebral disc joint (anatomical unit)	% affected of females	% affected of males
C1	41	52
2	16	24
3	23	42
4	33	51
5	45	57
6	40	55
7	24	33
T1	27	28
2	39	43
3	56	60
4	62	60
5	66	62
6	59	66
7	63	69
8	68	72
9	61	64
10	50	64
11	45	63
12	33	57
L1	36	61
2	43	69
3	56	71
4	51	68
5	57	66

Table 10 Calculation of Spearman's Rank Correlation Coefficient for the Sexes in Distribution throughout the Spine of Osteoarthrosis.

Facet Joint	Rank		Difference in Rank(di)	$(di)^2$
	Females	Males		
C1	24	24	0	0
2	8	4	4	16
3	3	2	1	1
4	5	5	0	0
5	13	11	2	4
6	16	15	1	1
7	10	3	7	49
T1	16	20	-4	16
2	12	16	-4	16
3	6	12	-6	36
4	6	8	-2	4
5	8	7	1	1
6	21	19	2	4
7	23	23	0	0
8	22	20	2	4
9	19	20	-1	1
10	19	17	2	4
11	14	18	-4	16
12	15	14	1	1
L1	18	13	5	25
2	10	8	2	4
3	4	5	-1	1
4	1	1	0	0
5	2	10	-8	64

$$\Sigma di^2 = 268$$

$$r_s = 1 - \frac{6(268)}{(24)^3 - 24}$$

$$= 0{\cdot}883$$

significantly correlated at $p < 0{\cdot}01$

Table 11 Calculation of Spearman's Rank Correlation Coefficient for the Sexes in Distribution throughout the spine of Osteophytosis

Disc Joint	Rank		Difference in Rank(di)	$(di)^2$
	Females	Males		
C2	23	23	0	0
3	22	20	2	4
4	18	18	0	0
5	12	15	-3	9
6	15	17	-2	4
7	21	21	0	0
T1	20	22	-2	4
2	16	19	-3	9
3	8	13	-5	25
4	4	13	-9	81
5	2	11	-9	81
6	6	6	0	0
7	3	3	0	0
8	1	1	0	0
9	5	8	-3	9
10	11	8	3	9
11	12	10	2	4
12	18	15	3	9
L1	17	12	5	25
2	14	4	10	100
3	8	2	6	36
4	10	5	5	25
5	7	6	1	1

$\Sigma di = 435$

$$r_s = 1 - \frac{6(435)}{(23)^3 - 23}$$

$= 0{\cdot}785$

significantly correlated at $p < 0{\cdot}01$

Table 12 Calculation of Spearman's Rank Correlation Coefficient for the Facets and Discs in Distribution throughout the Spine of Degenerative Joint Disease.

Joint	Females				Males			
	Rank		difference in rank		Rank			
	Facets	Discs	(di)	$(di)^2$	Facets	Discs	(di)	$(di)^2$
C2	8	23	-15	225	4	23	-19	361
3	3	22	-19	361	2	20	-18	324
4	5	18	-13	169	5	18	-13	169
5	13	12	1	1	11	15	- 4	16
6	16	15	1	1	15	17	- 2	4
7	10	21	-11	121	3	21	-18	324
T1	16	20	- 4	16	20	22	- 2	4
2	12	16	- 4	16	16	19	- 3	9
3	6	8	- 2	4	12	13	- 1	1
4	6	4	2	4	8	13	- 5	25
5	8	2	4	16	7	11	- 4	16
6	21	6	15	225	19	6	13	169
7	23	3	20	400	23	3	20	400
8	22	1	21	441	20	1	19	361
9	19	5	14	196	20	8	12	144
10	19	11	8	64	17	8	9	81
11	14	12	2	4	18	10	8	64
12	15	18	- 3	9	14	15	- 1	1
L1	18	17	1	1	13	12	1	1
2	10	14	- 4	16	8	4	4	16
3	4	8	- 4	16	5	2	3	9
4	1	10	- 9	81	1	5	- 4	16
5	2	7	- 5	25	10	6	4	16
			$\Sigma di^2 = 2412$				$\Sigma di^2 = 2531$	

$$r_s = 1 - \frac{6(2412)}{(23)^3 - 23}$$

$$= -0{\cdot}192$$

not significantly correlated at $p < 0{\cdot}01$

$$r_s = 1 - \frac{6(2531)}{(23)^3 - 23}$$

$$= -0{\cdot}201$$

not significantly correlated at $p < 0{\cdot}01$

Table 13 Comparison of Involvement of Superior and Inferior Portions of the Joints in Osteoarthrosis.

	Females		Males	
Joint	% Superior	% Inferior	% Superior	% Inferior
C1	4	5	5	5
2	25	23	36	35
3	34	28	42	37
4	27	21	37	28
5	16	12	24	18
6	15	11	19	11
7	21	15	35	28
T1	18	7	16	11
2	23	21	21	16
3	26	26	29	26
4	28	28	33	34
5	16	19	23	30
6	8	8	16	21
7	7	6	12	12
8	10	7	13	13
9	12	6	13	12
10	10	6	18	9
11	16	6	18	9
12	12	13	19	16
L1	11	13	16	21
2	4	20	21	31
3	8	28	22	37
4	8	41	33	48
5	2	31	28	25

Table 14 Comparison of Involvement of Superior and Inferior Portions of the Joint in Osteophytosis.

	Females		Males	
Joint	% Superior	% Inferior	% Superior	% Inferior
C2	10	9	12	14
3	12	13	29	31
4	17	31	26	49
5	33	35	47	48
6	33	33	49	44
7	13	20	20	23
T1	19	16	16	16
2	29	36	33	41
3	53	47	52	55
4	56	59	56	59
5	63	58	60	53
6	58	59	67	62
7	57	60	65	67
8	61	62	68	61
9	58	51	60	57
10	39	45	48	52
11	34	36	46	49
12	18	24	41	39
L1	17	31	47	54
2	25	39	46	62
3	25	52	45	65
4	29	47	43	64
5	44	54	53	60

Table 15 Comparison of Involvement of Right and Left Side of the Joint in Osteoarthrosis.

	Females		Males	
Joint	% Right	% Left	% Right	% Left
C1	4	5	5	4
2	23	25	30	36
3	23	34	36	42
4	25	27	36	37
5	16	15	24	24
6	12	15	19	19
7	21	21	34	35
T1	18	10	16	11
2	23	14	21	17
3	26	10	29	17
4	28	13	34	24
5	19	11	30	22
6	8	8	14	21
7	6	7	8	12
8	6	10	12	13
9	11	12	13	9
10	10	10	15	18
11	16	11	18	12
12	12	13	19	16
L1	11	13	20	21
2	16	20	31	30
3	27	28	33	37
4	41	38	46	48
5	33	30	28	24

Table 16 Comparison of Involvement of Right and Left Side of the Joint in Osteophytosis.

	Females		Males	
Joint	% Right	% Left	% Right	% Left
C2	10	9	14	14
3	13	13	31	29
4	31	28	49	42
5	35	34	48	47
6	33	33	44	49
7	12	20	21	23
T1	16	15	13	16
2	23	19	29	23
3	28	26	36	31
4	34	21	40	19
5	36	22	40	23
6	37	28	41	33
7	41	27	51	29
8	46	22	62	35
9	37	20	60	39
10	37	21	51	37
11	30	22	47	46
12	23	20	40	41
L1	31	31	51	54
2	36	39	60	62
3	52	45	63	65
4	47	47	62	64
5	54	50	60	58

Table 17 χ^2 and p Values for Sex Differences in Involvement of Facet and Disc Joints throughout the Spine.

Joint	Facet		Disc	
	χ^2	p	χ^2	p
C1	1·55681	0·5463	1·18906	0·0213
2	1·69544	0·0030	0·0	0·0759
3	1·83140	0·0450	3·27919	0·0002
4	2·31285	0·0306	4·62189	0·0005
5	1·12712	0·0592	2·08046	0·0185
6	1·80133	0·0297	3·77519	0·0047
7	9·78829	0·0001	0·31106	0·0922
T1	0·29154	0·7174	0·02855	1·0000
2	0·04109	0·9205	0·02995	0·6051
3	0·17969	0·6344	0·25543	0·5006
4	1·72210	0·0544	0·40975	0·7019
5	2·70625	0·0042	2·12508	0·4505
6	1·44166	0·0206	0·81780	0·1829
7	0·19392	0·0466	0·24914	0·2868
8	0·12816	0·0165	0·0	0·4824
9	0·00106	0·2700	0·00068	0·6540
10	0·90716	0·0082	1·87250	0·0111
11	0·28535	0·8973	2·48378	0·0010
12	0·59315	0·0370	5·66329	0·0000
L1	2·34249	0·0021	5·08278	0·0000
2	3·71944	0·0073	10·86326	0·0000
3	1·40061	0·0907	5·28054	0·0024
4	0·07089	0·6954	9·49770	0·0003
5	3·44957	0·0578	1·05906	0·0728

Table 18 χ^2 and p Values for Sex Differences in Severity of Involvement of Facet and Disc Joints throughout the Spine.

Joints	Facet		Disc	
	χ^2	p	χ^2	p
C1	4·9253	0·1774	6·1816	0·1031
2	9·7459	0·0449	4·4170	0·3525
3	5·2173	0·2657	15·4003	0·0039
4	11·1994	0·0244	14·4599	0·0060
5	4·4321	0·2184	9·9787	0·0408
6	6·7691	0·1486	12·6691	0·0130
7	19·5289	0·0006	4·8873	0·2991
T1	0·4481	0·7993	0·7353	0·8649
2	3·1681	0·5301	2·4995	0·4754
3	1·5504	0·8177	7·8498	0·0972
4	6·5108	0·1641	13·0384	0·0111
5	10·9366	0·0273	13·5147	0·0090
6	10·4601	0·0150	8·5492	0·0734
7	6·7424	0·1501	11·4641	0·0218
8	7·6945	0·1034	16·9064	0·0020
9	5·6354	0·2281	20·7847	0·0003
10	10·0463	0·0397	18·4309	0·0010
11	6·6878	0·1533	28·8439	0·0000
12	4·8022	0·0906	23·8321	0·0000
L1	11·3340	0·0231	33·9506	0·0000
2	9·7897	0·0441	39·1190	0·0000
3	4·8313	0·1846	26·3320	0·0000
4	2·7466	0·4324	18·7411	0·0003
5	6·3755	0·1728	5·1881	0·2685

Table 19 χ^2 and p Values for Sex Differences in Maximum Severity Spine Measures of Facet and Disc Joints.

Joint	Spine Score	χ^2	p
Facets	Column	6·64897	0·1556
	Cervical	8·96739	0·0619
	Thoracic	7·75598	0·1009
	Lumbar	1·17214	0·8827
Disc	Column	30·38255	0·0000
	Cervical	16·01697	0·0030
	Thoracic	25·78744	0·0000
	Lumbar	32·90105	0·0000

Table 20 U and p Values for Sex Differences in Combined Maximum Severity Spine Measures.

Spine Score	U	p
Column	22973·5	0·0011
Cervical	19389·0	0·0016
Thoracic	24304·5	0·0079
Lumbar	21953·0	0·0037

Table 21 U and p Values for Sex Differences in Extent (%) Spine Measures of Facet and Disc Joints and Combined.

Joint	Spine Score	U	p
Facets	Column	33697·0	0·0273
	Cervical	24775·0	0·0014
	Thoracic	34821·0	0·1066
	Lumbar	38602·0	0·2391
Discs	Column	23112·5	0·0003
	Cervical	18771·5	0·0000
	Thoracic	26479·0	0·0921
	Lumbar	20943·5	0·0000
Combined	Column	22677·5	0·0006
	Cervical	18337·0	0·0001
	Thoracic	25283·0	0·0509
	Lumbar	21753·0	0·0026

Table 22 U and p Values for Sex Differences in Extent (%) x Maximum Severity Spine Measures of Facet and Disc Joints and Combined.

Joint	Spine Score	U	p
Facets	Column	34230·5	0·0547
	Cervical	25120·5	0·0031
	Thoracic	35263·5	0·1699
	Lumbar	38080·0	0·3562
Discs	Column	22104·0	0·0000
	Cervical	18975·5	0·0001
	Thoracic	25038·5	0·0087
	Lumbar	19480·5	0·0000
Combined	Column	22182·0	0·0002
	Cervical	18884·0	0·0005
	Thoracic	24105·5	0·0061
	Lumbar	20651·5	0·0002

Table 23 Correlation between Involvement of Facet and Disc Joints with Dental Age.

Joint	Facets (coefficient/p)				Discs (coefficient/p)			
	Females		Males		Females		Males	
C1	·1714	·014	·2628	·000	·4210	·000	·5207	·000
2	·4863	·000	·5456	·000	·2804	·001	·4468	·000
3	·5075	·000	·6413	·000	·3871	·000	·4008	·000
4	·4857	·000	·5638	·000	·4339	·000	·4119	·000
5	·4000	·000	·4749	·000	·4762	·000	·4917	·000
6	·4319	·000	·5383	·000	·4493	·000	·4002	·000
7	·3733	·000	·4662	·000	·4143	·000	·3993	·000
T1	·2187	·002	·3578	·000	·3055	·000	·4680	·000
2	·2390	·001	·3248	·000	·2021	·012	·2991	·001
3	·2316	·001	·4452	·000	·3775	·000	·4769	·000
4	·3012	·000	·3753	·000	·3689	·000	·4928	·000
5	·2720	·000	·4080	·000	·3844	·000	·3854	·000
6	·2424	·001	·3036	·000	·4615	·000	·4298	·000
7	·1500	·028	·1897	·009	·3726	·000	·4590	·000
8	·2042	·004	·3913	·000	·3243	·000	·5080	·000
9	·2082	·004	·2481	·001	·3000	·000	·3376	·000
10	·2165	·003	·3299	·000	·2616	·002	·4491	·000
11	·2505	·001	·3045	·000	·3499	·000	·4529	·000
12	·3829	·000	·3559	·000	·4327	·000	·2785	·000
L1	·3780	·000	·3750	·000	·2593	·003	·4973	·000
2	·4589	·000	·5014	·000	·4757	·000	·4952	·000
3	·4433	·000	·5327	·000	·6237	·000	·5867	·000
4	·5325	·000	·4762	·000	·6207	·000	·5974	·000
5	·4675	·000	·4443	·000	·5920	·000	·5461	·000

Table 24 Correlation Between Severity of Involvement of Facet and Disc Joints with Dental Age.

Joint	Facets (coefficient/p)				Discs (coefficient/p)			
	Females		Males		Females		Males	
C1	·1738	·013	·2637	·000	·4420	·000	·5481	·000
2	·4946	·000	·5775	·000	·2749	·001	·4548	·000
3	·5264	·000	·6752	·000	·3942	·000	·4259	·000
4	·4953	·000	·5816	·000	·4280	·000	·4382	·000
5	·4018	·000	·4739	·000	·4915	·000	·5021	·000
6	·4359	·000	·5405	·000	·4538	·000	·4318	·000
7	·3863	·000	·5034	·000	·4117	·000	·3928	·000
T1	·2275	·001	·3657	·000	·3026	·001	·4664	·000
2	·2522	·000	·3401	·000	·2116	·009	·3115	·000
3	·2604	·000	·4402	·000	·3851	·000	·4480	·000
4	·3202	·000	·3979	·000	·3679	·000	·5151	·000
5	·2925	·000	·4490	·000	·3887	·000	·4193	·000
6	·2482	·001	·3140	·000	·4747	·000	·4472	·000
7	·1530	·026	·1948	·008	·4280	·000	·5134	·000
8	·2064	·004	·3912	·000	·3691	·000	·4623	·000
9	·2134	·003	·2574	·001	·3364	·000	·3113	·000
10	·2239	·002	·3340	·000	·2624	·002	·4896	·000
11	·2605	·000	·3157	·000	·3587	·000	·4547	·000
12	·3978	·000	·3585	·000	·4340	·000	·2906	·001
L1	·3797	·000	·3695	·000	·2564	·003	·5478	·000
2	·4723	·000	·4907	·000	·4806	·000	·5160	·000
3	·4530	·000	·5339	·000	·6386	·000	·5886	·000
4	·5524	·000	·5131	·000	·6171	·000	·6176	·000
5	·5288	·000	·4326	·000	·6298	·000	·5229	·000

Table 25 % Fequency by sex and dental age of facet and disc involvement of the regions and the whole column.

Region/ Column	Joint type	Sex	20 - 25	25 - 35	35 - 45	45+
Column	facet	female	60•7	87•3	100	100
		male	51•5	79•7	98•1	96•7
	disc	female	67•3	91•8	96•8	100•0
		male	63•0	84•3	100•0	100•0
Cervical	facet	female	23•2	55•9	82•8	91•4
		male	14•8	58•9	86•3	93•3
	disc	female	12•8	44•9	89•3	87•1
		male	21•7	58•3	88•5	89•7
Thoracic	facet	female	48•4	68•3	86•7	88•9
		male	41•2	64•5	90•7	95•1
	disc	female	65•3	77•8	86•7	100•0
		male	48•3	72•5	98•0	100•0
Lumbar	facet	female	27•7	64•2	100•	95•1
		male	33•3	50•8	87•0	92•9
	disc	female	27•8	71•4	96•4	100•0
		male	46•2	70•8	97•9	96•4

Table 26 U and p Values for Sex Differences of Maximum Severity Spine Measures in each Dental Age Group.

Spine Score	Dental Age (U/p)			
	20 - 25	25 - 35	35 - 45	45+
Facets Column	943·0 0·5714	1750·5 0·1504	829·0 0·9368	451·5 0·1617
Cervical	690·5 0·3631	1616·5 0·8268	707·5 0·7336	375·5 0·0345
Thoracic	986·0 0·5495	1712·5 0·1878	762·0 0·6231	478·5 0·5364
Lumbar	1005·0 0·5241	1864·5 0·2121	735·5 0·2146	556·0 0·8126
Discs Column	642·0 0·8119	1138·5 0·4172	659·0 0·2159	392·0 0·0870
Cervical	493·0 0·3476	1046·5 0·3082	697·0 0·7366	350·5 0·1270
Thoracic	607·5 0·2264	1329·0 0·7379	599·0 0·1139	388·0 0·3488
Lumbar	582·0 0·1381	1036·0 0·2849	477·0 0·0288	411·5 0·1789
Combined Column	581·0 0·4366	1241·0 0·9517	636·0 0·2384	402·0 0·1890
Cervical	535·0 0·9345	1074·0 0·5518	691·0 0·9230	322·5 0·0552
Thoracic	594·0 0·2582	1371·0 0·9683	574·5 0·1155	343·0 0·1582
Lumbar	621·5 0·3658	1148·5 0·8383	621·0 0·6734	393·0 0·3866

Table 27 % Frequency by Dental Age of the Maximum Grade of Osteophytosis in each Region and the Whole Column.

Region/ Column	Sex	Max grade	20 - 25	25 - 35	35 - 45	45+
Column	female	0	32·7	8·2	3·2	0
		1	57·1	53·1	16·1	14·7
		2	10·2	28·6	54·8	61·8
		3	0	6·1	12·9	20·6
		4	0	4·1	12·9	2·9
	male	0	37·0	15·7	0	0
		1	51·9	35·3	10·0	16·7
		2	7·4	27·5	54·0	36·7
		3	0	5·9	20·0	20·0
		4	3·7	15·7	16·0	26·7
Cervical	female	0	87·2	55·1	10·7	12·9
		1	8·5	28·6	32·1	38·7
		2	4·3	12·2	39·3	35·5
		3	0	2·0	10·7	12·9
		4	0	2·0	7·1	0
	male	0	78·3	41·7	11·5	10·3
		1	17·4	41·7	28·8	27·6
		2	0	14·6	51·9	31·0
		3	0	2·1	3·8	17·2
		4	4·3	0	3·8	13·8
Thoracic	female	0	34·7	22·2	13·3	0
		1	61·2	57·4	36·7	51·5
		2	4·1	14·8	40·0	42·4
		3	0	3·7	3·3	3·0
		4	0	1·9	6·7	3·0
	male	0	51·7	27·5	2·0	0
		1	41·4	45·1	40·0	48·1
		2	6·9	11·8	30·0	25·9
		3	0	2·0	16·0	11·1
		4	0	13·7	12·0	14·8
Lumbar	female	0	72·2	28·6	3·6	0
		1	20·4	49·0	46·4	41·7
		2	7·4	20·4	39·3	44·4
		3	0	2·0	3·6	13·9
		4	0	0	7·1	0
	male	0	53·8	29·2	2·1	3·6
		1	38·5	33·3	19·1	25·0
		2	7·7	27·1	61·7	42·9
		3	0	8·3	14·9	28·6
		4	0	2·1	2·1	0

Table 28 % Frequency by sex and dental age of the maximum grade of osteoarthrosis in each region and the whole column.

Region/ Column	Sex	Max grade	20 - 25	25 - 35	35 - 45	45+
Column	female	0	39·3	12·7	0	0
		1	55·7	57·1	32·3	13·5
		2	3·3	22·2	45·2	64·9
		3	1·6	4·8	3·2	18·9
		4	0	3·2	19·4	2·7
	male	0	48·5	20·3	1·9	3·3
		1	42·4	59·4	24·1	20·0
		2	9·1	12·5	53·7	26·7
		3	0	4·7	11·1	33·3
		4	0	3·1	9·3	16·7
Cervical	female	0	76·8	44·1	17·2	8·6
		1	21·4	40·7	44·8	25·7
		2	0	10·2	24·1	60·0
		3	1·8	1·7	0	5·7
		4	0	3·4	13·8	0
	male	0	85·2	41·1	13·7	6·7
		1	14·8	51·8	41·2	20·0
		2	0	5·4	37·3	36·7
		3	0	1·8	3·9	20·0
		4	0	0	3·9	16·7
Thoracic	female	0	51·6	31·7	13·3	11·1
		1	46·8	47·6	50·0	44·4
		2	1·6	20·6	33·3	41·7
		3	0	0	0	0
		4	0	0	3·3	2·8
	male	0	58·8	35·5	9·3	10·3
		1	38·2	58·1	51·9	41·4
		2	2·9	4·8	31·5	34·5
		3	0	0	0	10·3
		4	0	1·6	7·4	3·4
Lumbar	female	0	72·3	35·8	0	4·9
		1	24·6	49·3	50·0	31·7
		2	3·1	10·4	37·5	46·3
		3	0	4·5	9·4	17·1
		4	0	0	3·1	0
	male	0	66·7	49·2	13·0	7·1
		1	27·3	36·5	44·4	25·0
		2	6·1	9·5	33·3	50·0
		3	0	3·2	9·3	17·9
		4	0	1·6	0	0

Table 29 U and p Values for Sex Differences of Extent (%) Spine Measures in each Dental Age Group.

Spine Score	Dental Age (U/p)			
	20 - 25	25 - 35	35 - 45	45+
Facets Column	930·5 0·5304	1743·5 0·1877	572·5 0·0157	442·5 0·1559
Cervical	681·5 0·3038	1616·5 0·8350	526·0 0·0313	374·5 0·0451
Thoracic	979·5 0·5322	1731·5 0·2637	575·5 0·0283	438·5 0·2692
Lumbar	1011·0 0·5657	1887·0 0·2775	705·0 0·1510	511·0 0·4260
Discs Column	595·5 0·4647	1248·0 0·9917	559·5 0·0361	364·5 0·0499
Cervical	491·5 0·3328	1015·0 0·2165	671·0 0·5616	335·5 0·0866
Thoracic	550·0 0·0853	1333·0 0·7759	587·5 0·1046	386·5 0·3746
Lumbar	563·0 0·0897	1157·5 0·8915	412·0 0·0040	314·5 0·0028
Combined Column	553·5 0·2956	1199·5 0·7303	445·0 0·0024	356·5 0·0566
Cervical	535·5 0·9410	1049·5 0·4491	556·5 0·1349	310·5 0·0396
Thoracic	521·0 0·0630	1294·0 0·5941	483·0 0·0138	360·5 0·276
Lumbar	613·5 0·3316	1022·0 0·2645	454·5 0·0252	268·5 0·0069

Table 30 % Frequency of Extent (%) Spine Measures in each Dental Age Group.

Facets Column	Dental Age 20 - 25		25 - 35		35 - 45		45+	
	Female	Male	Female	Male	Female	Male	Female	Male
0	39	48	13	20	0	2	0	3
10	34	27	19	22	3	2	3	3
20	11	6	17	14	16	9	3	3
30	7	12	13	17	29	7	8	17
40	3	6	17	9	13	13	27	0
50	3	0	10	6	13	22	8	7
60	0	0	5	6	13	20	19	7
70	2	0	2	5	6	7	11	20
80	0	0	3	0	3	11	14	17
90	0	0	0	0	3	4	8	10
100	0	0	2	0	0	2	0	13
Cervical								
0	77	85	44	41	17	14	9	7
10	0	0	0	0	0	0	0	0
20	11	15	22	27	14	2	6	7
30	2	0	5	11	21	12	6	10
40	4	0	3	2	3	2	3	0
50	5	0	10	9	10	14	14	7
60	0	0	7	9	14	18	9	7
70	2	0	0	0	3	2	6	0
80	0	0	5	2	7	10	34	13
90	0	0	2	0	7	22	14	40
100	0	0	2	0	3	6	0	10
Thoracic								
0	52	59	32	35	13	9	11	10
10	23	15	11	21	13	4	14	14
20	11	15	16	13	23	15	11	3
30	2	6	14	10	10	15	3	3
40	5	0	10	5	10	13	25	3
50	3	6	8	11	17	9	8	21
60	3	0	2	5	7	13	3	14
70	0	0	3	0	0	6	14	7
80	2	0	0	0	3	11	6	3
90	0	0	2	0	3	6	6	7
100	0	0	3	0	0	0	3	14

Table 30 contd.

	Dental Age							
Facets	20 - 25		25 - 35		35 - 45		45+	
Lumbar	Female	Male	Female	Male	Female	Male	Female	Male
0	68	67	36	49	0	13	5	7
10	0	0	0	0	0	0	0	0
20	14	15	15	8	12	6	5	4
30	0	0	1	0	3	0	2	0
40	6	9	12	11	28	11	7	4
50	2	6	3	5	12	6	2	0
60	3	0	12	8	22	17	20	25
70	0	0	1	3	0	4	2	0
80	3	3	7	6	9	24	24	18
90	0	0	0	0	0	0	0	0
100	0	0	12	10	12	20	32	43
Discs								
Column								
0	33	37	8	16	3	0	0	0
10	10	22	4	10	0	2	0	3
20	18	15	27	6	3	0	0	3
30	14	4	8	8	13	8	0	3
40	12	11	4	14	0	4	12	10
50	8	4	12	10	23	2	9	7
60	2	4	4	4	6	18	15	3
70	2	4	14	12	16	16	24	3
80	0	0	6	6	16	14	21	7
90	0	0	6	14	10	12	9	13
100	0	0	6	2	10	24	12	47
Cervical								
0	87	78	55	42	11	12	13	10
10	0	0	0	0	0	0	0	0
20	9	13	6	8	4	15	23	10
30	0	4	2	2	4	2	0	0
40	2	0	12	17	21	13	6	3
50	0	4	8	8	18	10	23	7
60	0	0	6	2	0	2	0	3
70	0	0	4	12	14	6	6	14
80	2	0	2	4	14	2	0	7
90	0	0	0	2	4	10	6	10
100	0	0	4	2	11	29	23	34

Table 30 contd.

Discs	Dental Age 20 - 25		25 - 35		35 - 45		45+	
Thoracic	Female	Male	Female	Male	Female	Male	Female	Male
0	35	52	22	29	13	2	0	0
10	4	3	2	2	0	2	0	0
20	4	14	11	6	7	6	0	4
30	4	0	9	10	3	4	0	11
40	18	7	6	2	10	4	0	11
50	16	17	7	17	7	4	24	4
60	4	3	4	0	7	6	6	4
70	8	3	9	6	13	18	12	4
80	4	0	13	4	7	12	27	4
90	0	0	4	8	13	6	9	7
100	2	0	13	15	20	36	21	52
Lumbar								
0	72	54	29	29	4	2	0	4
10	0	0	0	0	0	0	0	0
20	9	12	6	12	7	0	0	0
30	6	4	2	2	0	0	3	0
40	4	19	14	6	11	4	14	4
50	2	0	10	10	11	2	14	0
60	2	4	4	8	11	13	6	4
70	0	0	4	0	11	2	3	0
80	2	8	6	6	18	15	14	4
90	0	0	0	0	0	0	0	0
100	4	0	24	25	29	62	47	86
Combined Column								
0	17	19	0	6	0	0	0	0
20	33	52	14	14	0	0	0	3
40	29	7	24	18	3	0	0	0
60	10	7	12	16	0	6	0	7
80	10	15	12	16	27	8	12	13
100	0	0	12	12	27	14	21	10
120	0	0	10	8	23	24	24	3
140	0	0	10	4	10	18	18	3
160	0	0	2	8	7	16	15	27
180	0	0	0	0	3	10	9	17
200	0	0	2	0	0	4	0	17

Table 30 contd.

	Dental Age							
	20 - 25		25 - 35		35 - 45		45+	
	Female	Male	Female	Male	Female	Male	Female	Male
Combined Cervical								
0	66	65	27	17	4	2	0	7
20	17	22	19	23	0	0	3	0
40	9	9	10	10	11	8	0	3
60	4	4	10	15	7	18	19	7
80	4	0	10	10	21	8	13	3
100	0	0	8	4	18	10	10	3
120	0	0	8	15	14	6	10	3
140	0	0	2	6	14	14	16	17
160	0	0	2	0	4	12	13	14
180	0	0	0	0	7	6	16	21
200	0	0	2	0	0	16	0	21
Thoracic								
0	23	34	9	14	3	0	0	0
20	15	28	11	8	0	2	0	4
40	19	7	15	18	3	4	0	4
60	15	14	15	12	14	4	12	7
80	19	17	13	16	17	14	6	15
100	8	0	9	12	34	16	22	7
120	2	0	11	10	10	20	9	4
140	0	0	11	8	3	12	31	15
160	0	0	2	4	14	20	9	22
180	0	0	0	0	0	8	9	11
200	0	0	4	0	0	0	0	11
Lumbar								
0	52	42	14	21	0	0	0	0
20	19	19	4	6	0	0	0	4
40	13	19	8	12	4	0	0	0
60	7	0	16	17	7	2	3	0
80	2	4	10	6	0	9	0	0
100	4	0	12	12	21	15	11	0
120	0	12	14	6	25	11	14	0
140	0	4	4	10	14	4	17	16
160	0	0	4	4	18	26	19	24
180	4	0	12	4	4	17	19	12
200	0	0	0	4	7	17	17	44

Table 31 Means, Standard Deviations and Coefficients of Variation of Extent (%) Spine Measures in each Dental Age Group.

		Females				Males			
Spine Score	Statistic	20 - 25	25 - 35	35 - 45	45+	20 - 25	25 - 35	35 - 45	45+
Facet Column	mean	10	25	36	50	8	20	48	58
	S.D.	14	21	20	19	11	19	21	28
	C. of V.	1.479	.858	.551	.387	1.383	.947	.439	.478
Cervical	mean	7	21	38	55	2	18	53	64
	S.D.	15	26	29	26	5	20	30	31
	C. of V.	2.178	1.236	.748	.472	2.450	1.150	.572	.480
Thoracic	mean	10	22	28	38	8	16	40	47
	S.D.	17	24	23	27	12	18	25	32
	C. of V.	1.608	1.117	.813	.713	1.532	1.114	.635	.677
Lumbar	mean	10	35	54	70	12	30	60	74
	S.D.	20	35	24	29	20	35	32	30
	C. of V.	1.929	.994	.447	.433	1.685	1.178	.536	.407
Disc Column	mean	17	40	57	66	15	41	70	74
	S.D.	17	30	25	18	19	31	23	29
	C. of V.	.985	.741	.443	.279	1.262	.751	.330	.391
Cervical	mean	4	22	52	50	6	28	56	66
	S.D.	13	29	30	36	13	30	37	35
	C. of V.	3.359	1.325	.571	.718	2.228	1.044	.623	.528
Thoracic	mean	29	44	57	72	17	43	70	74
	S.D.	27	35	34	19	22	36	28	31
	C. of V.	.933	.795	.594	.262	1.293	.834	.408	.419

Table 31 contd.

Spine Score	Statistic	Females 20 - 25	Females 25 - 35	Females 35 - 45	Females 45+	Males 20 - 25	Males 25 - 35	Males 35 - 45	Males 45+
Disc Lumbar	mean	12	46	67	76	19	45	85	92
	S.D.	24	39	28	26	25	40	23	23
	C. of V.	2·074	·838	·420	·343	1·309	·872	·270	·246
Combined Column	mean	26	66	96	116	22	62	119	132
	S.D.	21	45	31	29	25	42	34	50
	C. of V.	·832	·677	·318	·248	1·147	·682	·282	·379
Cervical	mean	10	46	90	107	8	48	111	132
	S.D.	18	48	43	48	14	42	56	58
	C. of V.	1·774	1·049	·481	·447	1·662	·877	·509	·443
Thoracic	mean	38	67	88	111	23	60	110	120
	S.D.	33	52	36	34	25	44	39	53
	C. of V.	·855	·757	·416	·310	1·079	·737	·359	·441
Lumbar	mean	24	82	123	149	32	71	145	171
	S.D.	39	56	40	38	42	59	41	39
	C. of V.	1·666	·674	·327	·254	1·312	·831	·283	·229

Table 32 U and p Values for Sex Differences of Extent (%) x Maximum Severity Spine Measures in each Dental Age Group.

Spine Score	Dental Age (U/p)			
	20 - 25	25 - 35	35 - 45	45+
Facets Column	934·0 0·5496	1723·5 0·1574	643·0 0·0765	442·0 0·1541
Cervical	681·5 0·3038	1611·0 0·8101	593·0 0·1409	363·5 0·0327
Thoracic	982·0 0·5461	1678·0 0·1655	629·0 0·0907	455·0 0·3757
Lumbar	1008·5 0·5502	1905·0 0·3186	823·0 0·7131	508·5 0·4198
Disc Column	592·5 0·4446	1162·0 0·5459	566·5 0·0427	367·5 0·0551
Cervical	492·5 0·3430	1030·0 0·2627	726·0 0·9839	320·0 0·0545
Thoracic	565·5 0·1201	1351·0 0·8665	567·5 0·0695	413·5 0·6337
Lumbar	565·5 0·0957	1111·0 0·6342	387·5 0·0027	334·5 0·0191
Combined Column	551·0 0·2830	1236·5 0·9286	527·0 0·0267	357·0 0·0575
Cervical	536·0 0·9469	1066·0 0·5254	657·5 0·6579	296·0 0·0231
Thoracic	545·5 0·1098	1350·5 0·8649	524·5 0·0414	372·5 0·3652
Lumbar	623·0 0·3865	1096·0 0·5624	456·5 0·0272	323·5 0·0629

Table 33 . Frequency Distributions of the Extent (.) x Maximum Severity Spine Measures in each Dental Age Group.

Facets Column	Dental Age 20 - 25		25 - 35		35 - 45		45+	
	Female	Male	Female	Male	Female	Male	Female	Male
0	39	48	13	20	0	2	0	3
40	52	42	52	56	29	17	14	10
80	7	9	19	12	35	20	24	20
120	2	0	8	3	13	30	19	10
160	0	0	3	5	3	20	19	10
200	0	0	2	2	10	2	16	17
240	0	0	2	2	3	2	5	13
280	0	0	0	0	3	6	3	3
320	0	0	0	0	3	0	0	0
360	0	0	0	0	0	2	0	7
400	0	0	2	0	0	0	0	7
Cervical								
0	77	85	44	41	17	14	9	7
40	16	15	29	39	35	11	8	16
80	5	0	12	15	10	28	20	0
120	0	0	8	1	14	18	17	17
160	0	0	2	4	10	5	32	10
200	2	0	2	0	4	16	8	17
240	0	0	0	0	7	0	3	0
280	0	0	0	0	0	6	3	16
320	0	0	1	0	0	0	0	7
360	0	0	0	0	3	0	0	3
400	0	0	2	0	0	2	0	7
Thoracic								
0	52	59	32	35	13	9	11	10
40	38	35	46	49	50	41	39	21
80	10	3	11	11	17	17	17	31
120	0	3	6	5	13	11	5	14
160	0	0	2	0	0	13	17	0
200	0	0	3	0	4	3	8	17
240	0	0	0	0	0	2	0	0
280	0	0	0	0	0	2	3	0
320	0	0	0	0	3	0	0	4
360	0	0	0	0	0	2	0	0
400	0	0	0	0	0	0	0	3

Table 33 contd.

	Dental Age							
Facets	20 - 25		25 - 35		35 - 45		45+	
Lumbar	Female	Male	Female	Male	Female	Male	Female	Male
0	72	67	36	49	0	13	5	7
40	20	21	28	18	31	17	10	4
80	6	9	17	17	32	24	26	14
120	0	0	7	5	15	13	15	21
160	2	3	2	3	10	13	12	11
200	0	0	9	3	6	11	20	32
240	0	0	0	2	0	3	0	4
280	0	0	0	0	0	0	0	0
320	0	0	1	3	3	6	12	7
360	0	0	0	0	0	0	0	0
400	0	0	0	0	3	0	0	0
Discs								
Column								
0	33	37	9	16	3	0	0	0
40	51	48	37	25	10	8	9	17
80	10	7	24	20	13	8	9	7
120	4	4	8	8	19	14	21	7
160	2	0	8	12	26	22	26	10
200	0	4	4	2	10	18	18	13
240	0	0	2	0	3	2	9	0
280	0	0	4	4	6	6	3	10
320	0	0	0	6	6	12	6	10
360	0	0	0	8	3	0	0	3
400	0	0	4	0	0	10	0	23
Cervical								
0	87	78	55	42	11	12	13	10
40	11	18	14	25	21	20	26	11
80	0	0	23	14	14	19	13	20
120	0	0	4	9	11	12	19	4
160	2	0	0	8	22	5	13	7
200	0	4	0	0	7	25	16	17
240	0	0	2	2	3	0	0	7
280	0	0	0	0	4	0	0	0
320	0	0	0	0	7	4	10	14
360	0	0	0	0	0	0	0	0
400	0	0	2	0	0	4	0	10

Table 33 contd.

Discs	Dental Age 20 - 25		25 - 35		35 - 45		45+	
Thoracic	Female	Male	Female	Male	Female	Male	Female	Male
0	35	52	22	27	13	2	0	0
40	30	24	26	20	17	16	0	22
80	29	17	26	20	23	20	45	15
120	2	7	11	9	4	8	16	22
160	4	0	6	2	10	12	15	0
200	0	0	3	8	26	14	21	15
240	0	0	0	0	0	2	0	0
280	0	0	0	0	0	12	0	7
320	0	0	4	6	4	4	0	4
360	0	0	0	2	0	0	0	0
400	0	0	2	6	3	10	3	15
Lumbar								
0	72	54	29	29	4	2	0	4
40	19	31	18	19	14	4	6	4
80	4	12	24	15	29	9	11	7
120	0	0	12	10	18	15	25	14
160	2	4	2	4	11	15	11	0
200	4	0	12	12	18	38	22	43
240	0	0	0	2	0	0	4	0
280	0	0	0	0	0	0	0	0
320	0	0	2	8	4	15	11	29
360	0	0	0	0	0	0	0	0
400	0	0	0	0	4	2	0	0
Combined Column								
0	17	19	0	6	0	0	0	0
80	75	70	53	43	10	6	3	10
160	8	7	14	29	30	10	12	10
240	0	4	20	2	23	22	30	13
320	0	0	4	2	20	30	21	10
400	0	0	2	10	7	14	24	10
480	0	0	4	8	3	8	6	20
560	0	0	0	0	3	4	3	3
640	0	0	0	0	3	2	0	10
720	0	0	0	0	0	2	0	0
800	0	0	0	0	0	2	0	13

Table 33 contd.

Combined Cervical	Dental Age 20 - 25		25 - 35		35 - 45		45+	
	Female	Male	Female	Male	Female	Male	Female	Male
0	66	65	27	17	4	2	0	7
80	30	31	42	56	25	26	23	7
160	2	0	21	17	25	22	19	10
240	2	4	6	8	17	16	23	14
320	0	0	0	0	11	18	12	17
400	0	0	0	2	11	6	10	11
480	0	0	0	0	0	2	13	10
560	0	0	2	0	3	4	0	3
640	0	0	0	0	4	0	0	7
720	0	0	0	0	0	2	0	11
800	0	0	2	0	0	2	0	3
Thoracic								
0	23	34	9	14	3	0	0	0
80	62	56	47	45	21	18	9	19
160	15	10	22	17	28	20	32	25
240	0	0	13	10	31	24	37	12
320	0	0	3	0	7	12	13	7
400	0	0	2	8	3	14	6	18
480	0	0	2	6	4	8	3	12
560	0	0	2	0	3	0	0	3
640	0	0	0	0	0	4	0	0
720	0	0	0	0	0	0	0	0
800	0	0	0	0	0	0	0	4
Lumbar								
0	54	42	14	21	0	0	0	0
80	38	42	37	35	11	4	3	4
160	2	8	20	12	21	19	17	12
240	2	8	12	15	36	17	22	4
320	2	0	12	6	21	30	28	28
400	0	0	4	4	3	15	17	28
480	0	0	0	2	0	4	3	12
560	0	0	0	0	5	9	8	4
640	0	0	0	4	0	2	3	8
720	0	0	0	0	0	0	0	0
800	0	0	0	0	3	0	0	0

Table 34 Means, Standard Deviations and Coefficients of Variation of Extent (%) x Maximum Severity Spine Measures in each Dental Age Group.

		Females				Males			
Spine Score	Statistic	20 - 25	25 - 35	35 - 45	45+	20 - 25	25 - 35	35 - 45	45+
Facet Column	mean	12	45	85	112	11	32	104	157
	S.D.	20	63	77	59	18	44	71	114
	C. of V.	1·749	1·409	·905	·532	1·742	1·368	·683	·730
Cervical	mean	9	37	76	104	2	22	95	164
	S.D.	29	71	84	64	5	32	84	116
	C. of V.	3·096	1·900	1·111	·618	2·205	1·472	·880	·712
Thoracic	mean	11	30	49	69	9	19	72	90
	S.D.	17	41	62	67	17	26	73	91
	C. of V.	1·608	1·354	1·280	·968	1·867	1·350	1·005	1·014
Lumbar	mean	12	51	98	134	16	46	103	145
	S.D.	25	67	85	86	33	72	83	80
	C. of V.	2·163	1·309	·861	·643	2·069	1·561	·803	·553
Disc Column	mean	22	77	135	144	24	98	180	213
	S.D.	28	92	89	70	45	109	102	136
	C. of V.	1·261	1·193	·661	·485	1·826	1·112	·568	·639
Cervical	mean	6	35	109	96	12	40	110	162
	S.D.	23	68	92	94	42	52	98	130
	C. of V.	4·029	1·937	·839	·977	3·394	1·290	·892	·803
Thoracic	mean	31	67	108	118	20	90	156	156
	S.D.	33	82	96	74	29	116	118	128
	C. of V.	1·053	1·215	·888	·628	1·439	1·301	·755	·821

Table 34 contd.

Spine Score	Statistic	Females 20 - 25	Females 25 - 35	Females 35 - 45	Females 45+	Males 20 - 25	Males 25 - 35	Males 35 - 45	Males 45+
Disc Lumbar	mean	17	67	120	140	24	86	175	192
	S.D.	44	73	91	85	37	97	84	90
	C. of V.	2·554	1·088	·760	·604	1·562	1·121	·479	·468
Combined Column	mean	32	126	225	260	34	133	289	370
	S.D.	34	146	133	105	52	141	145	225
	C. of V.	1·077	1·157	·590	·404	1·550	1·058	·504	·608
Cervical	mean	31	120	222	283	42	128	276	344
	S.D.	65	106	151	133	66	150	129	139
	C. of V.	2·440	1·789	·793	·655	2·798	1·129	·809	·672
Thoracic	mean	15	78	186	203	15	65	209	331
	S.D.	37	140	148	133	42	73	169	222
	C. of V.	·928	1·065	·731	·506	1·233	1·155	·643	·732
Lumbar	mean	41	100	162	184	28	111	230	244
	S.D.	38	107	118	93	35	128	148	179
	C. of V.	2·101	·888	·681	·469	1·585	1·170	·466	·405

Table 35 % Frequency by sex of the maximum grade of osteoarthrosis in each region and the whole column.

Region/column facet joints	max score	% affected of females	% affected of males
Column	0	14	13·7
	1	42·7	39·5
	2	31·5	27·3
	3	7·5	11·4
	4	4·3	8·1
Cervical	0	40·3	31·2
	1	32·8	34·2
	2	20·6	21·4
	3	3·6	7·3
	4	2·8	6·0
Thoracic	0	28·7	28·4
	1	48·2	46·6
	2	21·6	19·8
	3	0	1·9
	4	1·4	3·4
Lumbar	0	31·3	32·1
	1	40·8	37·3
	2	21·1	22·9
	3	6·5	7·0
	4	0·3	0·7

Table 36 % Frequency by sex of the maximum grade of osteophytosis in each region and the whole column.

Region/column disc joints	max score	% affected of females	% affected of males
Column	0	10·4	10·1
	1	38·3	23·1
	2	37·1	34·5
	3	10·0	15·5
	4	4·2	16·8
Cervical	0	40·3	31·2
	1	32·8	34·2
	2	20·6	21·4
	3	3·6	7·3
	4	2·8	6·0
Thoracic	0	28·7	28·4
	1	48·2	46·6
	2	21·6	19·8
	3	0	1·9
	4	1·4	3·4
Lumbar	0	31·3	32·1
	1	40·8	37·3
	2	21·1	22·9
	3	6·5	7·0
	4	0·3	0·7

Table 37 % Frequency by sex of the combined maximum grades of facet and disc joints in each region and the whole column.

Region/column	max score	% affected of females	% affected of males
Column	0	4	4
	1	12	11
	2	25	15
	3	19	17
	4	22	19
	5	10	12
	6	4	8
	7	2	5
	8	2	7
Cervical	0	25	15
	1	25	23
	2	14	14
	3	15	19
	4	13	13
	5	4	8
	6	2	3
	7	1	2
	8	1	4
Thoracic	0	9	8
	1	21	19
	2	32	22
	3	21	18
	4	14	17
	5	2	9
	6	2	2
	7	0	0
	8	0	3
Lumbar	0	18	12
	1	18	15
	2	21	18
	3	21	23
	4	15	18
	5	6	10
	6	2	4
	7	0	0
	8	1	1

Table 38 % Frequency Distributions of the Extent (%) Spine Measures in Groups of Ten Units (Twenty for Combined Scores).

	Facets. Column		Cervical		Thoracic		Lumbar	
%	Female	Male	Female	Male	Female	Male	Female	Male
0	14	14	40	31	29	28	31	32
10	16	14	0	0	14	12	0	0
20	12	13	15	12	16	12	13	11
30	16	11	6	9	9	9	2	1
40	15	7	5	2	11	6	12	9
50	9	9	11	8	9	12	4	3
60	8	9	5	9	4	7	14	12
70	5	8	2	1	4	4	1	2
80	4	8	10	8	2	5	12	13
90	1	2	5	15	2	3	0	0
100	1	3	2	5	1	3	12	17

	Discs Column		Cervical		Thoracic		Lumbar	
%	Female	Male	Female	Male	Female	Male	Female	Male
0	10	10	45	28	17	16	28	18
10	4	7	0	0	2	2	0	0
20	12	5	8	9	5	5	5	4
30	9	5	2	1	5	7	3	1
40	6	8	10	12	10	5	9	6
50	13	6	9	8	12	9	7	3
60	6	9	3	3	5	6	5	8
70	13	9	8	9	10	7	4	1
80	11	8	4	2	12	6	10	11
90	6	11	2	7	6	8	0	0
100	8	22	8	19	17	29	28	48

	Combined Column		Cervical		Thoracic		Lumbar	
%	Female	Male	Female	Male	Female	Male	Female	Male
0	4	4	25	15	9	8	18	12
20	11	12	11	10	6	8	6	5
40	16	7	9	8	11	9	6	6
60	8	8	10	11	14	9	9	7
80	15	11	13	8	13	12	6	4
100	13	11	8	6	17	11	10	12
120	14	10	6	7	10	11	13	9
140	12	9	7	9	11	10	10	7
160	4	14	5	8	5	13	9	13
180	3	9	4	8	3	5	8	11
200	1	4	2	10	1	3	6	14

Table 39 % Frequency Distribution of the Extent (%) x Maximum Severity Spine Measures in Groups of Forty Units (Eighty for Combined Scores).

	Facets Column		Cervical		Thoracic		Lumbar	
%	Female	Male	Female	Male	Female	Male	Female	Male
0	14	14	40	31	29	28	31	32
40	40	36	24	21	57	73	23	18
80	19	15	11	13	12	17	19	18
120	12	10	8	11	7	7	10	8
160	7	10	9	4	4	5	6	8
200	4	5	4	9	3	2	7	9
240	2	5	2	0	0	4	1	3
280	1	2	0	6	1	0	0	0
320	1	0	1	2	0	1	3	4
360	0	1	1	1	0	0	0	0
400	0	2	0	2	0	1	0	0

	Discs Column		Cervical		Thoracic		Lumbar	
%	Female	Male	Female	Male	Female	Male	Female	Male
0	10	10	45	28	17	16	28	18
40	28	19	17	19	19	18	15	10
80	15	9	12	15	28	15	19	9
120	12	10	8	7	8	12	11	9
160	15	13	4	6	10	5	5	9
200	8	10	7	13	13	13	15	28
240	3	0	1	0	0	1	1	2
280	4	7	1	2	0	5	0	0
320	2	10	3	6	3	5	6	14
360	1	3	0	1	0	1	0	0
400	2	9	0	3	2	9	0	1

	Combined Column		Cervical		Thoracic		Lumbar	
%	Female	Male	Female	Male	Female	Male	Female	Male
0	4	4	25	15	9	8	18	12
80	38	27	32	32	37	32	25	19
160	16	15	16	14	21	16	15	11
240	18	11	12	11	19	15	17	17
320	11	11	5	8	8	9	14	16
400	7	11	15	6	3	10	7	12
480	3	10	2	5	2	6	1	5
560	2	4	2	3	1	1	2	5
640	0	4	0	3	0	1	1	2
720	1	0	1	2	0	1	0	1
800	0	3	0	1	0	1	0	0

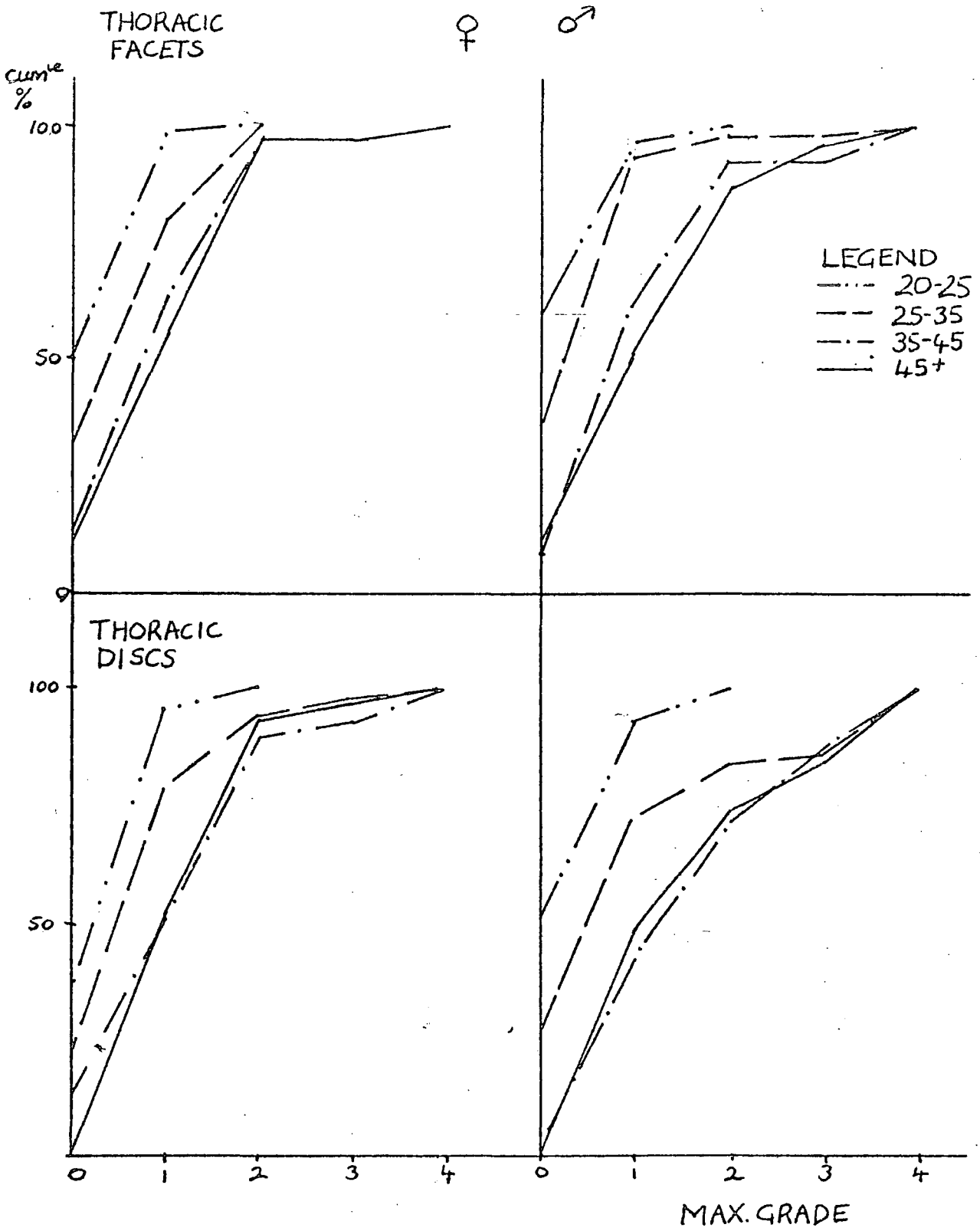


FIG. 1. CUMULATIVE % FREQUENCIES OF THE MAXIMUM GRADES OF THE FACET AND DISC JOINTS IN THE THORACIC REGION PLOTTED BY DENTAL AGE GROUP.

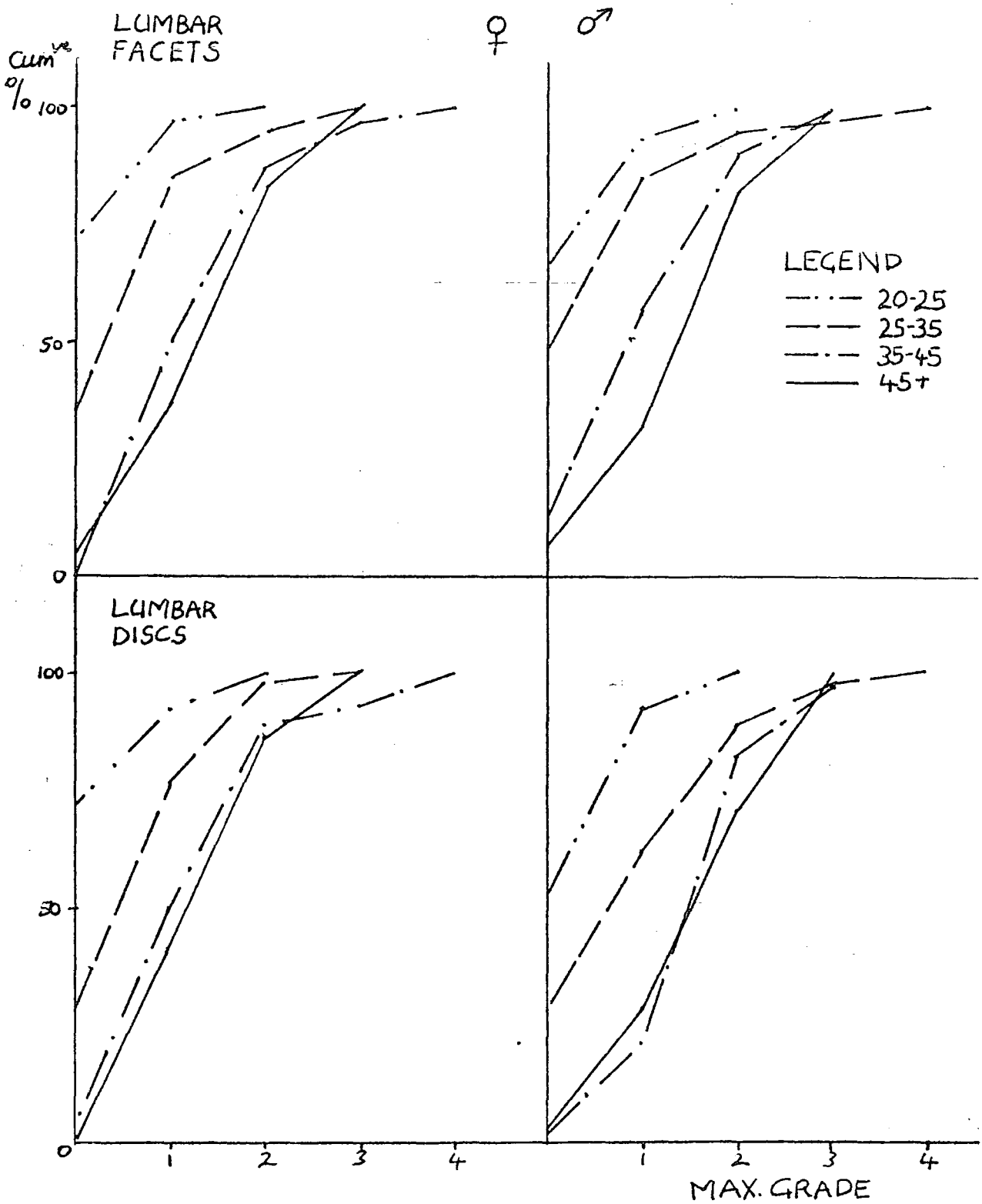


FIG. 2. CUMULATIVE % FREQUENCIES OF THE MAXIMUM GRADES OF THE FACET AND DISC JOINTS IN THE LUMBAR REGION PLOTTED BY DENTAL AGE GROUP.

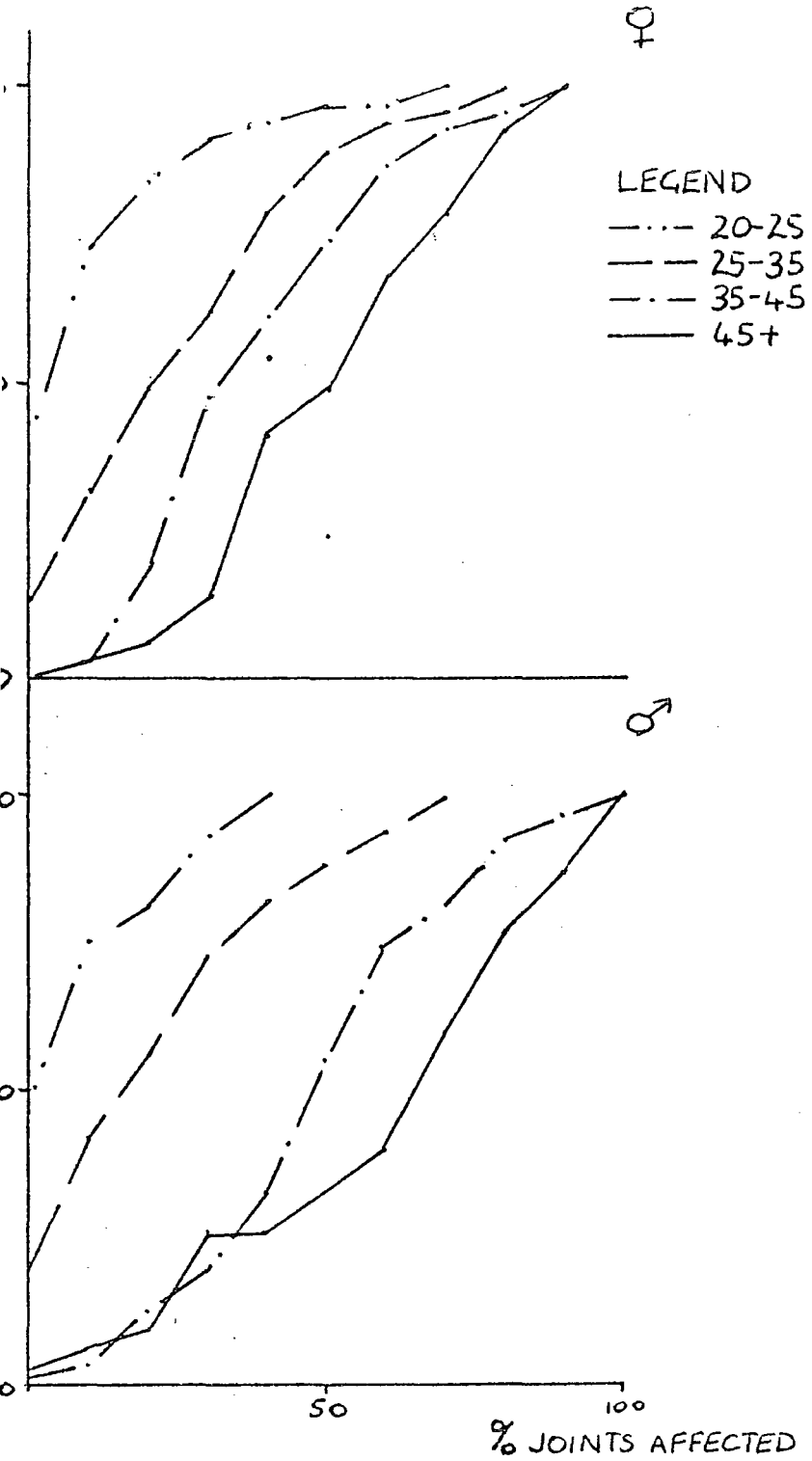


FIG. 3. CUMULATIVE % FREQUENCIES OF THE FACET EXTENT (%) SCORE WITHIN THE WHOLE SPINE PLOTTED BY DENTAL AGE GROUP.

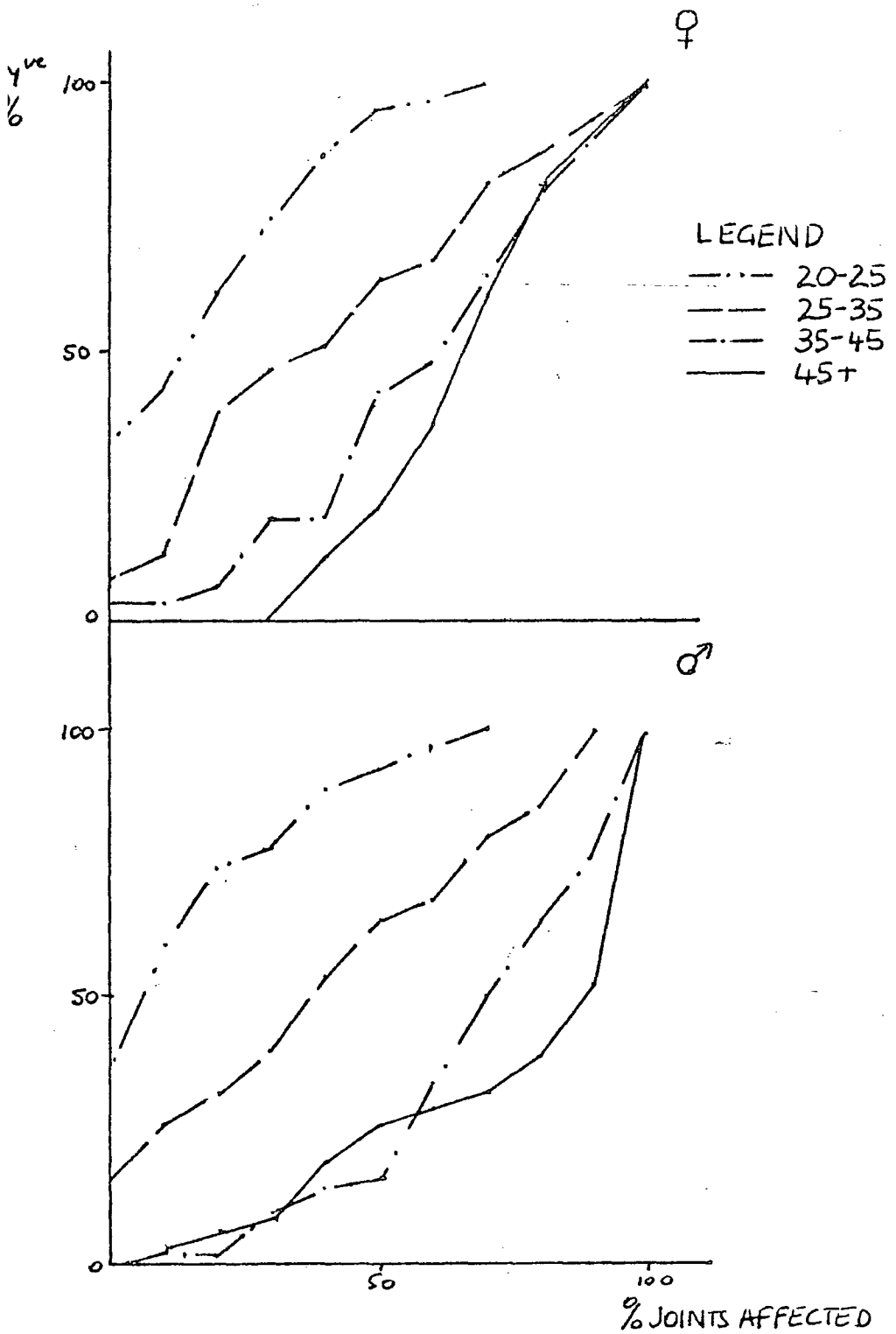


FIG. 4. CUMULATIVE % FREQUENCIES OF THE DISC EXTENT (%) SCORE WITHIN THE WHOLE SPINE PLOTTED BY DENTAL AGE GROUP.

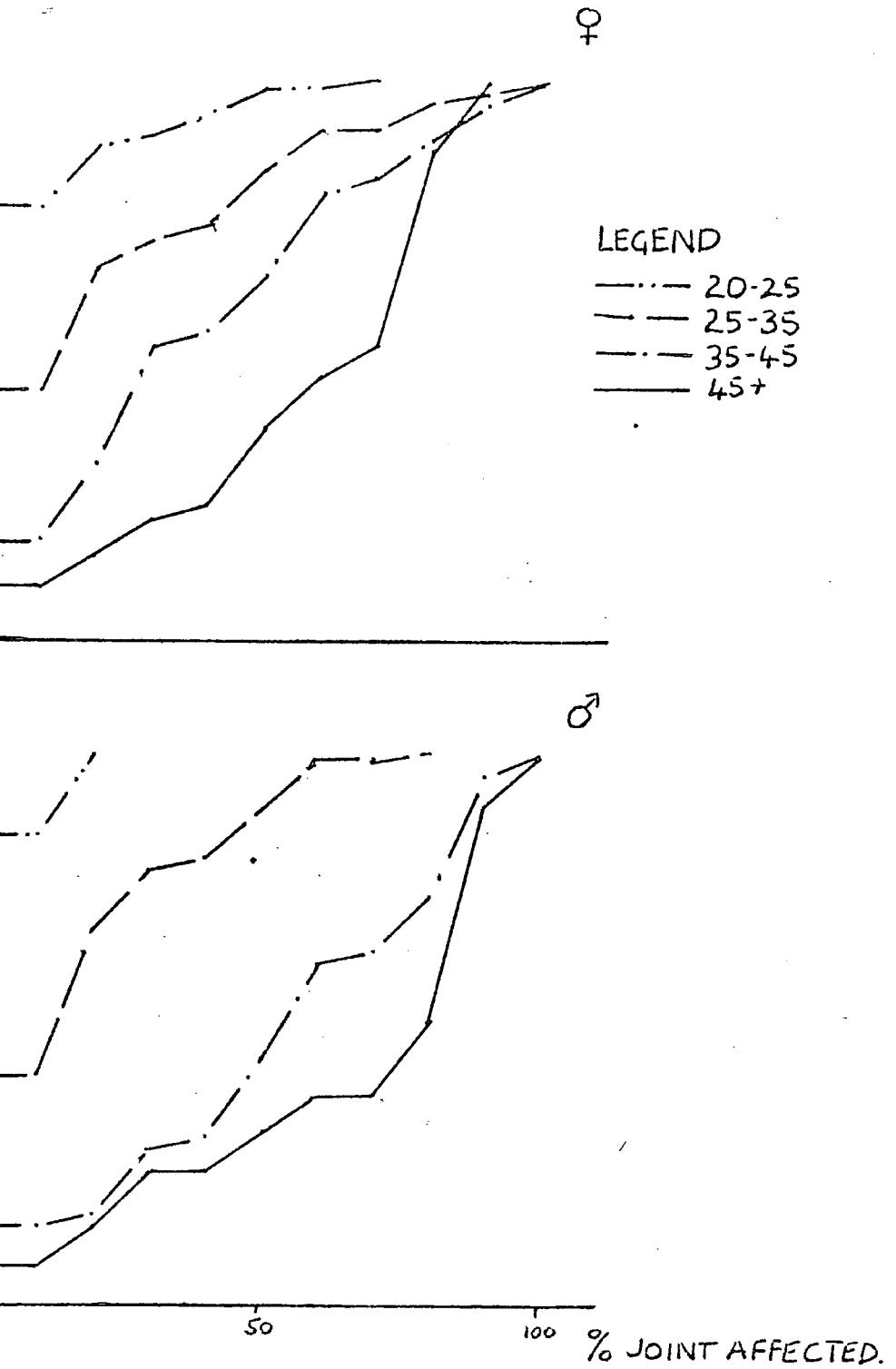


FIG. 5. CUMULATIVE % FREQUENCIES OF THE FACET EXTENT (%) SCORE WITHIN THE CERVICAL SPINE PLOTTED BY DENTAL AGE GROUP.

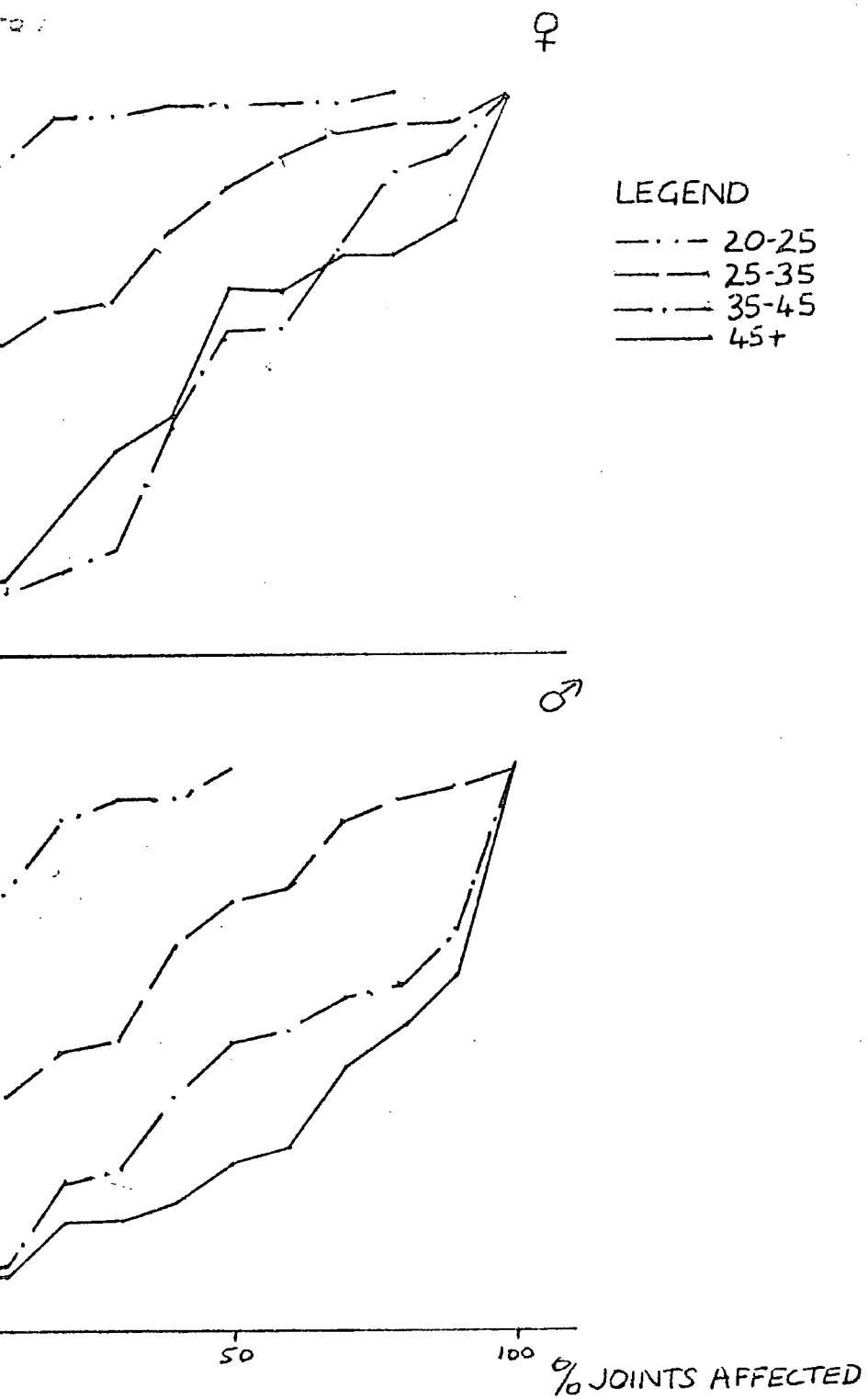


FIG. 6. CUMULATIVE % FREQUENCIES OF THE DISC EXTENT (%) SCORE WITHIN THE CERVICAL SPINE PLOTTED BY DENTAL AGE GROUP.

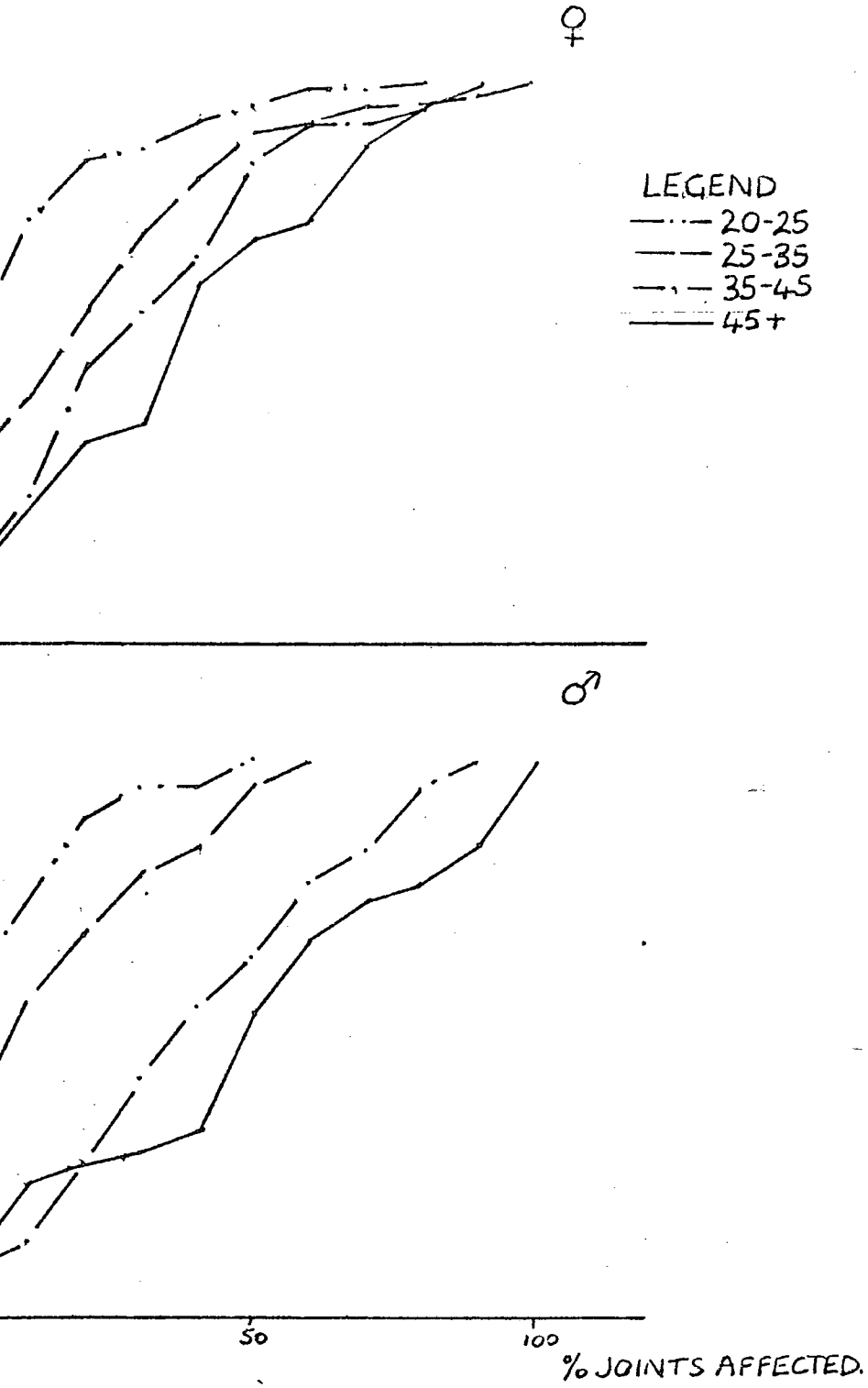


FIG. 7. CUMULATIVE % FREQUENCIES OF THE FACET EXTENT (%) SCORE WITHIN THE THORACIC SPINE PLOTTED BY DENTAL AGE GROUP.

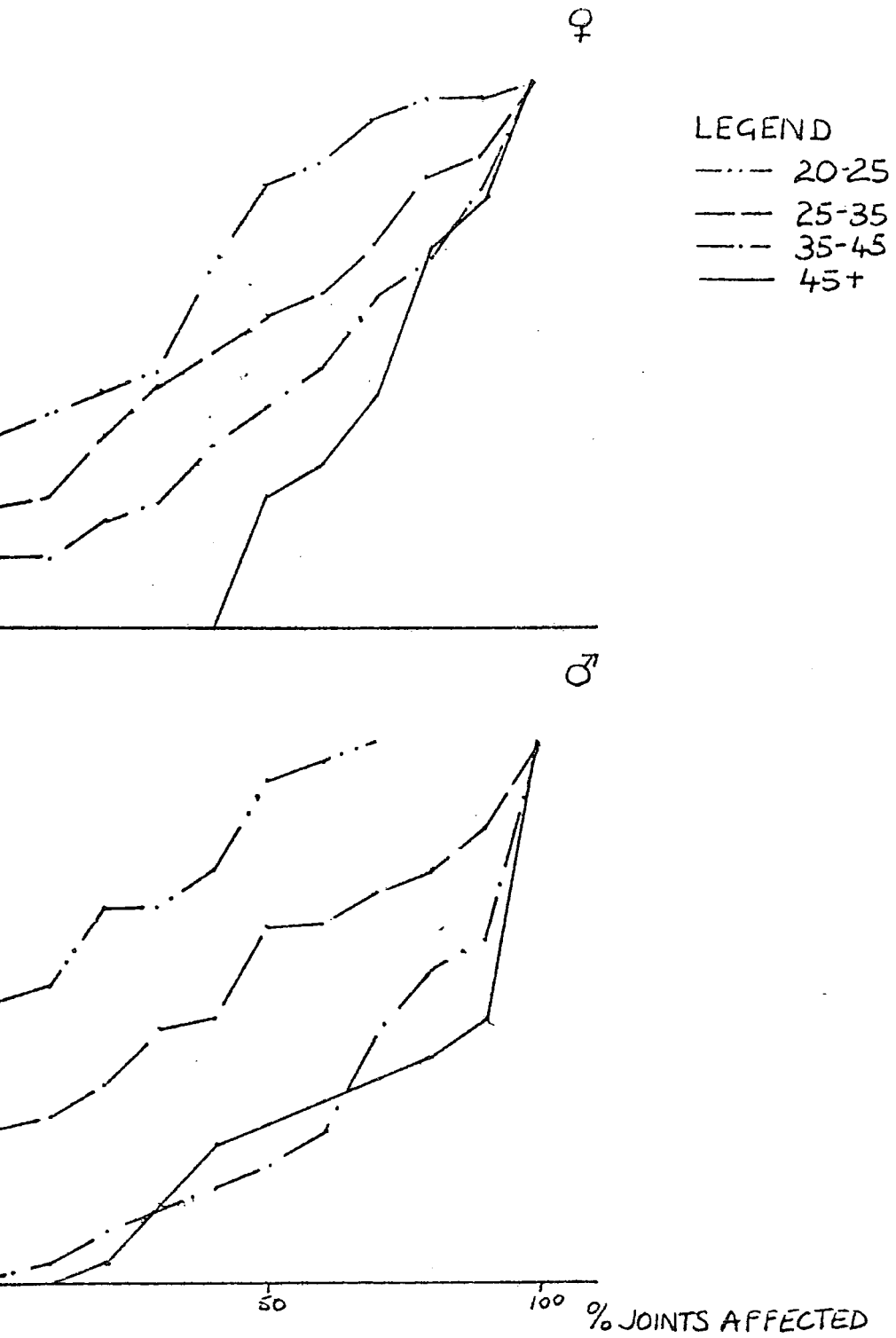


FIG. 8. CUMULATIVE % FREQUENCIES OF THE DISC EXTENT (%) SCORE WITHIN THE THORACIC SPINE PLOTTED BY DENTAL AGE GROUP.

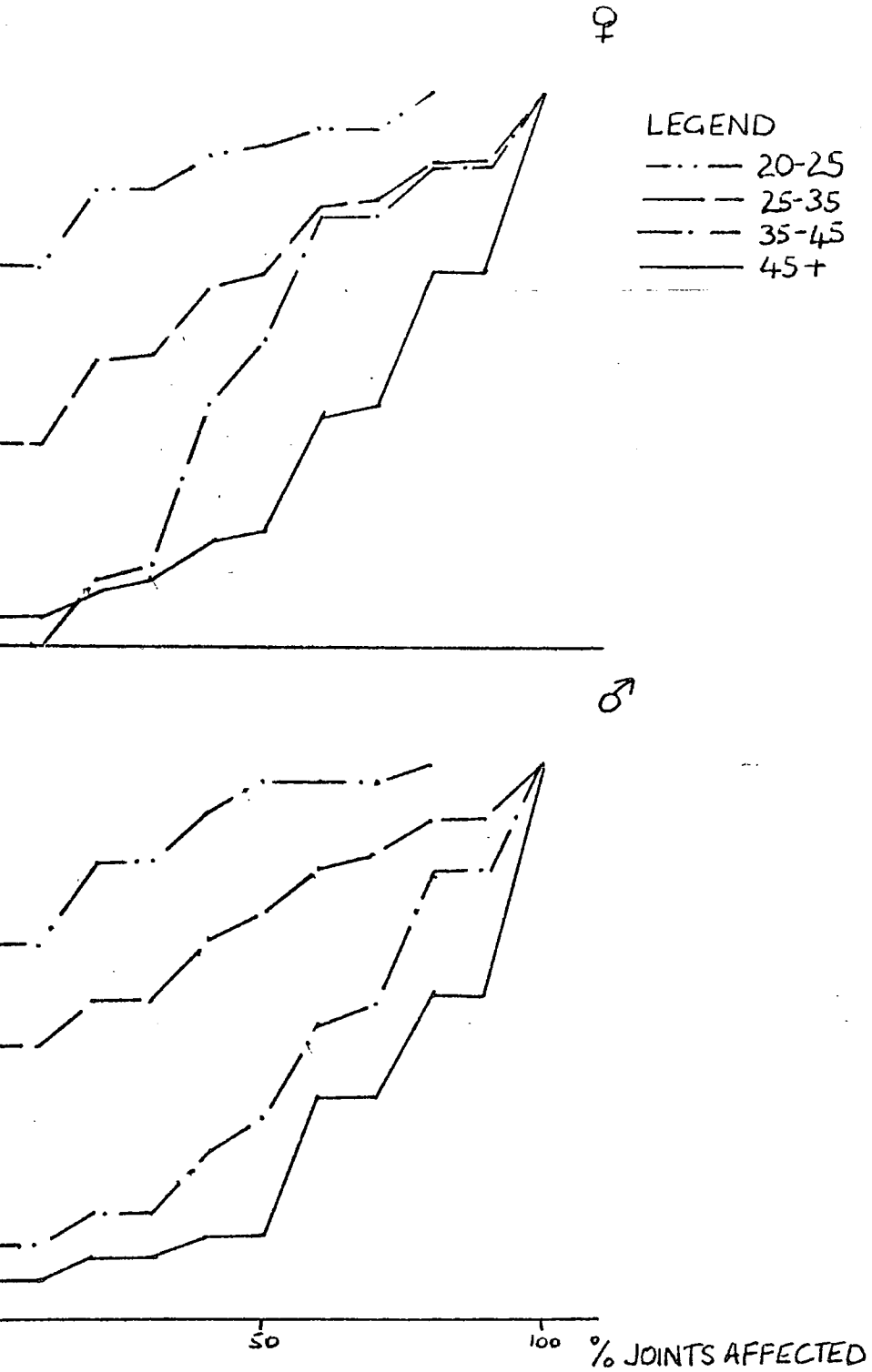


FIG. 9. CUMULATIVE % FREQUENCIES OF THE FACET EXTENT (%) SCORE WITHIN THE LUMBAR SPINE PLOTTED BY DENTAL AGE GROUP.

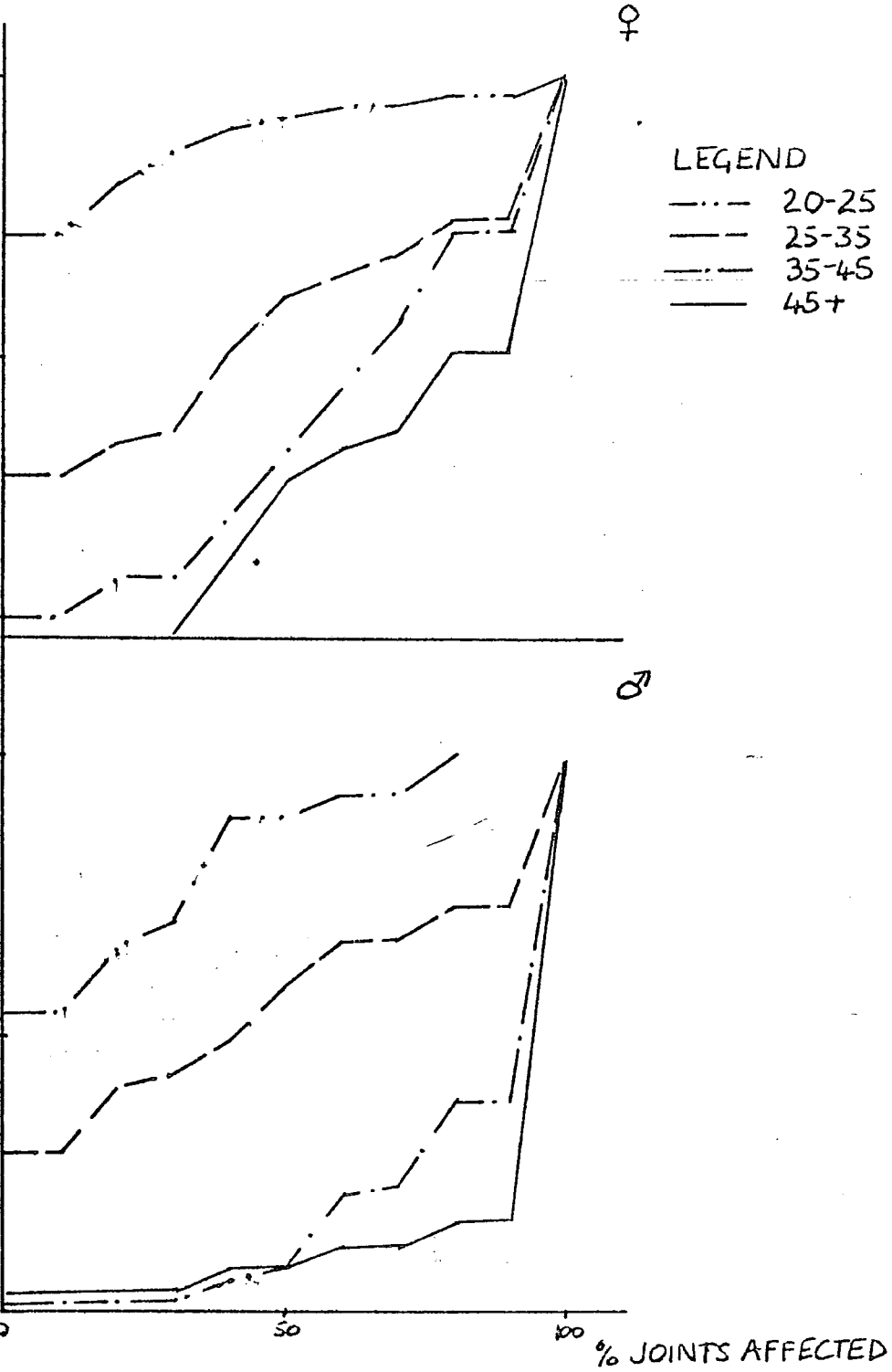


FIG. 10. CUMULATIVE % FREQUENCIES OF THE DISC EXTENT (%) SCORE WITHIN THE LUMBAR SPINE PLOTTED BY DENTAL AGE GROUP.

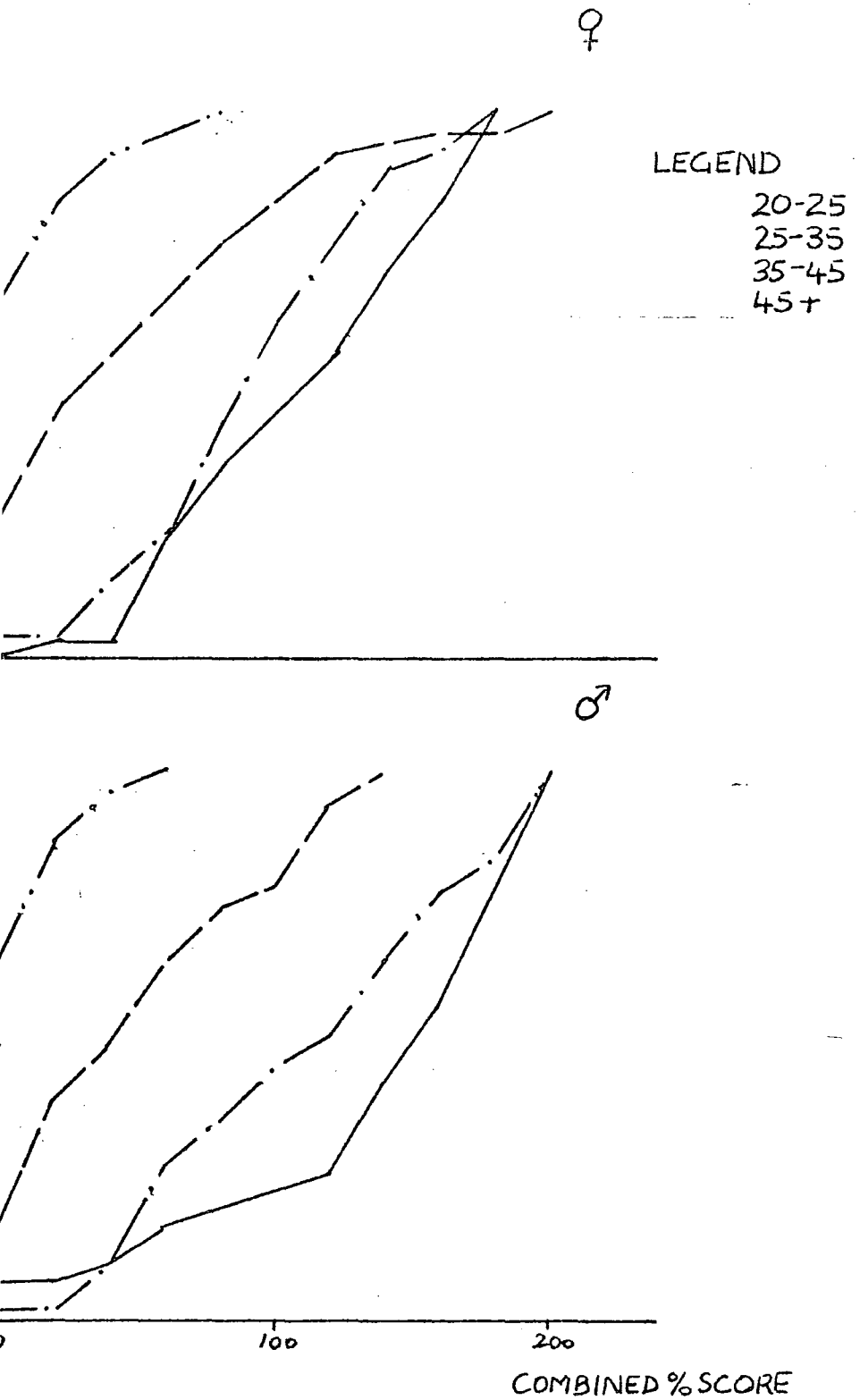


FIG. 11. CUMULATIVE % FREQUENCIES OF COMBINED EXTENT (%) SCORES WITHIN THE CERVICAL REGION PLOTTED BY DENTAL AGE GROUP.

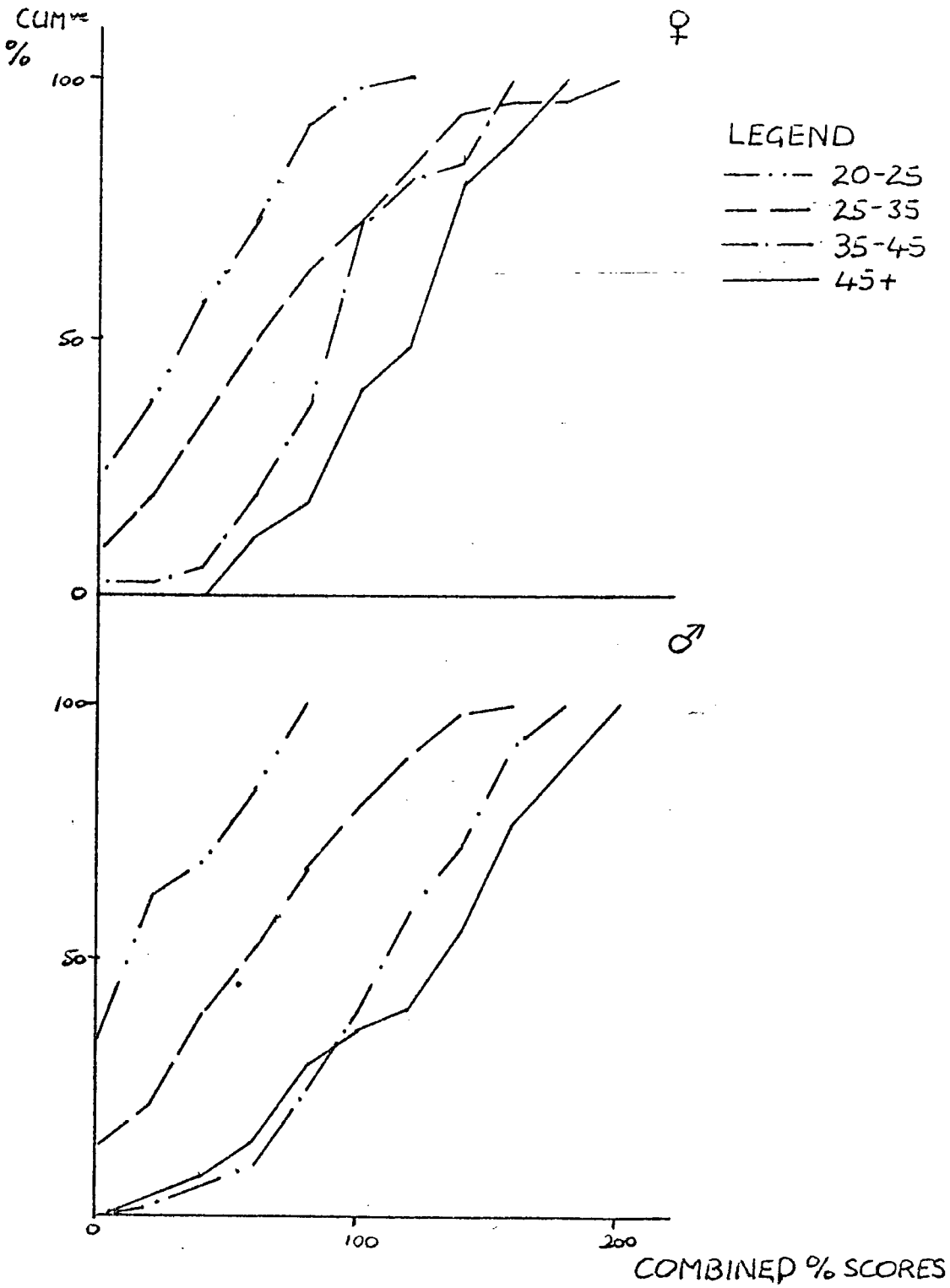


FIG. 12. CUMULATIVE % FREQUENCIES OF COMBINED EXTENT (%) SCORES WITHIN THE THORACIC REGION PLOTTED BY DENTAL AGE GROUP.

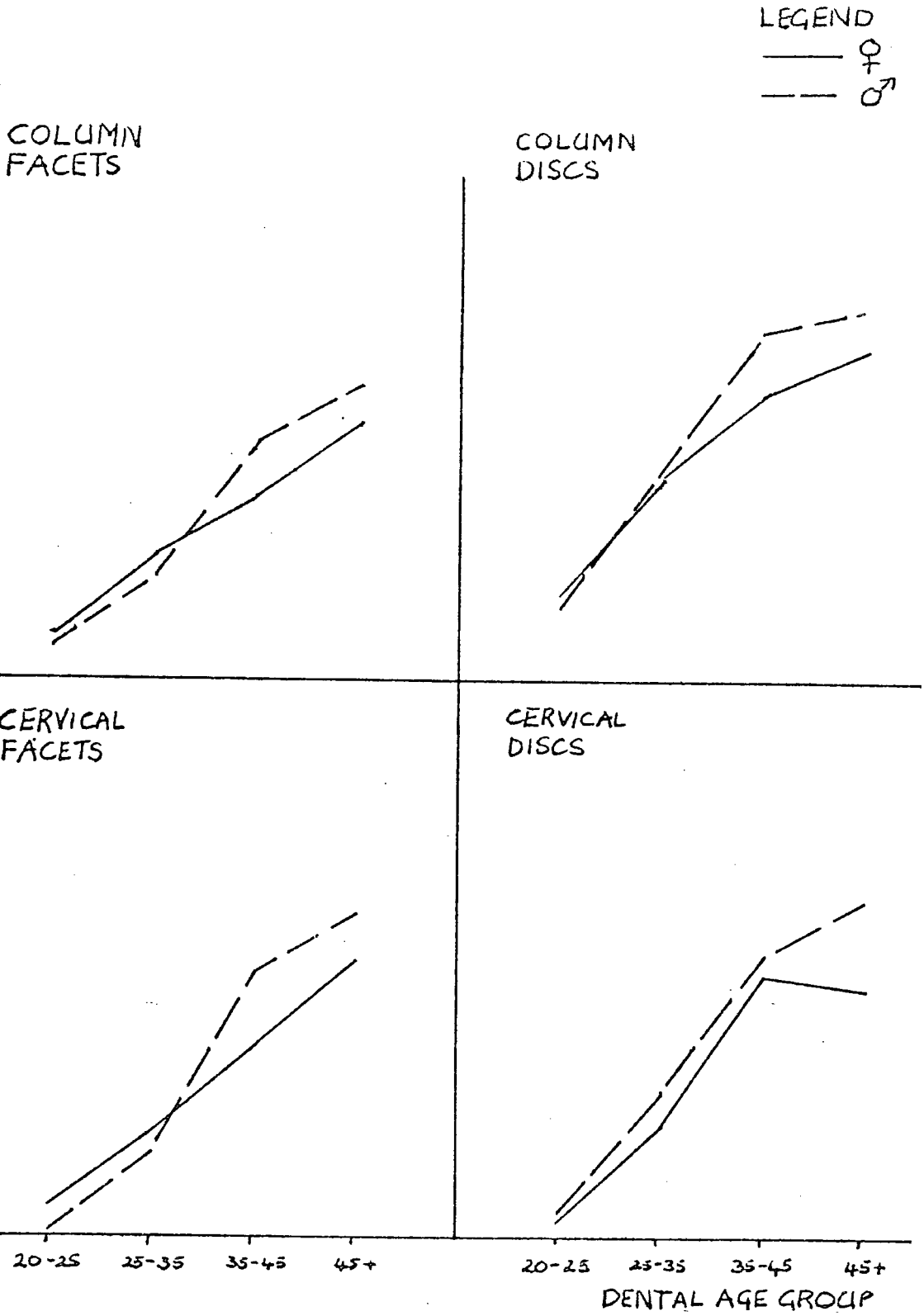
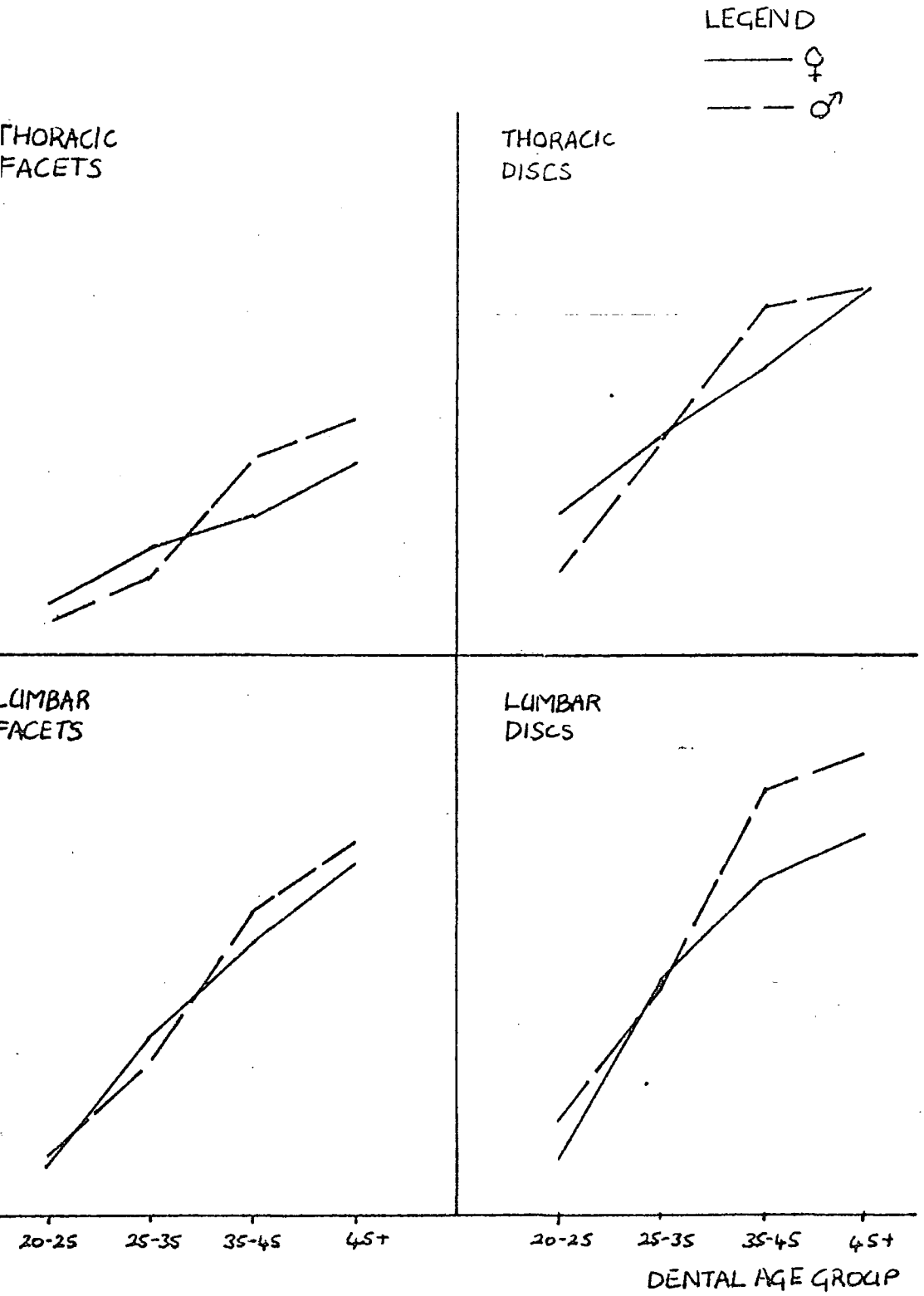


FIG. 13. MEAN VALUES OF FACET AND DISC EXTENT (%) SCORES WITHIN THE WHOLE SPINE AND BY REGION.


LEGEND
♀
♂
THORACIC FACETS
THORACIC DISCS
LUMBAR FACETS
LUMBAR DISCS
20-25
25-35
35-45
45+
20-25
25-35
35-45
45+
DENTAL AGE GROUP

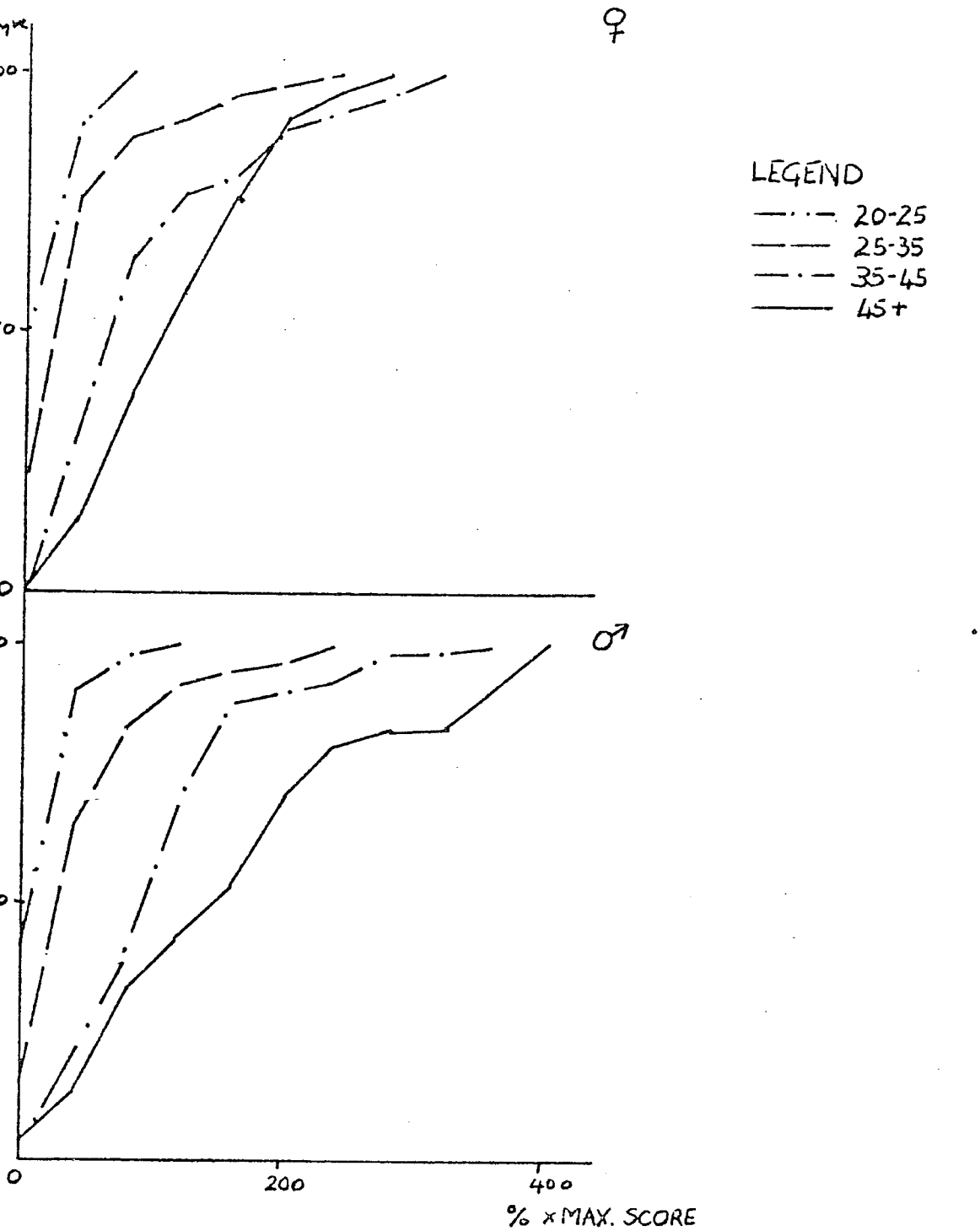


FIG. 14. CUMULATIVE % FREQUENCIES OF FACET EXTENT (%) X MAX. GRADE SCORES WITHIN THE WHOLE SPINE PLOTTED BY DENTAL AGE GROUP.

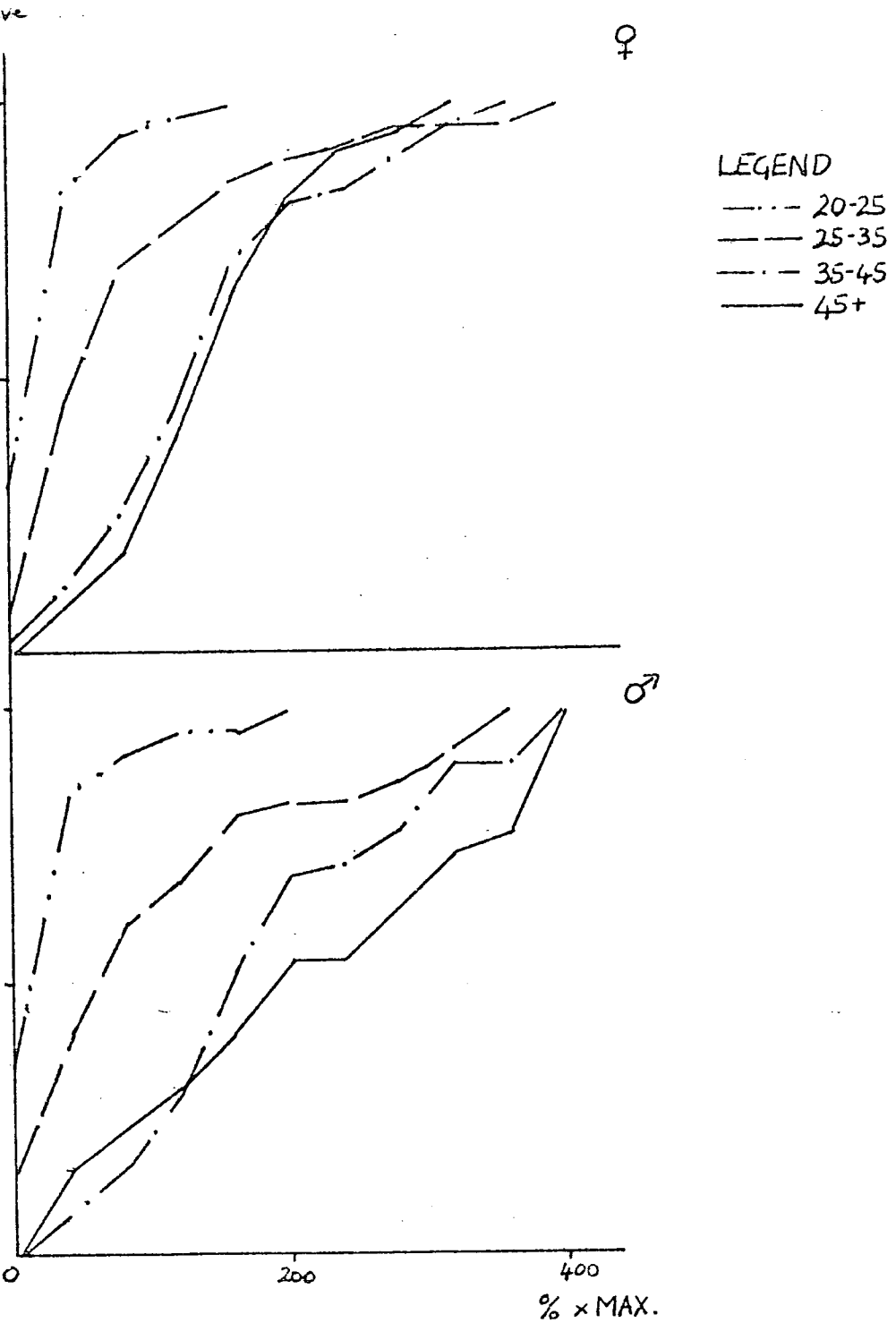


FIG. 15. CUMULATIVE % FREQUENCIES OF DISC EXTENT (%) X MAX. GRADE SCORES WITHIN THE WHOLE SPINE PLOTTED BY DENTAL AGE GROUP.

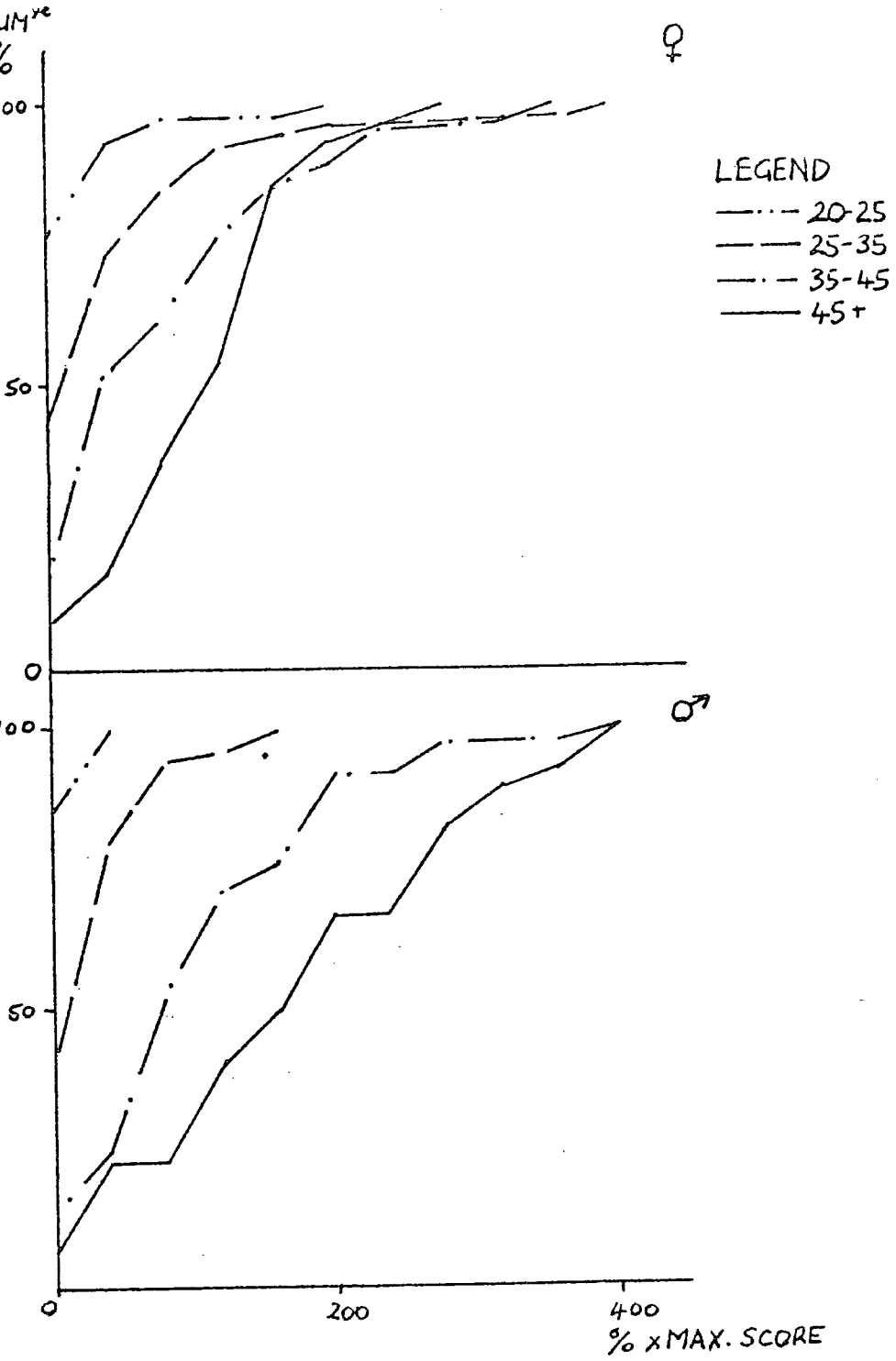


FIG. 16. CUMULATIVE % FREQUENCIES OF FACET EXTENT (%) X MAX. GRADE SCORES WITHIN THE CERVICAL REGION PLOTTED BY DENTAL AGE GROUP.

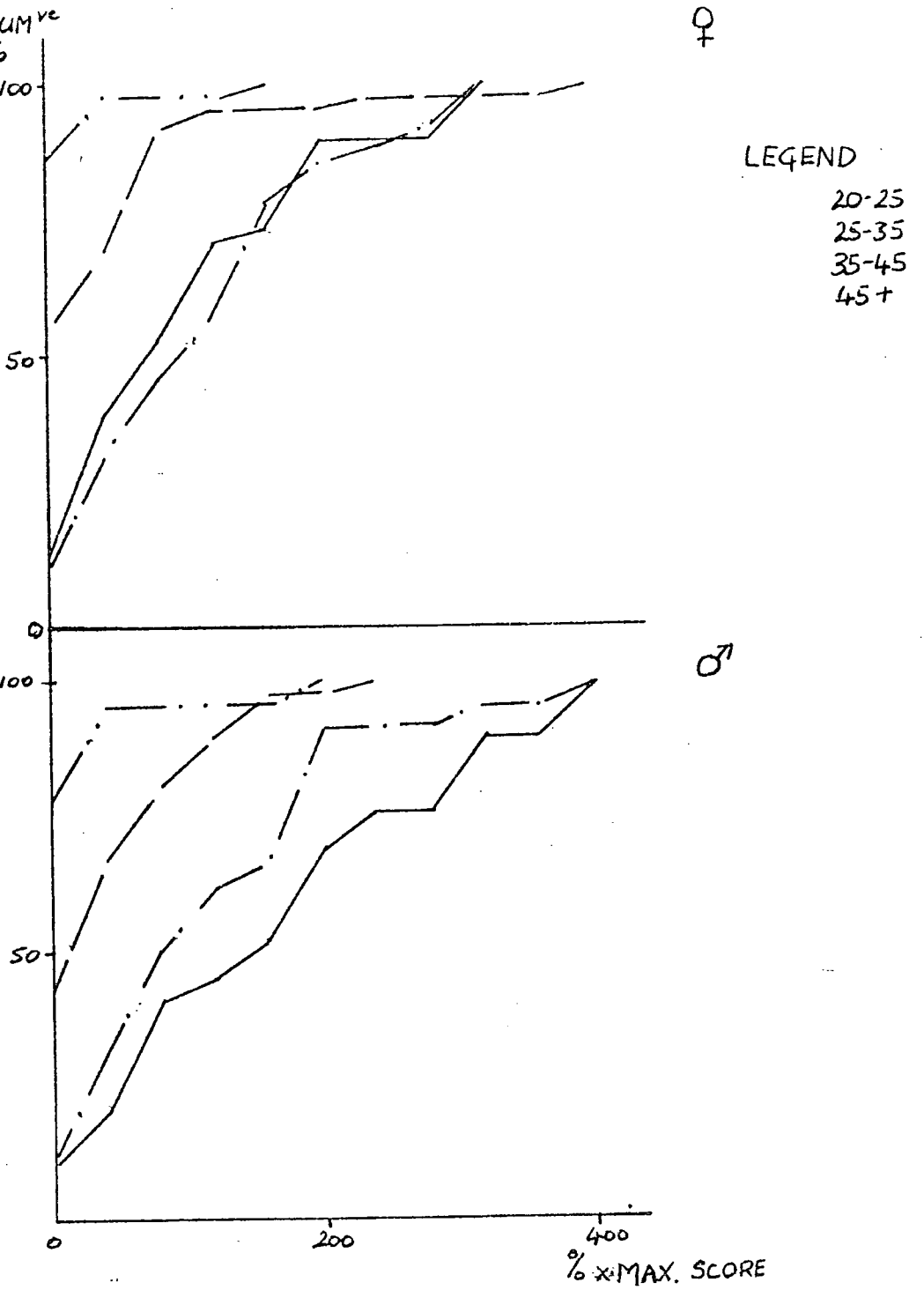


FIG. 17. CUMULATIVE % FREQUENCIES OF DISC EXTENT (%) X MAX. GRADE SCORES WITHIN THE CERVICAL REGION PLOTTED BY DENTAL AGE GROUP.

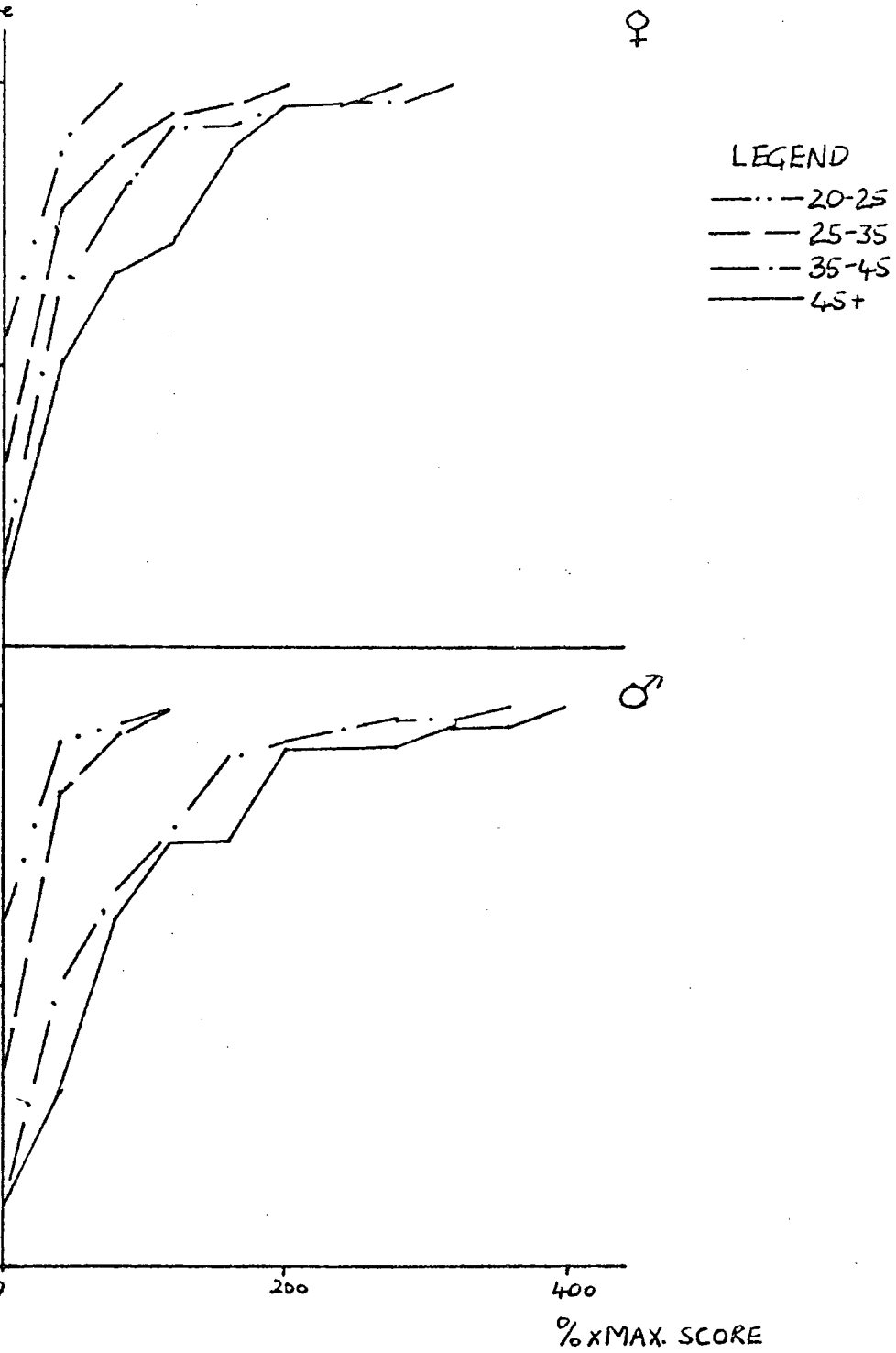


FIG. 18. CUMULATIVE % FREQUENCIES OF FACET EXTENT (%) X MAX. GRADE SCORES WITHIN THE THORACIC REGION PLOTTED BY DENTAL AGE GROUP.

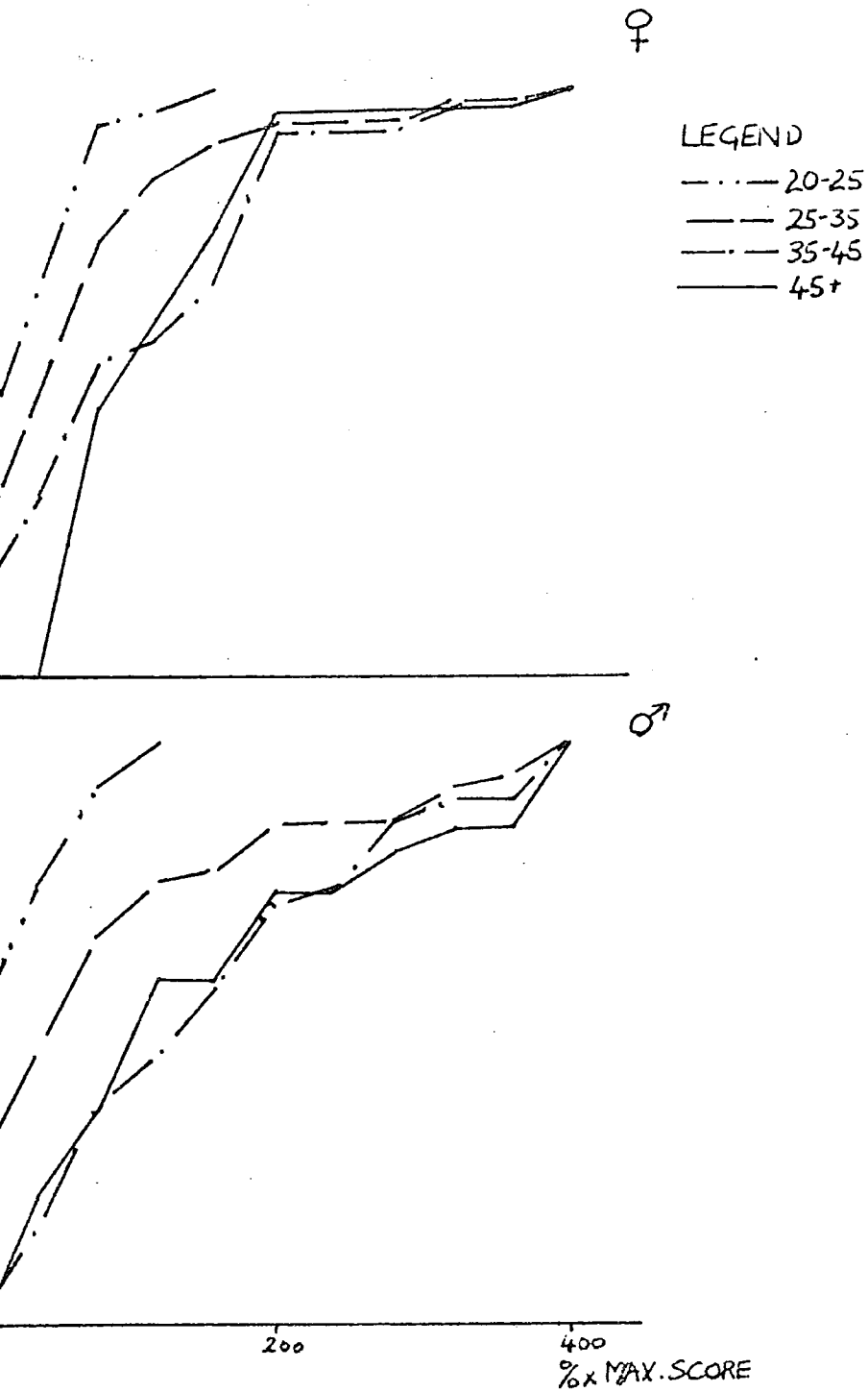


FIG. 19. CUMULATIVE % FREQUENCIES OF DISC EXTENT (%) X MAX. GRADE SCORES WITHIN THE THORACIC REGION PLOTTED BY DENTAL AGE GROUP.

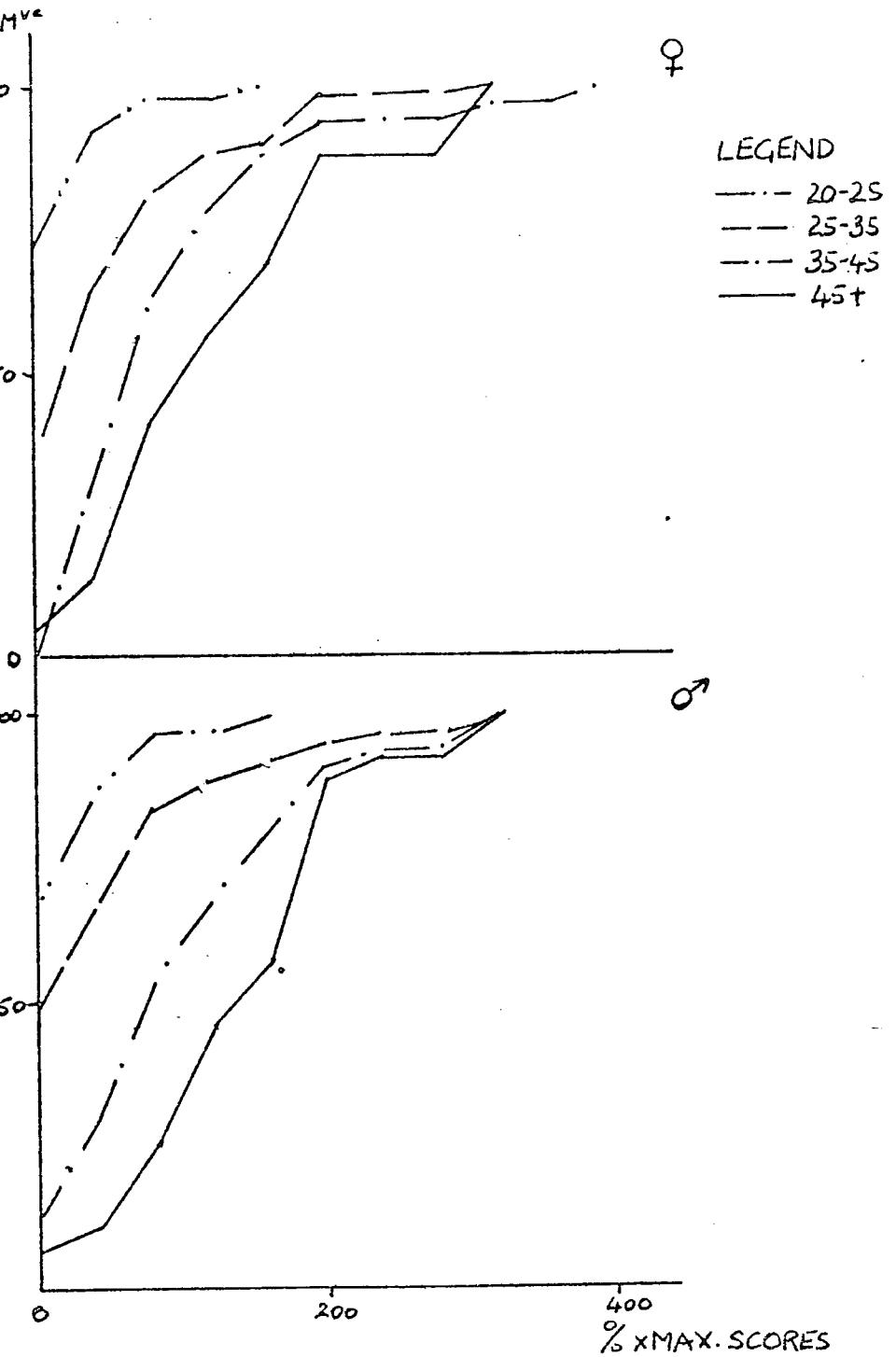


FIG. 20. CUMULATIVE % FREQUENCIES OF FACET EXTENT (%) X MAX. GRADE SCORES WITHIN THE LUMBAR REGION PLOTTED BY DENTAL AGE GROUP.

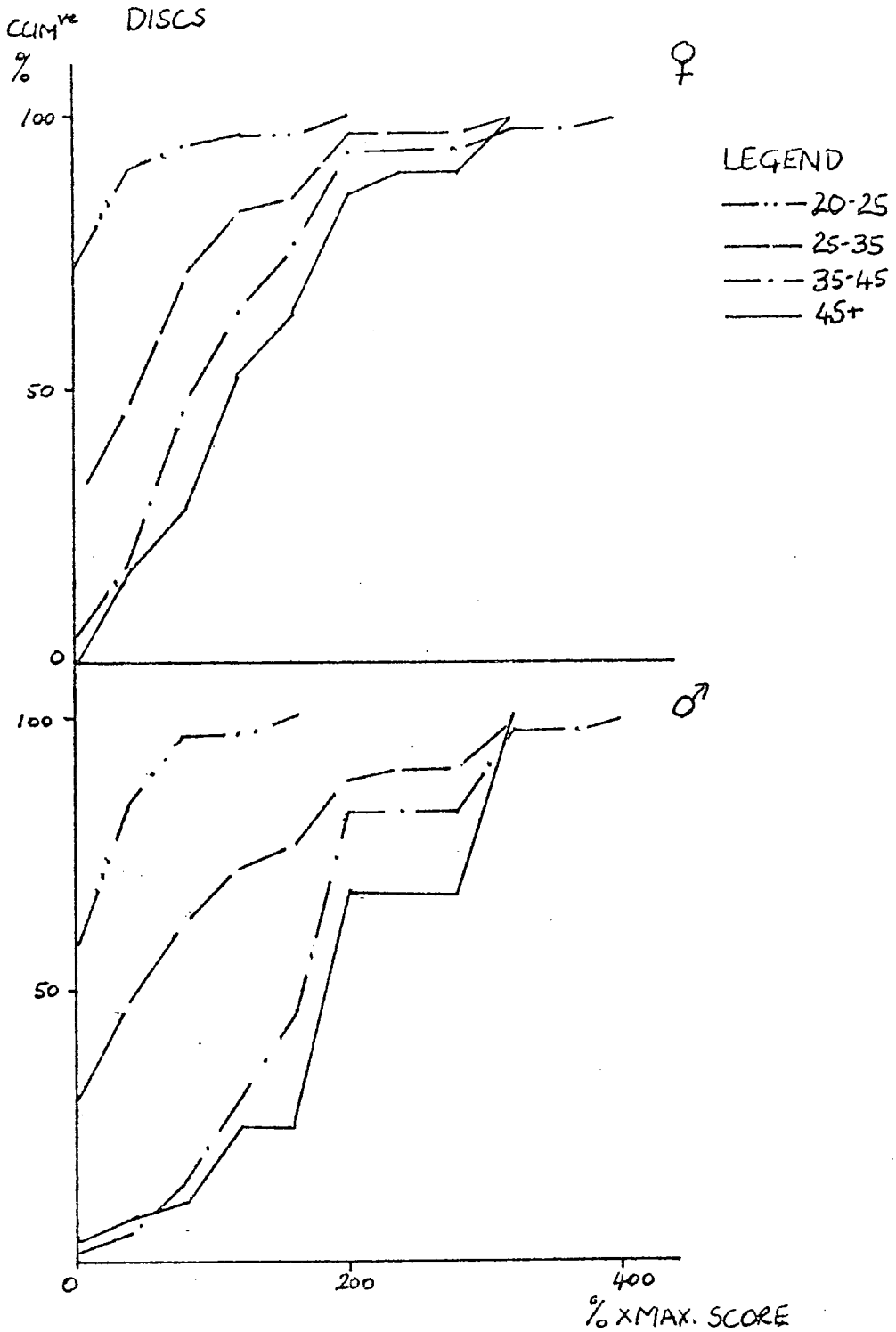


FIG. 21. CUMULATIVE % FREQUENCIES OF DISC EXTENT (%) X MAX. GRADE SCORES WITHIN THE LUMBAR REGION PLOTTED BY DENTAL AGE GROUP.

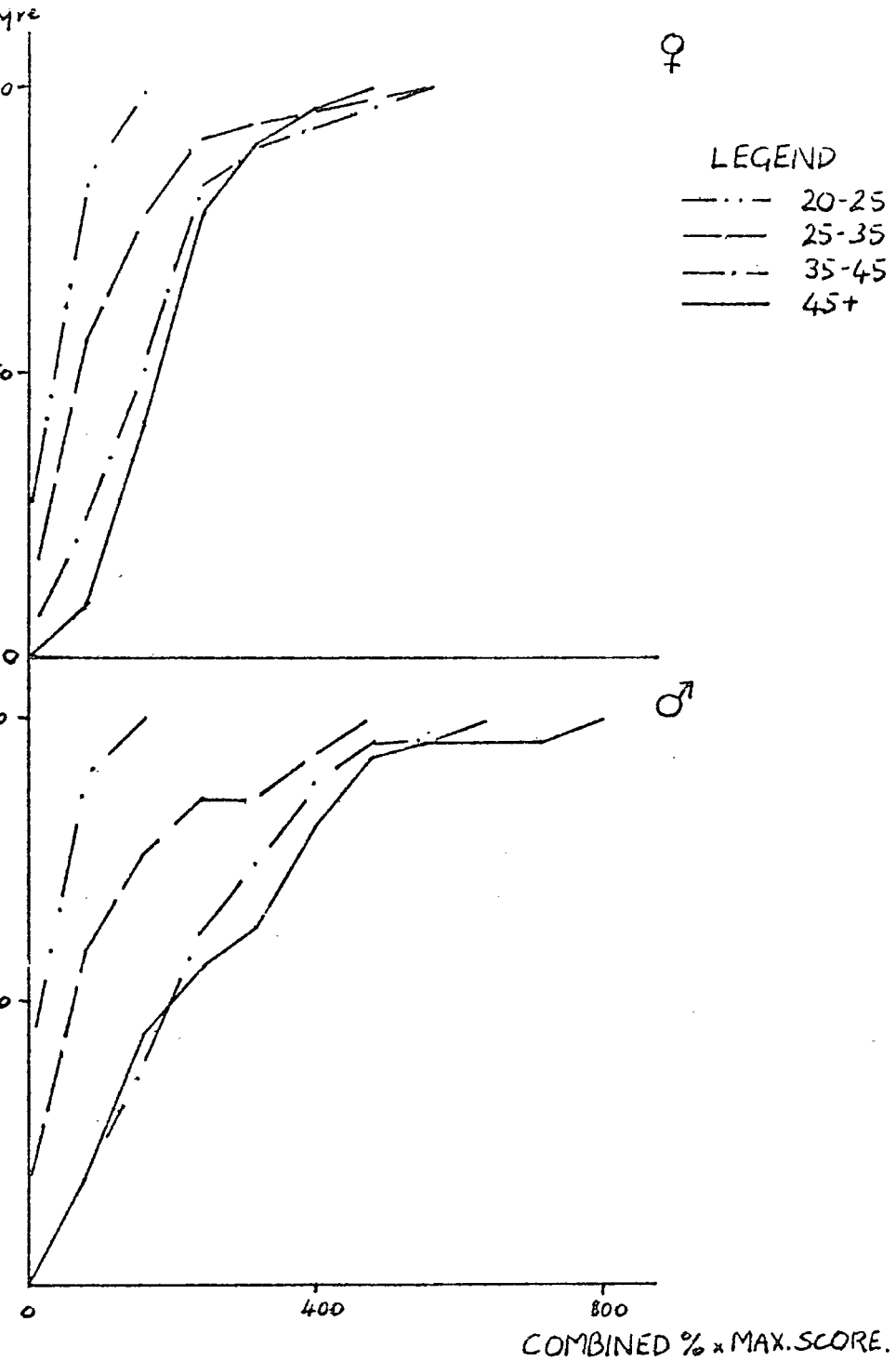


FIG. 22. CUMULATIVE % FREQUENCIES OF THE COMBINED EXTENT (%) X MAX. GRADE SCORES WITHIN THE THORACIC REGION PLOTTED BY DENTAL AGE GROUP.

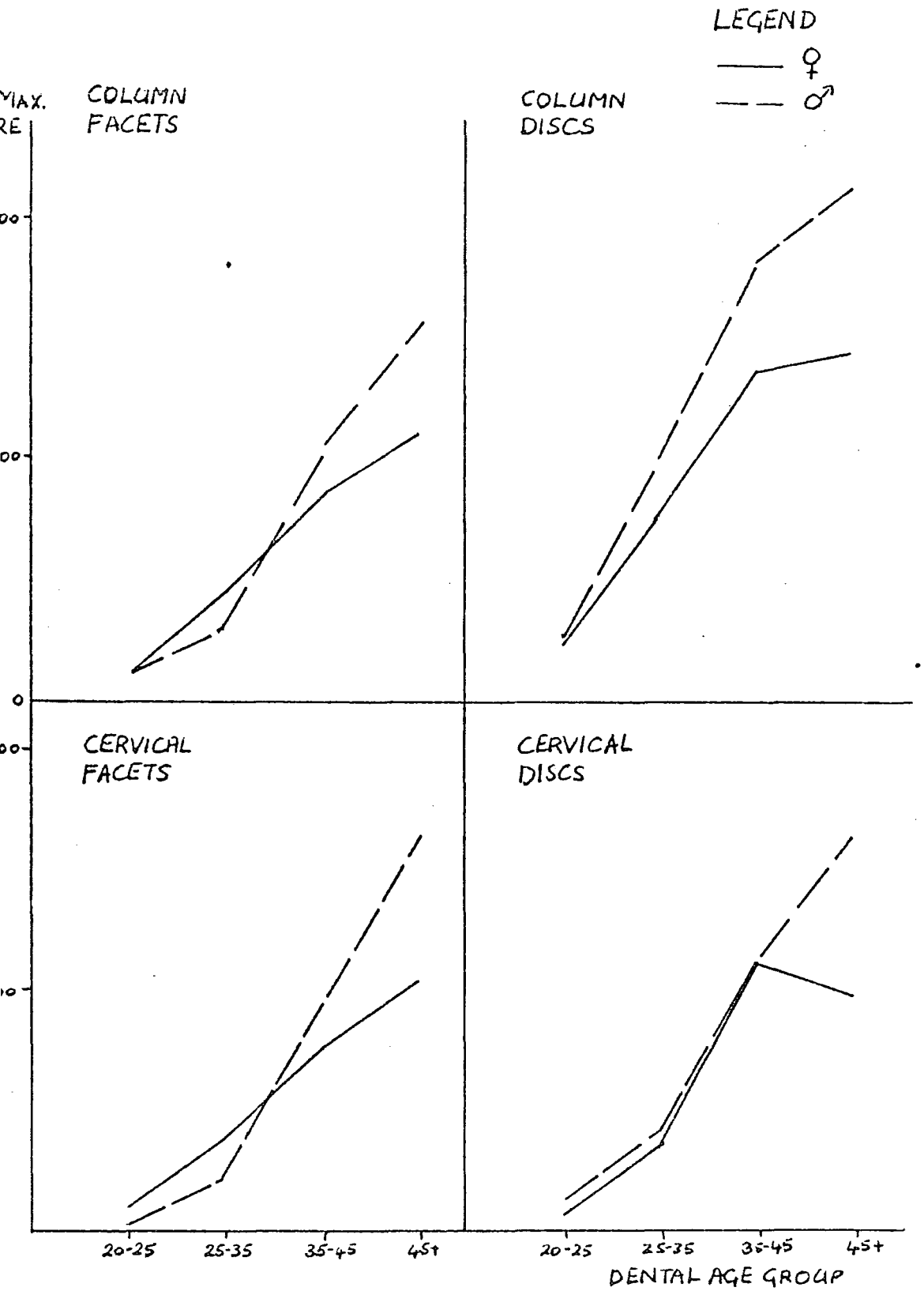
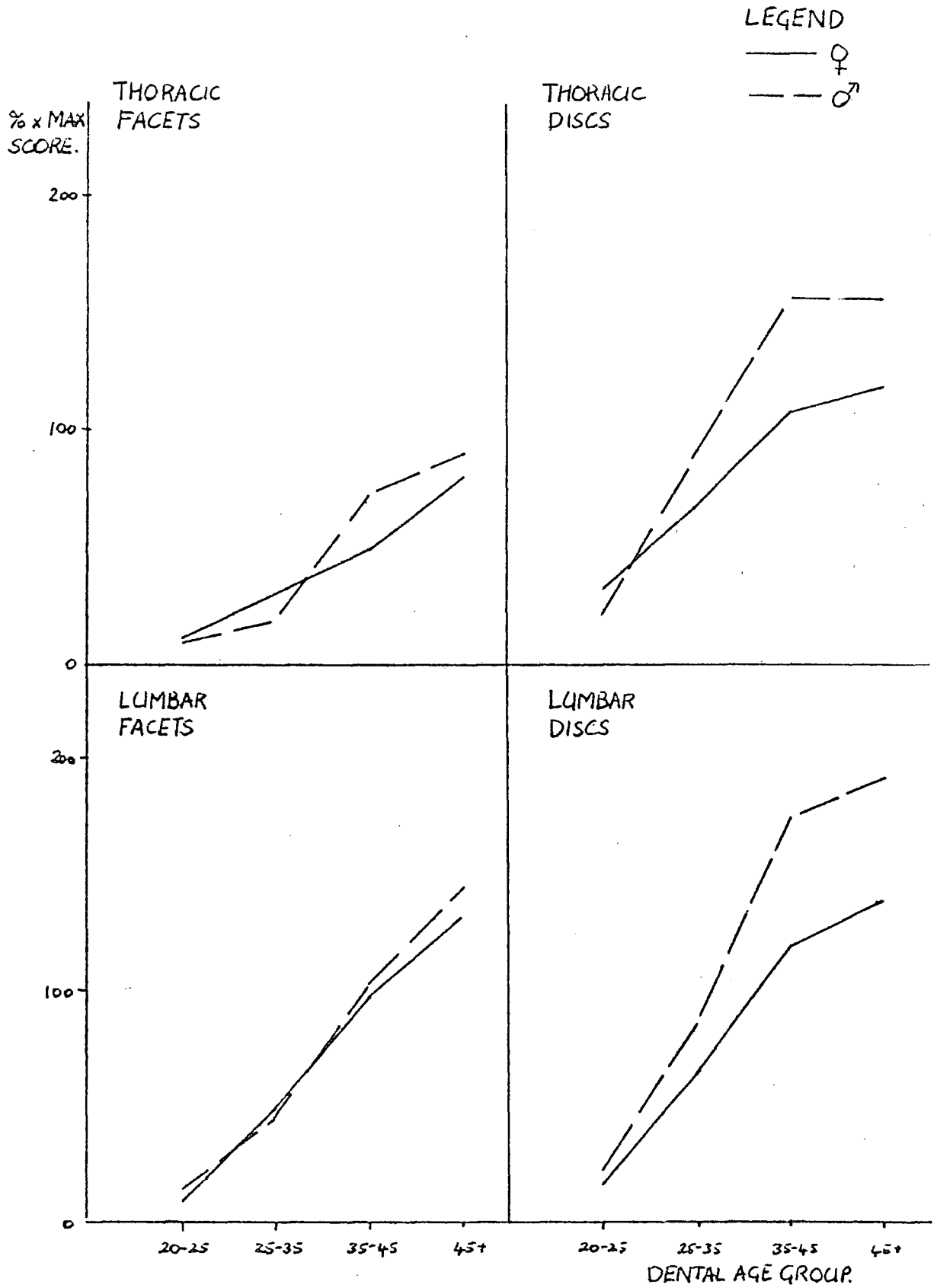
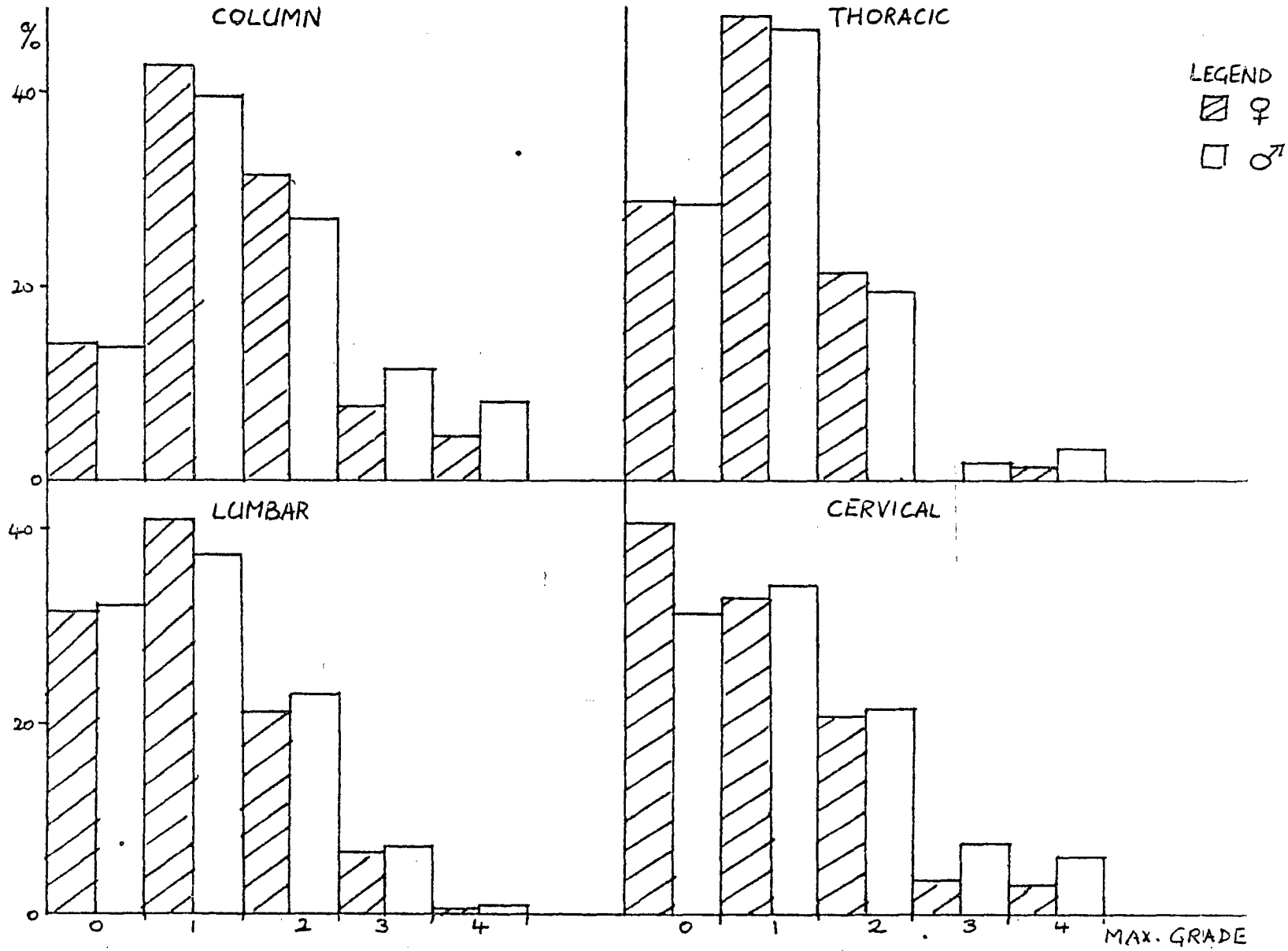
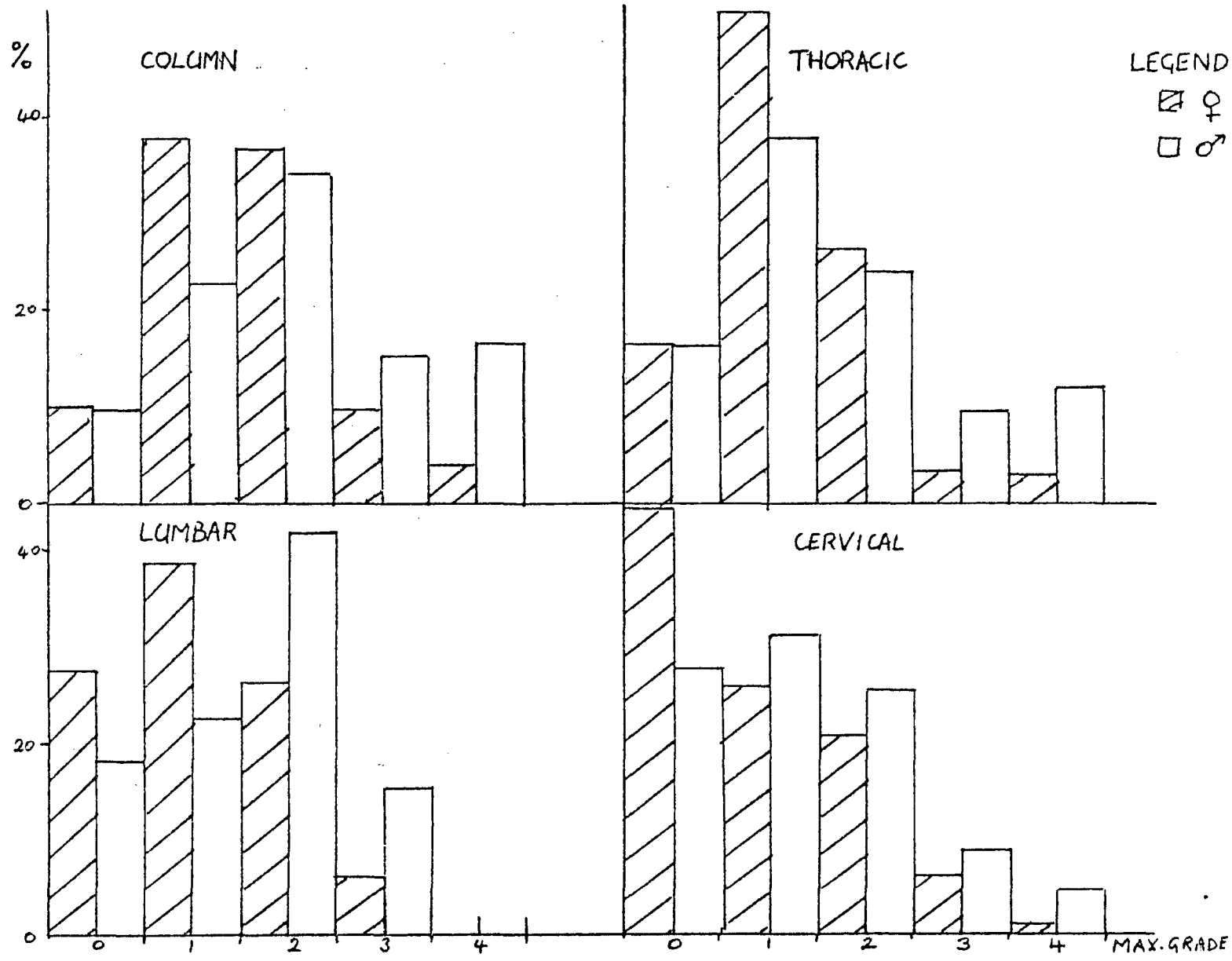


FIG. 23. MEAN VALUES OF THE FACET AND DISC EXTENT (S) X MAX. GRADE SCORES WITHIN THE WHOLE SPINE AND BY REGION IN EACH DENTAL AGE GROUP.

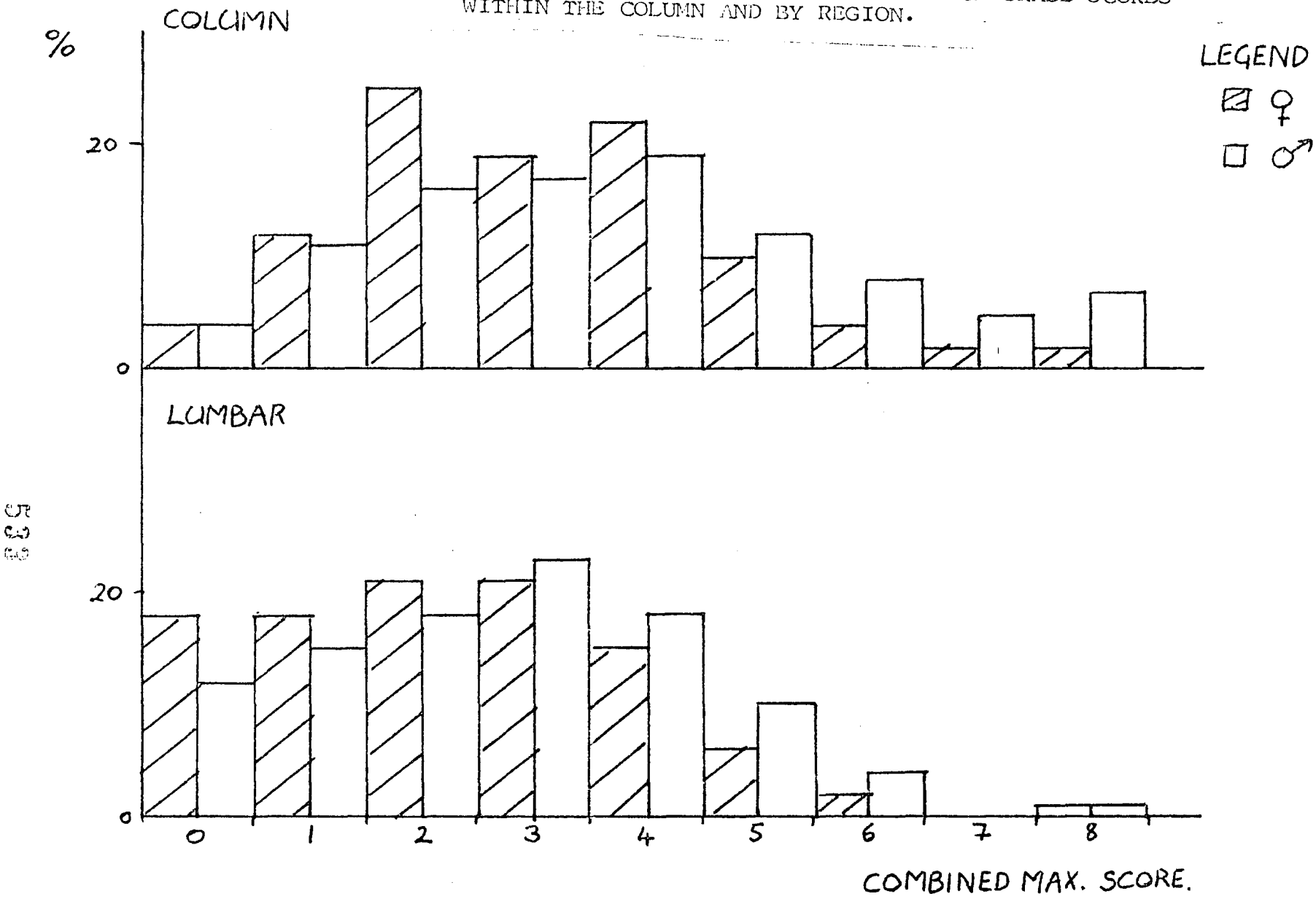

LEGEND
♀
♂
THORACIC FACETS
THORACIC DISCS
LUMBAR FACETS
LUMBAR DISCS
% x MAX SCORE.
200
100
0
200
100
0
20-25
25-35
35-45
45+
20-25
25-35
35-45
45+
DENTAL AGE GROUP.

WHOLE COLUMN AND BY REGION.




%
COLUMN
THORACIC
LEGEND
♀
♂
40
20
0
LUMBAR
CERVICAL
0
1
2
3
4
MAX. GRADE

WITHIN THE COLUMN AND BY REGION.



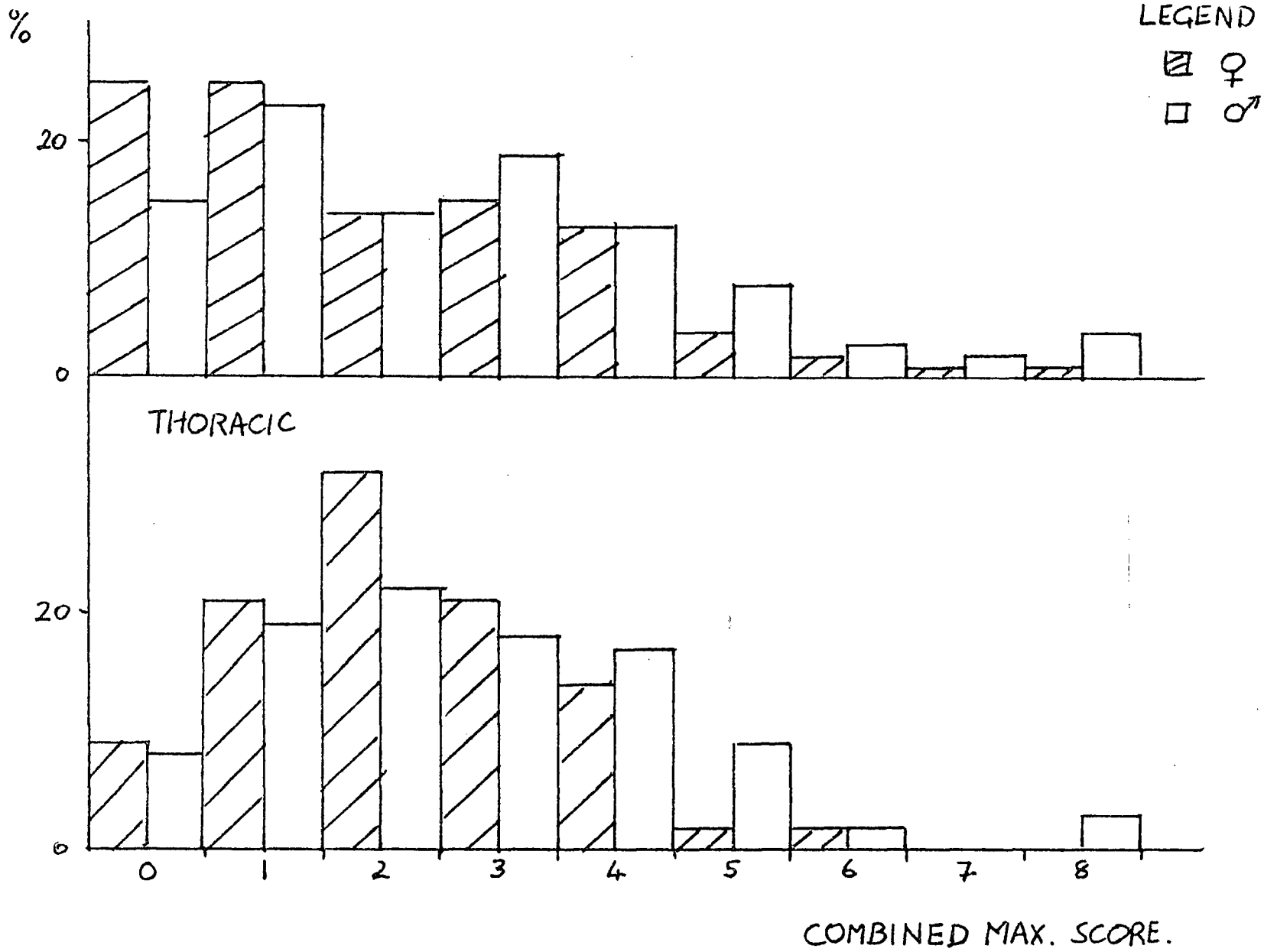

%
LEGEND
♀
♂
20
0
THORACIC
20
0
0
1
2
3
4
5
6
7
8
COMBINED MAX. SCORE.

FIG. 27. CUMULATIVE % FREQUENCIES OF FACET AND DISC EXTENT (%) SCORES WITHIN THE WHOLE COLUMN.

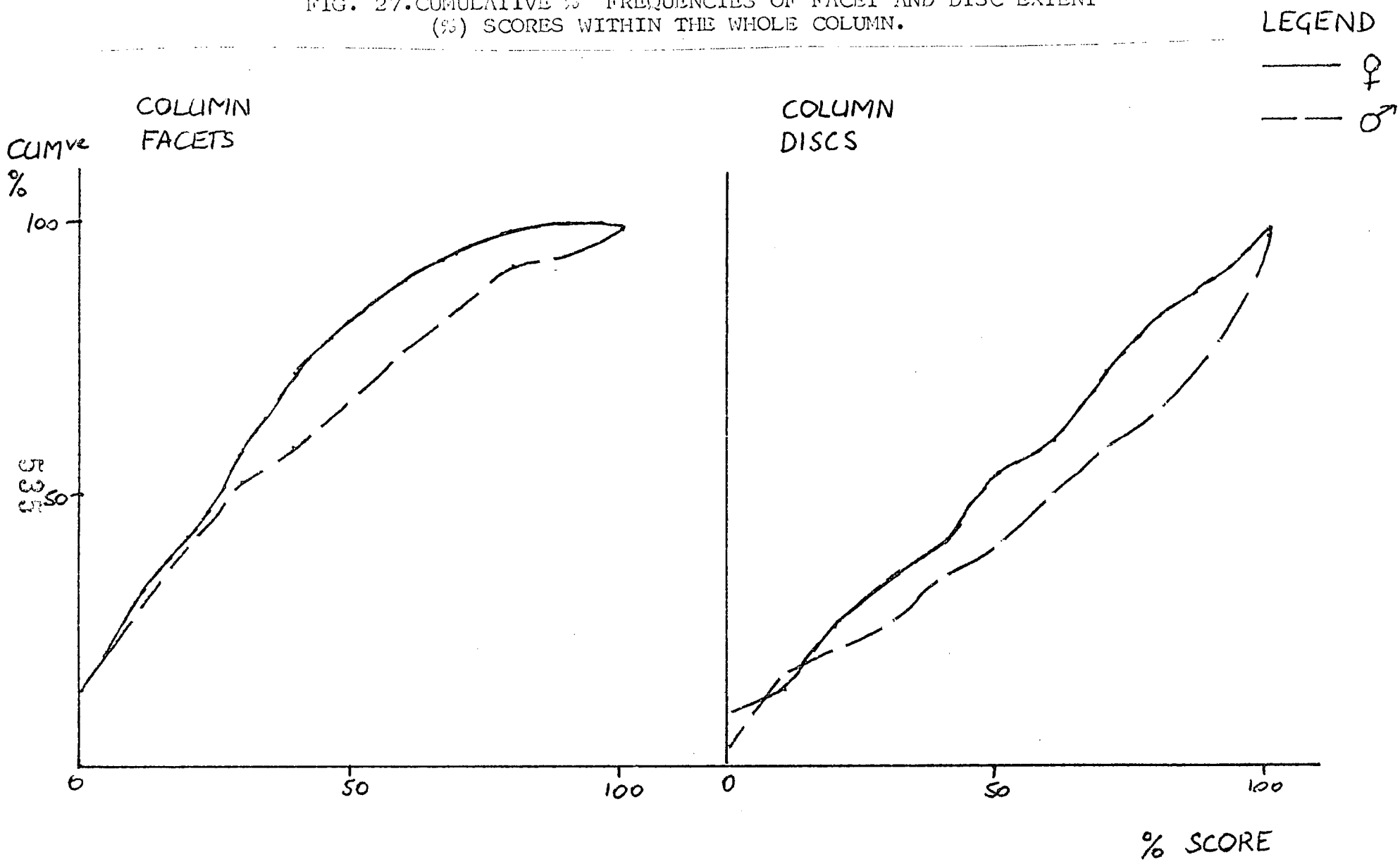


FIG. 28. CUMULATIVE % FREQUENCIES OF FACET AND DISC EXTENT (%) SCORES WITHIN THE CERVICAL REGION.

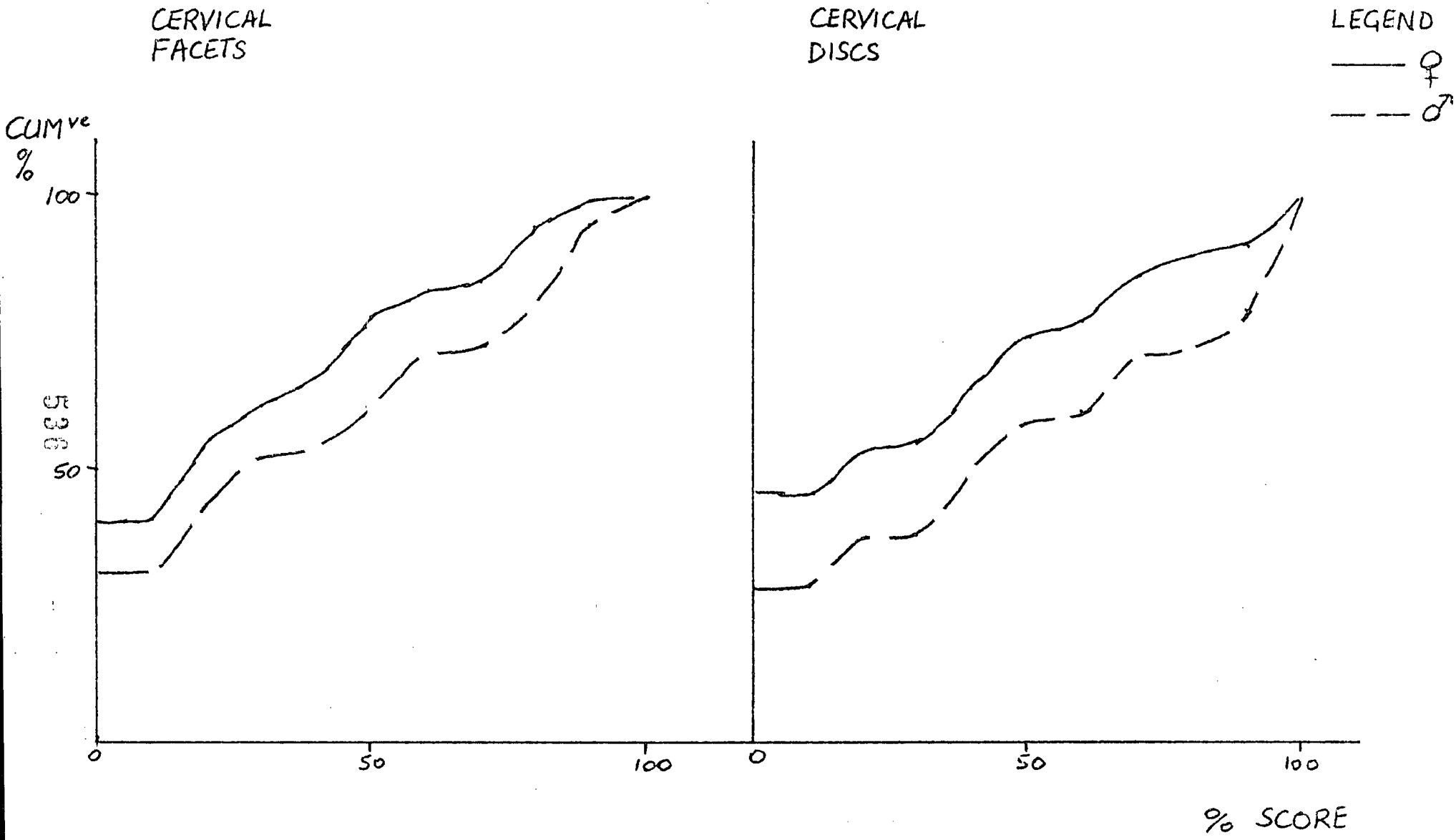


FIG. 29. CUMULATIVE % FREQUENCIES OF FACET AND DISC EXTENT (%) SCORES WITHIN THE THORACIC REGION.

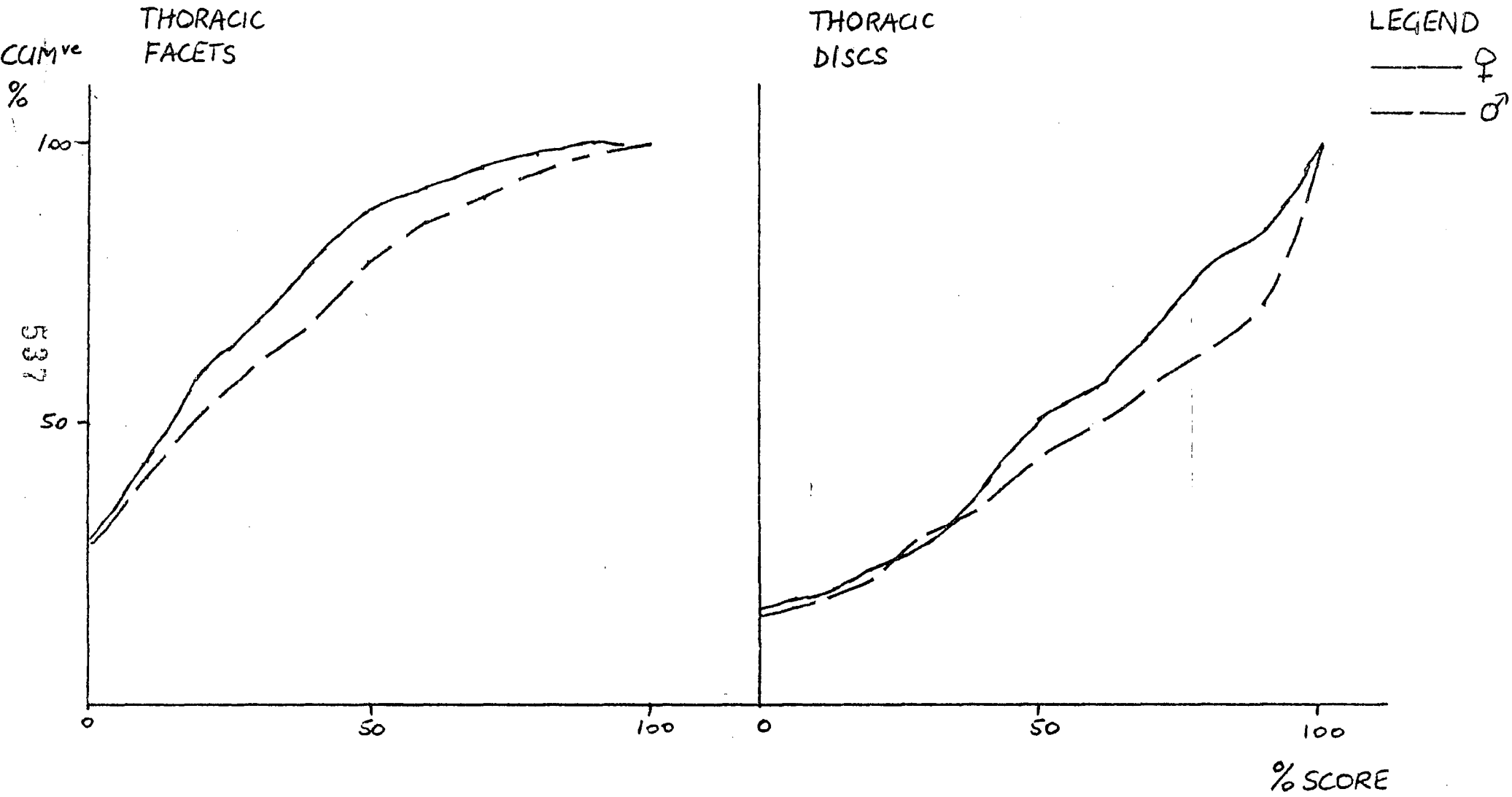


FIG. 30. CUMULATIVE % FREQUENCIES OF FACET AND DISC EXTENT (%) SCORES WITHIN THE LUMBAR REGION.

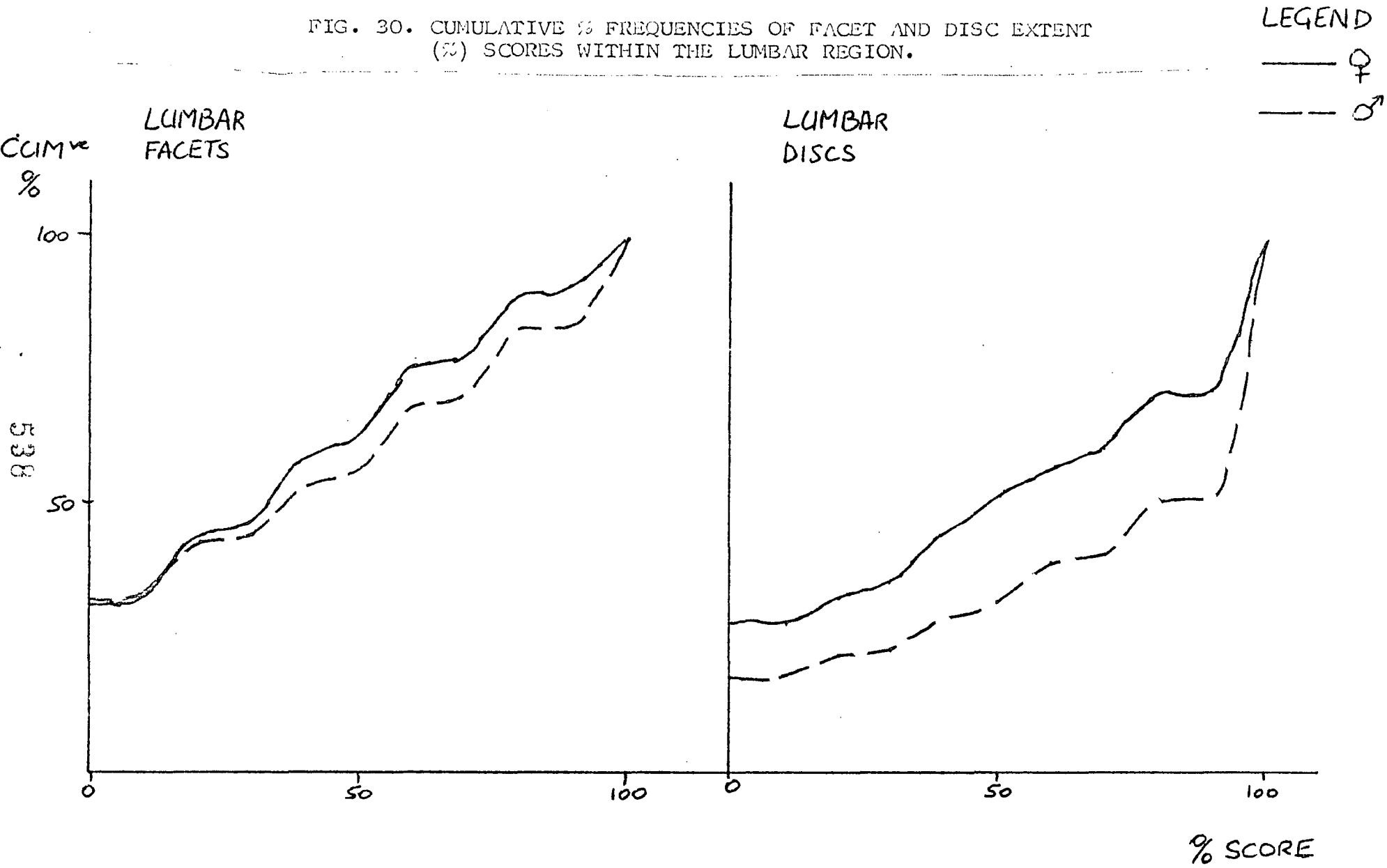


FIG. 31. CUMULATIVE % FREQUENCIES OF COMBINED EXTENT (%) SCORES WITHIN THE THORACIC AND CERVICAL REGIONS.

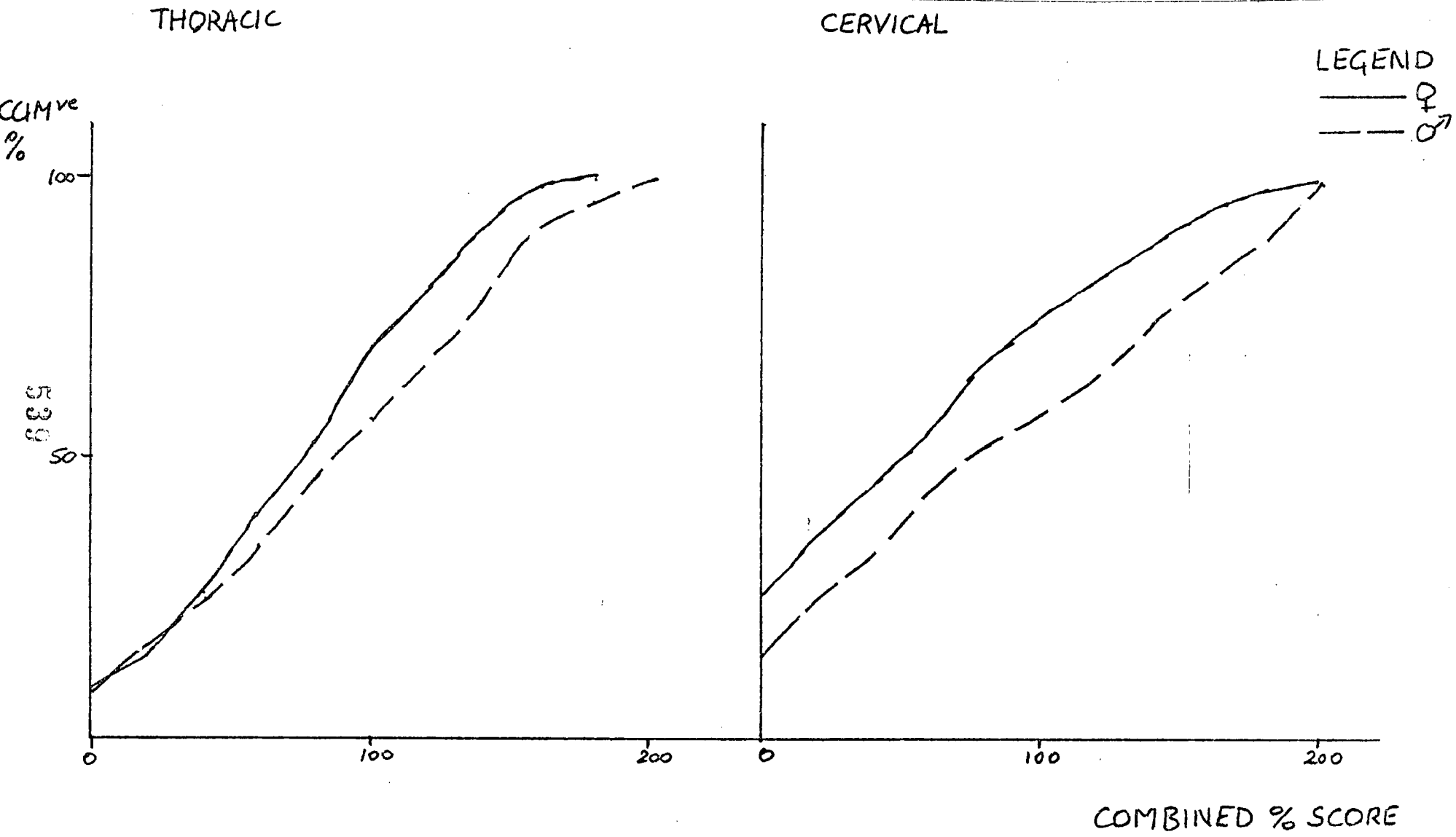


FIG. 32. CUMULATIVE % FREQUENCIES OF FACET AND DISC EXTENT (%) X MAX. GRADE SCORES WITHIN THE WHOLE COLUMN.

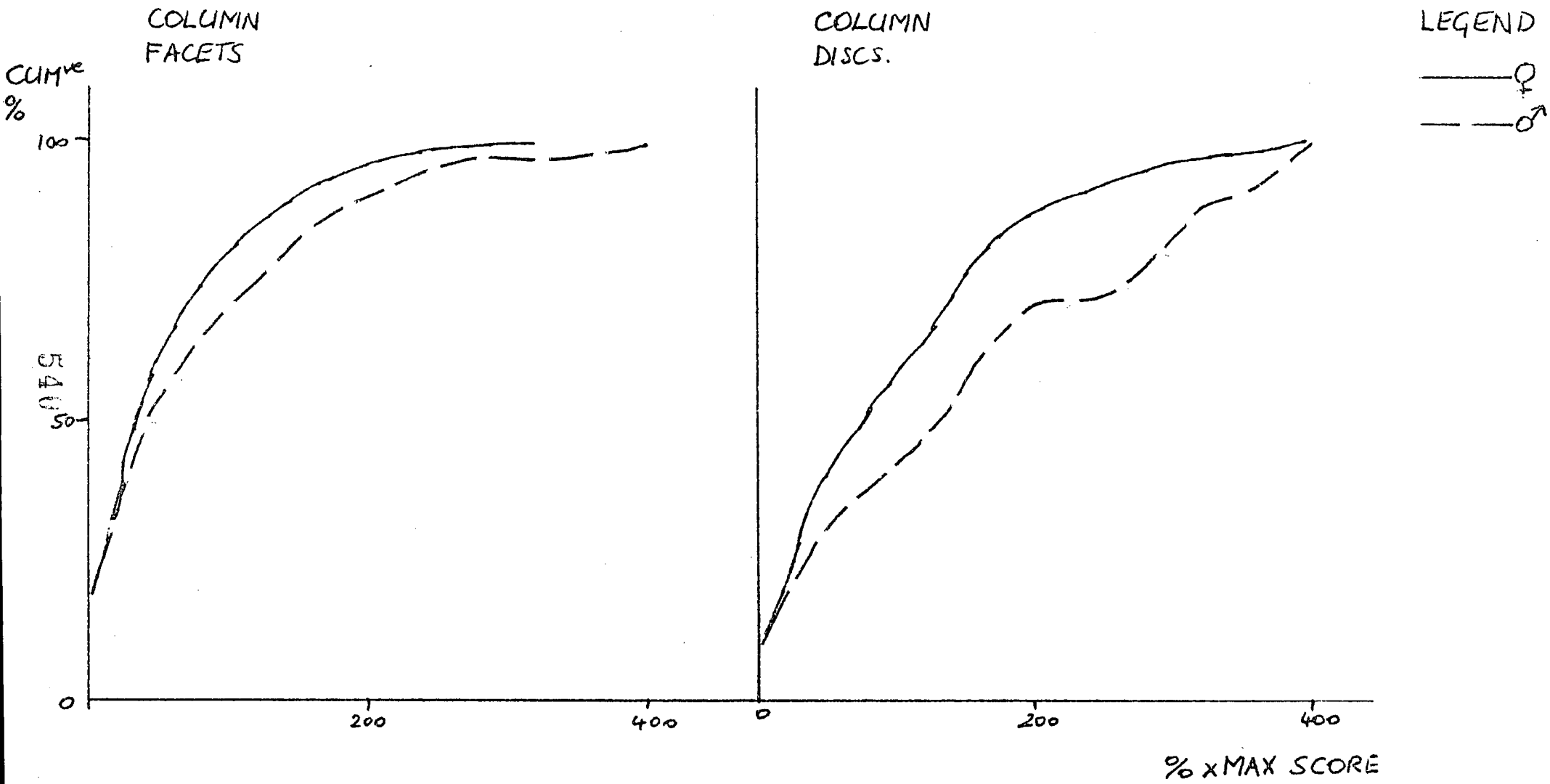


FIG. 33. CUMULATIVE % FREQUENCIES OF FACET AND DISC EXTENT (%) X MAX. GRADE SCORES WITHIN THE CERVICAL REGION.

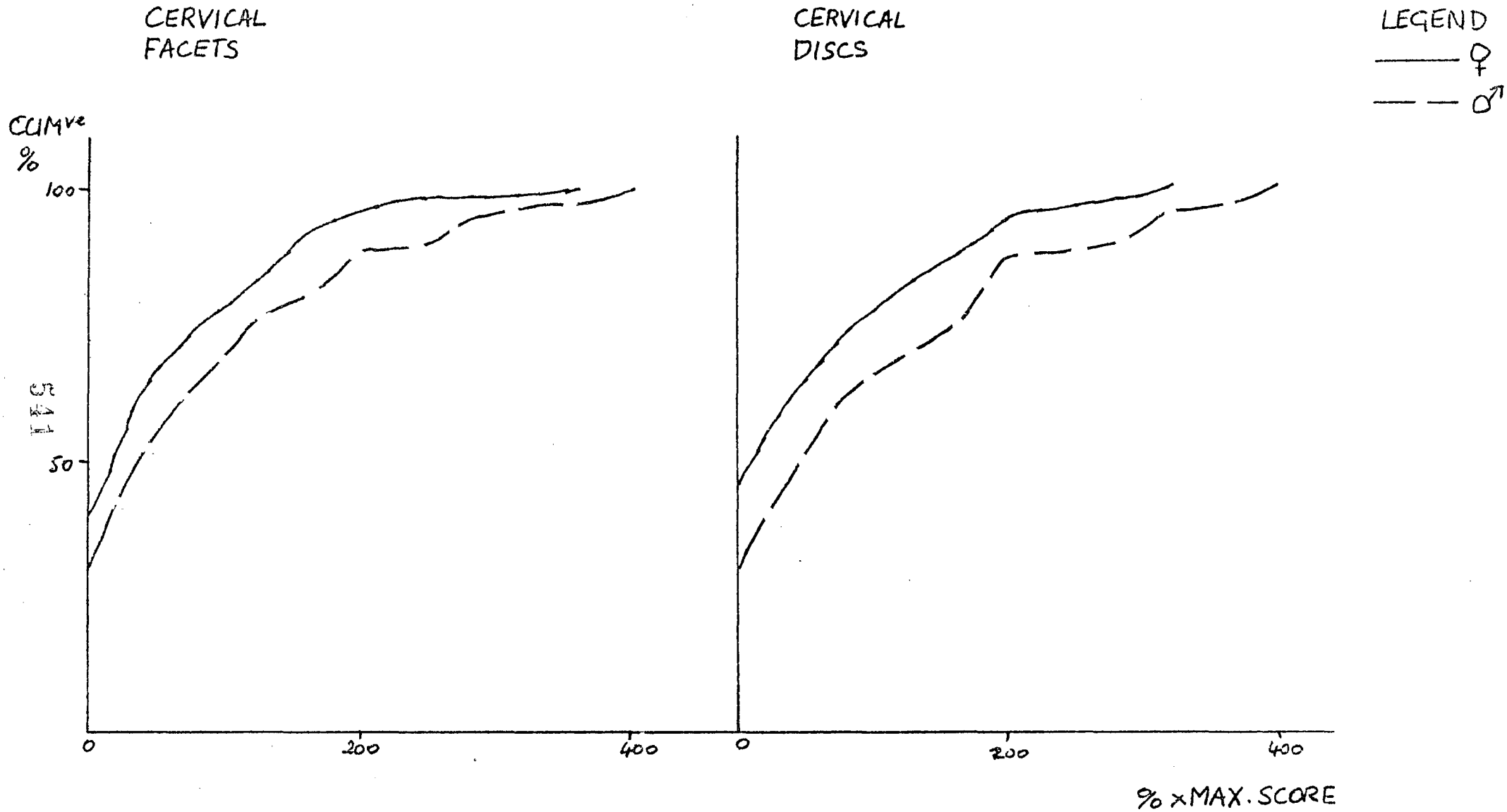


FIG. 34. CUMULATIVE % FREQUENCIES OF FACET AND DISC EXTENT (%) X MAX. GRADE SCORES WITHIN THE THORACIC REGION.

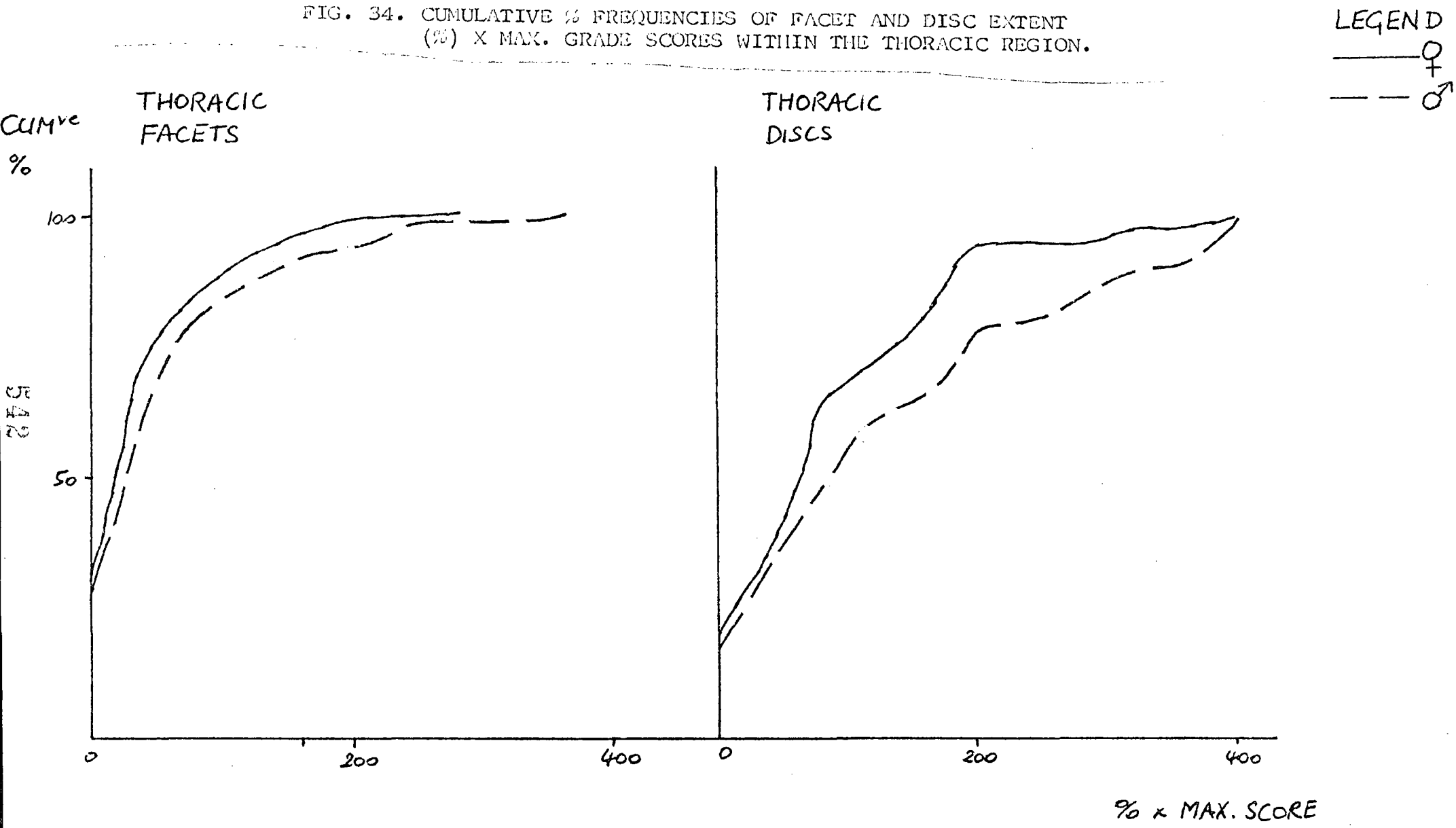


FIG. 35. CUMULATIVE % FREQUENCIES OF FACET AND DISC EXTENT (%) X MAX. GRADE SCORES WITHIN THE LUMBAR REGION.

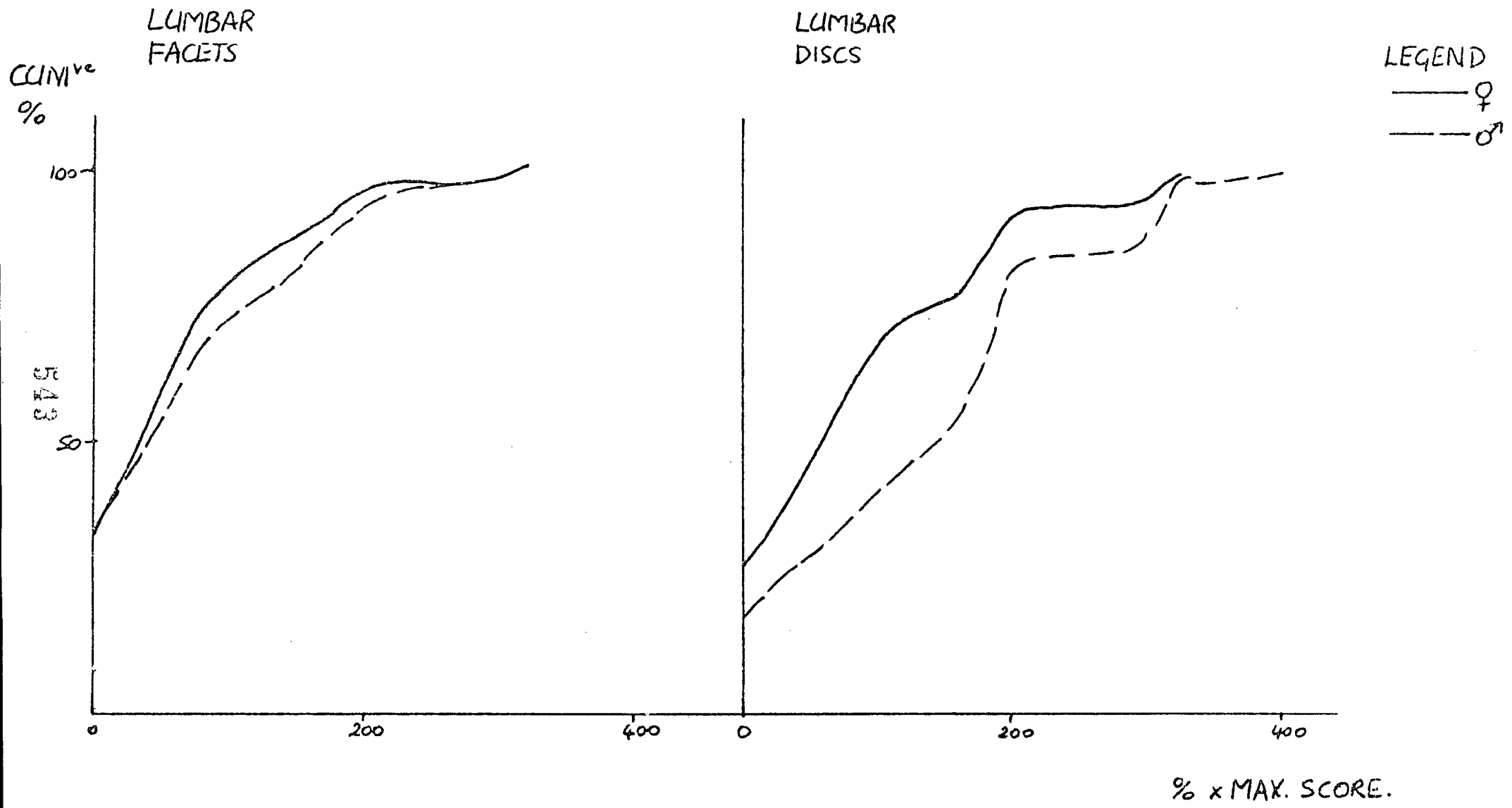
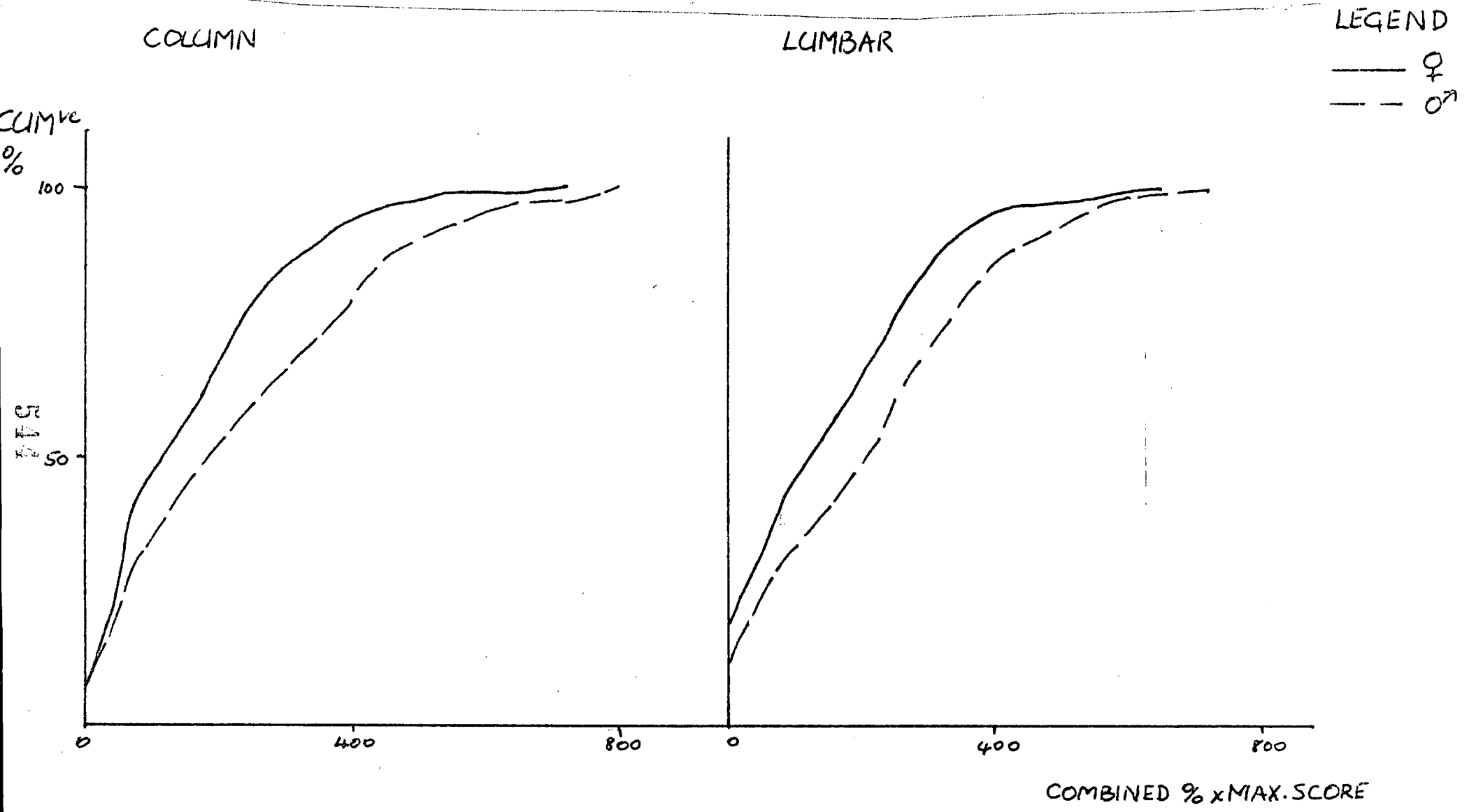
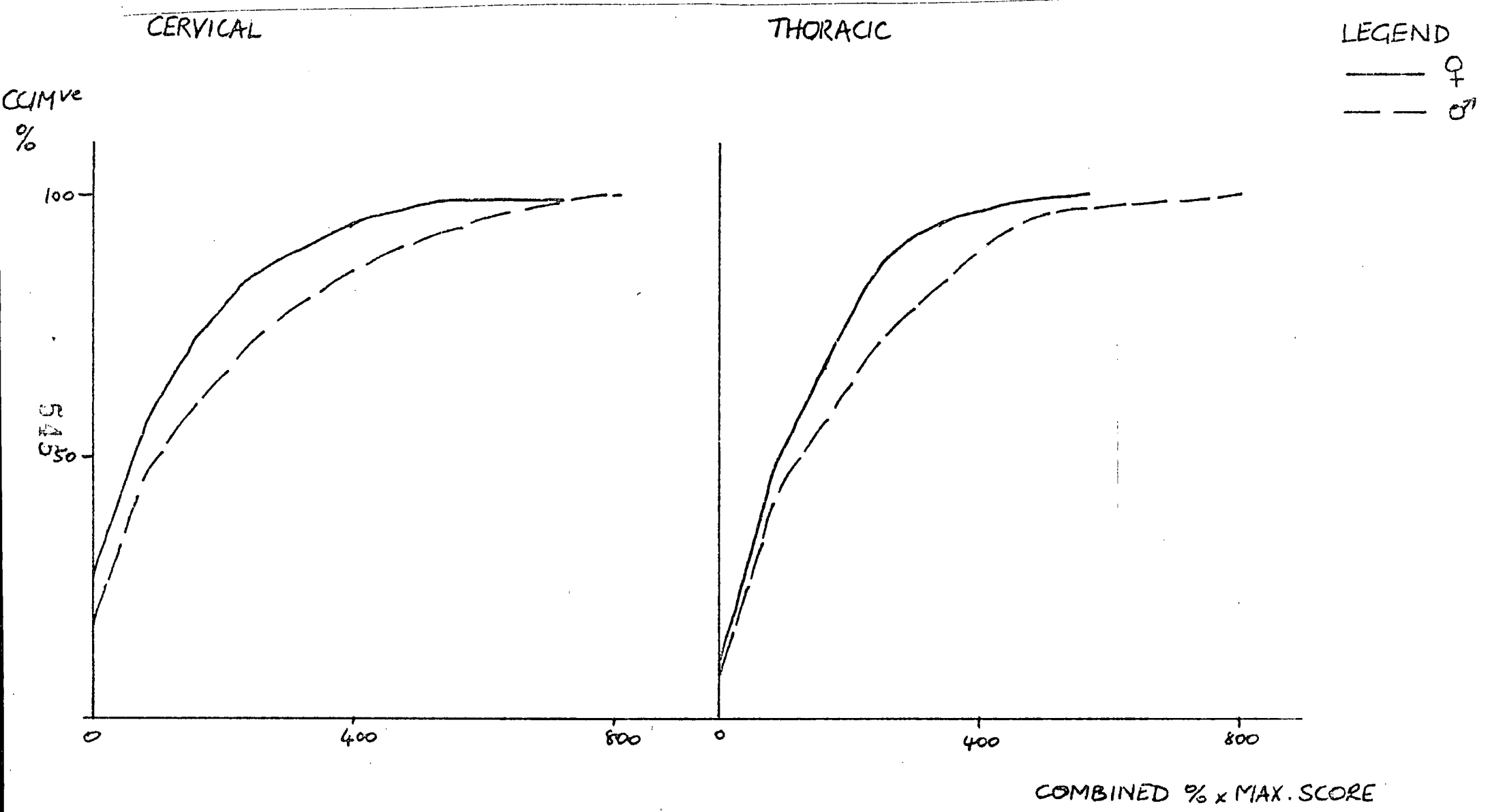


FIG. 36. CUMULATIVE % FREQUENCIES OF THE COMBINED EXTENT (%) X MAX. GRADE SCORES WITHIN THE WHOLE COLUMN AND THE LUMBAR REGION.



(%) X MAX. GRADE SCORES WITHIN THE CERVICAL AND THORACIC REGIONS.



APPENDIX D

Bone Structure and Degenerative Joint Disease.

Table 1 Correlation of Maximum Severity Spine Measures and Microage Estimates.

Microage	FACETS	Spine Measure						
	Females				Males			
	Column	Cervical	Thoracic	Lumbar	Column	Cervical	Thoracic	Lumbar
CLAGE	•2548	•2751	•2005	•2561	•1823	•1908	•0620	•0435
	•003	•002	•014	•002	•028	•029	•261	•327
ADAGE	•2232	•2781	•1470	•3420	•1595	•1198	•1177	•2280
	•013	•003	•069	•000	•050	•121	•115	•010
KAO	•1175	•1957	•0874	•2625	•1664	•2810	•0914	•1256
	•116	•027	•184	•002	•041	•003	•172	•095
KAF	•1952	•2273	•1199	•2963	•3945	•3561	•0530	•2518
	•108	•074	•219	•018	•003	•010	•359	•042
KAL	•3042	•3253	•2452	•3870	•5534	•3634	•3392	•4057
	•021	•015	•048	•002	•000	•011	•011	•002
KAN	•5842	•1943	•5492	•5241	•3647	•0928	•3647	•0707
	•011	•244	•014	•013	•167	•406	•167	•423
TAA	•2232	•2781	•1470	•3420	•1595	•1198	•1177	•2280
	•013	•003	•069	•000	•050	•121	•115	•010
TAB	•2549	•2905	•1647	•3816	•1924	•1361	•1218	•2569
	•005	•002	•048	•000	•024	•093	•108	•004
TAC	•2904	•3555	•1741	•3257	•1033	•1139	•0243	•0717
	•002	•000	•042	•000	•150	•138	•404	•238
TAD	•2127	•2403	•1195	•2333	•0250	•0857	•0435	•0302
	•018	•011	•118	•007	•401	•207	•332	•382
TAE	•2262	•2236	•0157	•3017	•0241	•0372	•0872	•0997
	•013	•017	•438	•001	•405	•362	•193	•163

Table 1 contd.

Microage	DISCS	Spine Measure						
	Females				Males			
	Column	Cervical	Thoracic	Lumbar	Column	Cervical	Thoracic	Lumbar
CLAGE	•2824	•4151	•0400	•3394	•3640	•3716	•2824	•2936
	•002	•000	•346	•000	•000	•000	•003	•002
ADAGE	•3231	•3739	•1127	•3969	•3028	•2225	•2525	•3452
	•001	•000	•151	•000	•002	•019	•007	•001
KAO	•2078	•2696	•0228	•3417	•3207	•2868	•2445	•2778
	•023	•006	•416	•000	•001	•003	•008	•004
KAF	•1118	•0768	•1838	•2729	•1199	•3021	-•0073	•1465
	•252	•321	•138	•042	•225	•031	•481	•197
KAL	•3971	•3676	•0442	•3966	•5792	•4793	•6056	•5236
	•006	•010	•396	•003	•000	•002	•000	•000
KAN	•4944	•5737	•3318	-•1219	•5582	•7180	•4341	•3870
	•043	•020	•114	•339	•096	•035	•165	•152
TAA	•3231	•3739	•1127	•3969	•3028	•2225	•2525	•3452
	•001	•000	•151	•000	•002	•019	•007	•001
TAB	•3634	•3712	•1387	•4395	•3318	•2229	•2641	•3609
	•000	•000	•100	•000	•001	•020	•005	•000
TAC	•3765	•2934	•1676	•2735	•0712	•1098	•0814	•1277
	•000	•004	•065	•005	•258	•162	•223	•126
TAD	•3142	•1658	•1798	•1612	•0461	•0304	•000	-•0239
	•002	•072	•052	•065	•337	•392	•500	•416
TAE	•3107	•1669	•1924	•2284	•0342	•0166	•0548	-•0381
	•002	•071	•041	•015	•378	•441	•305	•368

Table 1 contd.

Microage	COMBINED Spine Measure							
	Females				Males			
	Column	Cervical	Thoracic	Lumbar	Column	Cervical	Thoracic	Lumbar
CLAGE	•2722	•3751	•1593	•3211	•3570	•3131	•2710	•2119
	•003	•000	•058	•000	•000	•001	•004	•024
ADAGE	•3134	•3687	•1932	•4198	•2716	•1936	•2530	•3539
	•002	•000	•038	•000	•005	•036	•007	•000
KAO	•1841	•2833	•0997	•3616	•3104	•3229	•2190	•2641
	•041	•004	•178	•000	•001	•001	•017	•007
KAF	•1037	•1493	-•0582	•2698	•2508	•3372	•0090	•1143
	•274	•186	•368	•048	•055	•018	•477	•260
KAL	•4443	•4328	•2780	•3655	•5961	•4466	•6044	•5212
	•003	•003	•050	•008	•000	•003	•000	•000
KAN	•6053	•5531	•6941	•0519	•4231	•5255	•3884	•1750
	•019	•025	•003	•452	•172	•113	•195	•326
TAA	•3134	•3687	•1933	•4198	•2716	•1936	•2530	•3539
	•002	•000	•038	•000	•005	•005	•007	•000
TAB	•3493	•3730	•2123	•4703	•3102	•2013	•2637	•3771
	•000	•000	•026	•000	•002	•032	•006	•000
TAC	•3526	•3929	•2452	•3037	•1071	•1012	•0766	•1057
	•001	•000	•013	•002	•163	•181	•238	•175
TAD	•2781	•2505	•1986	•1812	-•0147	•0372	-•0234	-•0516
	•005	•013	•037	•047	•447	•369	•414	•325
TAE	•3085	•2296	•1469	•3000	-•0377	•0043	•0148	•0153
	•002	•022	•094	•002	•366	•485	•445	•447

Table 2 Correlation of Maximum Severity Spine Measures and Microparameters.

	FACETS	Spine Measure						
micro-parameter	Females				Males			
	Column	Cervical	Thoracic	Lumbar	Column	Cervical	Thoracic	Lumbar
KNSO	•1175	•1957	•0874	•2625	•1664	•2810	•0914	•1256
	•116	•027	•184	•002	•041	•003	•172	•095
KNOP	•1952	•2273	•1199	•2963	•3983	•3582	•0607	•2555
	•108	•074	•219	•018	•002	•010	•339	•040
KPCL	-•2817	-•3059	-•2338	-•3879	-•5534	-•3634	-•3392	-•4057
	•030	•020	•057	•002	•000	•011	•011	•002
KNNH	-•5271	-•3391	-•3977	-•4527	-•3647	-•0928	-•3647	-•0707
	•022	•108	•064	•030	•167	•406	•167	•423
ADPO	•2232	•2781	•1470	•3420	•1595	•1198	•1177	•2280
	•013	•003	•069	•000	•050	•121	•115	•010
TNSO	•1536	•3347	•1697	•2888	•2175	•2716	•2043	•1785
	•063	•001	•043	•001	•013	•004	•019	•036
TOPL	•1987	•3102	•2604	•2342	•2415	•2176	•2461	•2273
	•026	•001	•004	•007	•007	•018	•006	•011
TMOPL	-•0529	-•0544	-•0497	-•0201	•0684	-•0370	•0824	•1595
	•303	•305	•312	•417	•245	•362	•204	•055
CTHICK	-•1919	-•1324	-•0240	-•1962	-•1168	-•0524	•0034	-•0550
	•004	•043	•372	•003	•064	•265	•482	•236
AMOD	•0824	•0439	•0984	•0545	•0873	-•0876	•0339	•0411
	•188	•327	•143	•268	•180	•193	•363	•333
CNSO	•1254	•2047	•0555	•1775	•1683	•2573	•0699	•0538
	•081	•014	•267	•018	•033	•004	•226	•281

Table 2 contd.

micro-parameter	DISCS Spine Measures							
	Females				Males			
	Column	Cervical	Thoracic	Lumbar	Column	Cervical	Thoracic	Lumbar
KNSO	•2078	•2696	•0228	•3417	•3207	•2868	•2445	•2778
	•023	•006	•416	•000	•001	•003	•008	•004
KNOF	•1118	•0768	-•1838	•2729	•1208	•3021	-•0042	•1487
	•252	•321	•138	•042	•223	•031	•489	•193
KPCL	-•3941	-•3676	-•0357	-•3892	-•5792	-•4793	-•6056	-•5236
	•007	•010	•416	•004	•000	•002	•000	•000
KNNH	-•4944	-•6368	-•2802	-•0741	-•5582	-•7180	-•4341	-•3870
	•043	•010	•156	•401	•096	•035	•165	•152
ADPO	•3231	•3739	•1127	•3969	•3028	•2225	•2525	•3452
	•001	•000	•151	•000	•002	•019	•007	•001
TNSO	•2532	•2905	•0328	•3094	•2036	•2225	•1555	•2472
	•009	•004	•382	•001	•029	•020	•069	•012
TOPL	•1037	•2190	-•0698	•2652	•4321	•3835	•2812	•4363
	•173	•026	•265	•006	•000	•000	•003	•000
TNOPL	-•1748	-•0148	-•1266	•0036	•1735	•1034	•1425	•1263
	•055	•449	•127	•487	•054	•175	•089	•128
CTHICK	-•2381	-•2161	-•1733	-•2168	-•0913	-•0437	-•1024	-•0331
	•001	•004	•015	•003	•134	•307	•106	•349
AMOD	•2210	•2191	•1929	•2369	•0877	•1501	•1408	•0399
	•013	•017	•027	•007	•196	•077	•083	•354
CNSO	•1015	•2400	-•0822	•2177	•3565	•3206	•2433	•2839
	•146	•008	•200	•010	•000	•001	•006	•002

Table 2 contd.

micro-parameter	COMBINED Spine Measures							
	Females				Males			
	Column	Cervical	Thoracic	Lumbar	Column	Cervical	Thoracic	Lumbar
KNSO	•1841	•2833	•0997	•3616	•3104	•3229	•2190	•2641
	•041	•004	•178	•000	•001	•001	•017	•007
KNOF	•1037	•1493	-•0582	•2698	•2526	•3388	•0158	•1172
	•274	•186	•368	•048	•053	•017	•460	•255
KPCL	-•4189	-•4171	-•2590	-•3645	-•5961	-•4466	-•6044	-•5212
	•005	•004	•064	•008	•000	•003	•000	•000
KNNH	-•5464	-•6695	-•5477	-•1556	-•4231	-•5253	-•3884	-•1750
	•033	•006	•021	•298	•172	•113	•195	•326
ADPO	•3134	•3687	•1932	•4198	•2716	•1936	•2530	•3539
	•000	•000	•038	•000	•005	•036	•007	•000
TNSO	•2089	•3517	•1183	•3342	•2736	•2921	•2072	•2460
	•027	•001	•140	•001	•005	•003	•024	•013
TOPL	•1461	•2956	•1244	•2639	•4313	•3537	•3369	•4050
	•094	•004	•133	•007	•000	•000	•001	•000
THOPL	-•1094	-•0623	-•1036	•0127	•1335	•0690	•1530	•1859
	•162	•294	•177	•454	•109	•266	•075	•048
CTHICK	-•2346	-•1893	-•1028	-•2414	-•1323	-•0831	-•0771	-•0144
	•001	•011	•100	•001	•054	•171	•176	•434
AMOD	•1429	•1136	•1580	•1530	•0896	•0469	•1450	-•0215
	•071	•139	•059	•062	•191	•329	•078	•421
CNSO	•0993	•2455	-•0158	•2157	•3298	•3155	•2205	•2361
	•153	•007	•436	•011	•000	•001	•013	•010

Table 3 Correlation of Extent (%) Spine Measures and Microage Estimates.

FACETS	Spine Measure							
microage	Females				Males			
	Column	Cervical	Thoracic	Lumbar	Column	Cervical	Thoracic	Lumbar
CLAGE	•1590	•2330	•0963	•2149	•1484	•2082	•0295	-•0136
	•007	•008	•149	•007	•060	•019	•380	•448
ADAGE	•1788	•2408	•0699	•2595	•2098	•1601	•1303	•1585
	•038	•010	•241	•003	•015	•059	•092	•053
KAC	•1367	•1809	•0280	•2555	•1812	•2458	•0682	•1277
	•082	•038	•387	•003	•029	•007	•240	•092
KAP	-•1515	•2326	-•0565	•1771	•1462	•2853	•2326	•1273
	•163	•052	•361	•131	•158	•034	•054	•194
KAL	•3964	•3926	•2491	•3760	•5236	•4477	•3943	•3607
	•004	•004	•046	•002	•000	•002	•003	•006
KAN	•4742	•1751	•3801	•4739	•3149	•1309	•2639	•1449
	•037	•266	•073	•023	•205	•369	•246	•345
TAA	•1788	•2408	•0699	•2595	•2098	•1601	•1303	•1585
	•038	•010	•241	•003	•015	•059	•092	•053
TAB	•2024	•2571	•0825	•2905	•1999	•1544	•1270	•1452
	•022	•006	•204	•001	•020	•067	•098	•071
TAC	•2388	•3480	•0475	•2467	•0761	•0487	•0934	-•0752
	•009	•000	•319	•005	•225	•313	•187	•226
TAD	•2485	•2455	•0126	•1832	-•0059	-•0131	•0769	-•1387
	•009	•009	•451	•036	•477	•448	•232	•082
TAE	•0902	•2248	-•1162	•1621	•0621	•0365	•1038	-•1157
	•190	•017	•125	•045	•271	•358	•162	•125

Table 3 contd.

DISCS	Spine Measure							
microage	Females				Males			
	Column	Cervical	Thoracic	Lumbar	Column	Cervical	Thoracic	Lumbar
CLAGE	•2964	•4225	•1050	•3025	•3548	•3504	•2557	•3199
	•001	•000	•149	•001	•000	•000	•006	•001
ADAGE	•4043	•3871	•1727	•4387	•3449	•2207	•2808	•4125
	•000	•000	•056	•000	•000	•020	•003	•000
KAO	•2302	•2502	•1132	•2796	•2852	•2580	•1923	•2838
	•014	•010	•145	•003	•003	•008	•031	•004
KAF	•0670	•0946	-•1846	•2541	•2212	•3398	•1525	•1103
	•345	•283	•137	•070	•079	•017	•164	•261
KAL	•3901	•3681	•1313	•4156	•6817	•5604	•6828	•6351
	•007	•010	•216	•000	•000	•000	•000	•000
KAN	•5519	•6325	•1941	-•0196	•5929	•7416	•5112	•2961
	•025	•010	•244	•474	•080	•028	•120	•220
TAA	•4043	•3871	•1727	•4387	•3449	•2207	•2808	•4125
	•000	•000	•056	•000	•000	•020	•003	•000
TAB	•4444	•3934	•2097	•4723	•3556	•2232	•2899	•4352
	•000	•000	•026	•000	•000	•019	•002	•000
TAC	•3456	•2508	•2167	•2893	•1808	•1615	•0727	•2056
	•001	•013	•025	•003	•048	•072	•248	•032
TAD	•2235	•1187	•1563	•1710	•1009	•0991	•0268	•0382
	•020	•149	•079	•054	•128	•186	•401	•367
TAE	•2650	•1994	•1064	•2665	•1181	•0227	•1431	•1204
	•007	•039	•169	•006	•141	•420	•090	•142

Table 3 contd.

	COMBINED				Spine Measure			
microage	Females				Males			
	Column	Cervical	Thoracic	Lumbar	Column	Cervical	Thoracic	Lumbar
CLAGE	•2710	•3621	•1338	•2586	•3130	•3217	•1746	•1535
	•003	•000	•093	•004	•001	•001	•044	•077
ADAGE	•3260	•3527	•1596	•4215	•3179	•1987	•2548	•3273
	•001	•001	•072	•000	•001	•033	•007	•001
KAO	•2497	•2730	•1207	•2892	•2960	•2669	•1686	•2254
	•009	•006	•131	•002	•002	•006	•052	•018
KAF	-•0333	•1066	-•2392	•2470	•1051	•3295	•1503	-•0111
	•424	•262	•080	•065	•254	•020	•171	•475
KAL	•4525	•4805	•3151	•3422	•6550	•5205	•6442	•5222
	•003	•001	•031	•012	•000	•001	•000	•000
KAN	•6116	•5920	•3289	•0863	•3706	•6732	•4447	-•0474
	•017	•017	•125	•385	•207	•049	•159	•452
TAA	•3260	•3527	•1596	•4215	•3179	•1987	•2548	•3273
	•001	•001	•072	•000	•001	•033	•007	•001
TAB	•3674	•3712	•1952	•4541	•3167	•1940	•2582	•3363
	•000	•000	•037	•000	•001	•037	•006	•001
TAC	•3167	•3746	•1879	•2718	•1429	•1123	•0169	•1582
	•002	•000	•045	•005	•095	•156	•437	•081
TAD	•2109	•2379	•1213	•1419	•0547	•0715	-•0548	•0195
	•028	•018	•139	•095	•308	•260	•305	•432
TAE	•1821	•2510	-•0032	•2500	•0719	•0652	•0421	•1171
	•050	•013	•488	•010	•256	•280	•348	•152

Table 4 Correlation of Extent(%) Spine Measure and the Microparameters.

micro-parameter	FACETS Spine Measure							
	Females				Males			
	Column	Cervical	Thoracic	Lumbar	Column	Cervical	Thoracic	Lumbar
KNSO	•1367	•1809	•0280	•2555	•1812	•2458	•0682	•1277
	•082	•038	•387	•003	•029	•007	•240	•092
KNOF	-•0565	•1771	-•1515	•2326	•1529	•2878	•2400	•1306
	•361	•131	•163	•052	•147	•032	•048	•188
KPCL	-•3834	-•3759	-•2459	-•3785	-•5236	-•4477	-•3943	-•3607
	•005	•005	•048	•002	•000	•002	•003	•006
KNNH	-•4670	-•3229	-•2876	-•3747	-•3149	-•1309	-•2639	-•1449
	•040	•120	•140	•063	•205	•369	•246	•345
ADPO	•1788	•2408	•0699	•2595	•2098	•1601	•1303	•1585
	•038	•010	•241	•003	•015	•059	•092	•053
TNSO	•2243	•3270	•1129	•2721	•2045	•1931	•1140	•2011
	•012	•001	•127	•002	•018	•030	•125	•021
TOPL	•2786	•2733	•2666	•2815	•2553	•1887	•2208	•2019
	•003	•005	•004	•001	•004	•034	•012	•021
TMOPL	-•0950	-•0773	-•0012	-•0165	•1274	•0327	•1852	•0976
	•177	•235	•495	•432	•099	•377	•031	•165
CTHICK	-•0940	-•0910	-•0150	-•1384	•0081	-•0241	•2092	•0251
	•102	•120	•419	•024	•458	•386	•353	•371
AMOD	•0012	•0162	•0286	•0195	-•0149	-•0376	-•0287	-•0753
	•495	•434	•378	•413	•438	•355	•383	•217
CNSO	•0721	•1592	-•0161	•1805	•1599	•2472	•0538	•0328
	•211	•045	•429	•016	•040	•005	•281	•362

Table 4 contd.

DISCS micro-parameter	Spine Measure: Females Column	Females Cervical	Females Thoracic	Females Lumbar	Males Column	Males Cervical	Males Thoracic	Males Lumbar
KNSO	•2302	•2502	•1132	•2302	•2852	•2580	•1923	•2838
	•014	•010	•145	•014	•003	•008	•031	•004
KNOF	•0670	•0946	-•1846	•0670	•2228	•3398	•1556	•1117
	•345	•283	•137	•345	•078	•017	•160	•258
KPCL	-•3838	-•3681	-•1052	-•3838	-•6871	-•5604	-•6828	-•6351
	•008	•010	•265	•008	•000	•000	•000	•000
KNNH	-•5740	-•6891	-•1905	-•5740	-•5929	-•7416	-•5112	-•2961
	•020	•005	•248	•020	•080	•028	•120	•220
ADPO	•4043	•3871	•1727	•4387	•3449	•2207	•2808	•4125
	•000	•000	•056	•000	•000	•020	•003	•000
TNSO	•2337	•2691	•1043	•2822	•2523	•2323	•1500	•3015
	•014	•007	•170	•003	•009	•016	•077	•003
TOPL	•1635	•1875	•0674	•2491	•3761	•3665	•2237	•3916
	•067	•049	•272	•009	•000	•000	•017	•000
TMOPL	-•0439	•0706	-•0955	•0258	•0610	•0270	•1193	•1437
	•345	•268	•195	•405	•287	•404	•130	•097
CTHICK	-•3095	-•2660	-•2173	-•2210	-•1286	-•0427	-•1782	-•1258
	•000	•000	•003	•002	•059	•311	•015	•069
AMOD	•3036	•2615	•2073	•2861	•1030	•0845	•1711	•0665
	•001	•005	•019	•002	•158	•212	•046	•266
CNSO	•0782	•2196	-•0229	•1085	•3126	•3281	•1832	•2880
	•208	•013	•408	•125	•001	•001	•031	•002

Table 4 contd.

micro-parameter	COMBINED	Females			Males			
	Spine Measure							
	Column	Cervical	Thoracic	Lumbar	Column	Cervical	Thoracic	Lumbar
KNSO	•2497	•2730	•1207	•2892	•2960	•2669	•1686	•2254
	•009	•006	•131	•002	•002	•006	•052	•018
KNOF	-•0333	•1066	-•2392	•2470	•1073	•3323	•1572	-•0090
	•424	•262	•080	•065	•249	•019	•160	•480
KPCL	-•4415	-•4663	-•2885	-•3479	-•6550	-•5205	-•6442	-•5222
	•004	•001	•044	•011	•000	•001	•000	•000
KNNH	-•5835	-•7023	-•3113	-•1369	-•3706	-•6732	-•4447	-•0474
	•023	•004	•139	•320	•207	•049	•159	•452
ADPO	•3260	•3527	•1596	•4215	•3179	•1987	•2548	•3273
	•001	•001	•072	•000	•001	•033	•007	•001
TNSO	•2322	•3562	•0812	•2748	•3135	•2378	•1937	•2797
	•016	•001	•230	•004	•001	•014	•033	•005
TOPL	•2699	•2523	•1870	•2826	•4000	•3030	•2855	•3055
	•004	•013	•046	•004	•000	•003	•003	•003
TMOPL	-•0884	-•0350	-•0807	•0475	•0952	•0530	•1832	•1388
	•192	•381	•235	•331	•190	•316	•042	•108
CTHICK	-•1517	-•2204	-•1746	-•1992	-•0585	-•0557	-•0901	-•0326
	•020	•004	•014	•006	•240	•262	•138	•352
AMOD	•0421	•1097	•1890	•1710	•0412	•0415	•0700	-•0449
	•325	•148	•030	•043	•344	•348	•248	•338
CNSO	•1035	•2174	-•0376	•1226	•3028	•3132	•1528	•1728
	•124	•014	•351	•100	•001	•001	•062	•046

Table 5 Correlation Between Extent (%) x Maximum Severity Spine Measures and Microages.

	FACETS	Spine Measure						
microage	Females				Males			
	Column	Cervical	Thoracic	Lumbar	Column	Cervical	Thoracic	Lumbar
CLAGE	•2177	•2579	•1281	•2474	•1658	•2128	•0574	•0134
	•009	•004	•082	•002	•041	•017	•277	•445
ADAGE	•2110	•2595	•0990	•2917	•2077	•1667	•1300	•2121
	•018	•000	•160	•001	•016	•051	•092	•015
KAO	•1490	•1886	•0414	•2721	•1799	•2776	•0931	•1304
	•065	•032	•335	•001	•030	•003	•168	•087
KAF	•0365	•1833	-•0579	•2833	•2720	•3668	•1948	•1799
	•409	•123	•354	•023	•029	•008	•090	•111
KAL	•3732	•3839	•2702	•3696	•5614	•4465	•4287	•4137
	•006	•005	•033	•003	•000	•002	•001	•002
KAN	•4742	•2002	•4261	•4762	•3830	•1309	•4511	•1449
	•037	•237	•050	•023	•154	•369	•111	•345
TAA	•2110	•2595	•0990	•2917	•2077	•1667	•1300	•2121
	•018	•006	•160	•001	•016	•051	•092	•015
TAB	•2432	•2748	•1127	•3299	•2149	•1702	•1322	•2220
	•007	•004	•129	•000	•013	•049	•089	•012
TAC	•2857	•3513	•0910	•2708	•0865	•0941	-•0358	•1032
	•002	•000	•184	•002	•192	•185	•361	•152
TAD	•2156	•2429	•0526	•1681	•0151	•0594	•1056	•0026
	•017	•011	•301	•039	•440	•286	•145	•490
TAE	•1550	•2340	-•0698	•2059	•0450	•0633	-•1132	•0914
	•065	•013	•245	•015	•327	•274	•130	•184

Table 5 contd.

	DISCS	Spine Measure						
microage		Females				Males		
	Column	Cervical	Thoracic	Lumbar	Column	Cervical	Thoracic	Lumbar
CLAGE	•3218	•4187	•0891	•3269	•3816	•3514	•2889	•3209
	•000	•000	•189	•000	•000	•000	•002	•001
ADAGE	•4006	•3760	•1676	•4228	•3337	•2143	•2735	•3828
	•000	•000	•062	•000	•001	•023	•004	•000
KAO	•2379	•2521	•0731	•3109	•3188	•2561	•2176	•2930
	•011	•010	•248	•001	•001	•008	•017	•003
KAF	•0938	•0587	-•2101	•2628	•1763	•3390	•0404	•1584
	•288	•361	•106	•048	•132	•017	•399	•178
KAL	•4561	•3469	•1222	•4291	•6424	•5298	•6505	•5934
	•002	•014	•232	•002	•000	•000	•000	•000
KAN	•5712	•6010	•2603	-•0192	•5929	•7416	•5112	•3171
	•021	•015	•174	•474	•080	•028	•120	•203
TAA	•4006	•3760	•1676	•4228	•3337	•2143	•2735	•3838
	•000	•000	•062	•000	•001	•023	•004	•000
TAB	•4446	•3795	•2055	•4612	•3549	•2169	•2857	•4057
	•000	•000	•029	•000	•000	•022	•003	•000
TAC	•3875	•2807	•2202	•2906	•1199	•1357	•0804	•1822
	•000	•006	•023	•003	•136	•111	•226	•051
TAD	•2754	•1522	•1823	•1748	•0155	•0685	•0134	•0197
	•005	•090	•051	•050	•443	•269	•450	•430
TAE	•3035	•1839	•1617	•2623	•0355	•0062	•0945	•0234
	•002	•052	•072	•006	•374	•478	•189	•418

Table 5 contd.

	COMBINED				Spine Measure			
microage		Females				Males		
	Column	Cervical	Thoracic	Lumbar	Column	Cervical	Thoracic	Lumbar
CLAGE	•3029	•3608	•1719	•3005	•3460	•3091	•2732	•1952
	•001	•000	•044	•001	•000	•002	•004	•034
ADAGE	•3310	•3453	•1848	•4045	•3197	•1876	•3072	•3923
	•001	•001	•045	•000	•001	•041	•001	•000
KAO	•2319	•2690	•1058	•4054	•3231	•2812	•2221	•2642
	•014	•006	•163	•000	•001	•004	•016	•007
KAF	•0275	•1385	-•1557	•3113	•1866	•3635	•0711	•0405
	•437	•204	•182	•001	•118	•011	•327	•410
KAL	•4627	•4678	•3812	•2706	•6148	•5153	•6375	•5335
	•002	•001	•011	•048	•000	•001	•000	•000
KAN	•6643	•6592	•5651	•3391	•4447	•6732	•4447	-•0043
	•009	•007	•018	•013	•159	•049	•159	•496
TAA	•3310	•3453	•1848	•4054	•3197	•1876	•3072	•3923
	•001	•001	•045	•000	•001	•041	•001	•000
TAB	•3734	•3618	•2170	•4477	•3429	•1889	•3145	•4129
	•000	•000	•023	•000	•001	•041	•001	•000
TAC	•3456	•3997	•2194	•2589	•1242	•0954	•0843	•1842
	•001	•000	•024	•008	•127	•196	•216	•051
TAD	•2476	•2621	•1617	•1330	•0056	•0468	-•0148	•0176
	•012	•010	•073	•110	•480	•337	•445	•439
TAE	•2423	•2527	•0810	•2720	•0234	•0411	•0734	•0621
	•014	•013	•235	•005	•416	•357	•248	•293

Table 6 Correlation Between Extent (%) x Maximum Severity Spine Measures and the Microparameters.

micro-parameter	FACETS Spine Measure							
	Females				Males			
	Column	Cervical	Thoracic	Lumbar	Column	Cervical	Thoracic	Lumbar
KNSO	•1490	•1886	•0414	•2721	•1799	•2776	•0931	•1304
	•065	•032	•335	•001	•030	•003	•168	•087
KNOF	•0365	•1833	-•0579	•2833	•2787	•3697	•2020	•1829
	•409	•123	•354	•023	•026	•008	•082	•107
KPCL	-•3533	-•3645	-•2637	-•3724	-•5614	-•4465	-•4287	-•4137
	•009	•007	•037	•003	•000	•002	•001	•002
KNNH	-•4670	-•3418	-•3274	-•3819	-•3830	-•1309	-•4511	-•1449
	•040	•106	•108	•059	•154	•369	•111	•345
ADPO	•2110	•2595	•0990	•2917	•2077	•1667	•130	•2121
	•018	•006	•160	•001	•016	•051	•092	•015
TNSO	•2204	•3292	•1208	•2771	•2244	•2353	•1599	•2079
	•013	•001	•111	•001	•011	•014	•011	•052
TOPL	•2699	•2872	•2674	•2731	•2641	•2257	•2539	•2367
	•004	•003	•004	•002	•003	•014	•005	•009
TMOPL	-•0894	-•0590	-•0168	-•0026	•1092	•0330	•1492	•1207
	•192	•290	•434	•489	•135	•376	•066	•113
CTHICK	-•1517	-•1156	-•0259	-•1922	-•0433	-•0430	-•0196	-•0093
	•020	•067	•362	•003	•287	•303	•400	•452
AMOD	•0421	•0409	•0479	•0417	•0170	-•0527	-•0048	-•0380
	•325	•338	•302	•318	•429	•301	•480	•347
CNSO	•1035	•1760	-•0002	•1847	•1707	•2661	•0739	•0505
	•124	•030	•499	•014	•031	•003	•213	•293

Table 6 contd.

micro-parameter	DISCS	Spine Measure						
	Females				Males			
	Column	Cervical	Thoracic	Lumbar	Column	Cervical	Thoracic	Lumbar
KNSO	·2379	·2521	·0731	·3109	·3188	·2561	·2176	·2930
	·011	·010	·248	·001	·001	·008	·017	·003
KNOF	·0938	·0587	-·2101	·2628	·1774	·3390	·0431	·1597
	·288	·361	·106	·048	·131	·017	·392	·176
KPCL	-·4513	-·3469	-·0961	-·4253	-·6424	-·5298	-·6505	-·5934
	·002	·014	·283	·002	·000	·000	·000	·000
KNNH	-·5823	-·6576	-·2567	-·1469	-·5929	-·7416	-·5112	-·3171
	·018	·007	·178	·308	·080	·028	·120	·203
ADPO	·4006	·3760	·1676	·4228	·3337	·2143	·2735	·3838
	·000	·000	·062	·000	·001	·023	·004	·000
TNSO	·2625	·2798	·0761	·2939	·2349	·2153	·1561	·2928
	·007	·005	·243	·002	·014	·011	·069	·003
TOPL	·1541	·2021	·0122	·2541	·4254	·3644	·2697	·4485
	·080	·037	·456	·008	·000	·000	·005	·000
TMOPL	-·1019	·0179	-·1147	·0147	·1345	·0614	·1384	·1214
	·177	·438	·151	·445	·107	·289	·095	·137
CTHICK	-·3121	-·2430	-·2431	-·2284	-·1085	-·0470	-·1526	-·0792
	·000	·001	·001	·002	·094	·294	·031	·176
AMOD	·2955	·2514	·2282	·2733	·1065	·1082	·1926	·0421
	·001	·007	·011	·002	·149	·152	·029	·346
CNSO	·0914	·2225	-·0629	·1570	·3453	·3167	·2118	·2995
	·171	·012	·260	·048	·000	·001	·015	·001

Table 6 contd.

micro-parameter	COMBINED	Females			Males			
	Spine Measure							
	Column	Cervical	Thoracic	Lumbar	Column	Cervical	Thoracic	Lumbar
KNSO	•2319	•2690	•1058	•3113	•3231	•2812	•2221	•2642
	•014	•006	•163	•001	•001	•004	•016	•007
KNOF	•0275	•1385	-•1557	•2706	•1888	•3662	•0778	•0423
	•437	•204	•182	•048	•116	•011	•312	•406
KPCL	-•4474	-•4510	-•3518	-•3463	-•6148	-•5153	-•6375	-•5335
	•003	•002	•018	•011	•000	•001	•000	•000
KNNH	-•6222	-•7519	-•5166	-•1723	-•4447	-•6732	-•4447	-•0043
	•015	•002	•029	•278	•159	•049	•159	•496
ADPO	•3310	•3453	•1848	•4054	•3197	•1876	•3072	•3923
	•001	•001	•045	•000	•001	•041	•001	•000
TNSO	•2353	•3492	•0779	•2806	•2874	•2507	•2085	•3012
	•015	•001	•239	•004	•003	•010	•024	•003
TOPL	•2020	•2674	•1502	•2475	•4406	•3060	•3426	•4234
	•034	•009	•089	•010	•000	•002	•000	•000
TMOPL	-•0934	-•0618	-•0945	•0597	•1521	•0686	•1778	•1488
	•196	•296	•199	•291	•080	•267	•047	•092
CTHICK	-•2651	-•2226	-•1982	-•2194	-•1075	-•0787	-•0965	-•0252
	•000	•003	•006	•003	•097	•184	•122	•385
AMOD	•1856	•1151	•2211	•1712	•0579	•0511	•1348	•0602
	•032	•136	•014	•043	•287	•315	•094	•288
CNSO	•0967	•2109	-•0365	•1615	•3354	•3040	•2206	•2284
	•160	•017	•355	•045	•000	•001	•013	•013

Table 7 Mean Values of the Microparameters in each Category of Combined Extent (%) Spine Measure of the Column.

micro-parameter	Spine Categories: Females 0	1	2	3	4	Males 0	1	2	3	4
KNSO	93·7	99·4	106·9	111·3	108·5	98·7	94·4	110·6	118·3	114·1
KNOF	40·	41·1	40·7	43·9	49	-	46·5	49·5	55·6	56·7
KPCL	25·	52·5	28·8	22·3	28·0	32·0	43·2	25·9	14·5	11·4
KNNH	-	34·8	13·3	5·0	-	10·0	23·0	-	10·0	-
ADPO	57·7	46·9	56·2	62·1	57·5	58·3	45·8	55·4	58·6	59·1
TNSO	5·7	5·6	6·0	6·5	6·0	6·3	5·1	5·6	6·2	6·4
TOPL	7·3	7·5	9·0	8·7	8·6	6·6	6·9	7·8	8·7	8·6
TMOPL	1·5	1·5	1·5	1·4	1·2	1·2	1·4	1·5	1·4	1·4
CTHICK	3·1	3·2	2·8	2·8	3·0	3·7	3·8	3·7	3·7	3·6
AMOD	45·3	44·5	46·3	48·6	44·0	45·7	45·0	45·9	45·3	46·4
CNSO	12·3	12·4	12·9	12·7	13·7	12·0	12·0	13·4	14·3	14·5

Table 8 Correlations of Continuous Age-Related Measurements with the Rotated Factors in Females.

Variable	FACTOR 1	2	3	4	5	6	7
% Thoracic disc	•9155	-•0185	•1223	•2084	•1264	-•0199	-•0965
% Column disc	•8524	•1542	•2865	•2976	•0807	-•0120	•1854
% x max Thoracic disc	•8498	•0052	•2783	•1328	•0842	•0186	-•0437
% x max Column disc	•7618	•1194	•4729	•2234	•0232	•0470	•2394
% combined Thoracic	•7451	-•0026	•2424	•2342	•5386	-•0382	-•0114
% x max combined Thoracic	•7295	•0190	•3863	•1420	•4295	•0210	•0650
% combined Column	•6579	•1281	•4385	•4301	•3772	•0076	•0884
% Lumbar disc	•6453	•1900	•1340	•5506	•0263	-•0094	•2495
% Cervical disc	•5849	•1940	•5553	•1888	•0421	•0308	•2688
% x max Lumbar disc	•5353	•1746	•2814	•5244	-•0845	•0006	•3136
Pubic Symphysis score	•4514	•3302	-•0854	•4509	•2721	•0580	-•1536
CNSO	-•0311	•9239	•0513	•0386	-•0274	•1659	-•0833
TNSO	•0544	•8945	•0625	•1337	-•0083	•1613	-•0936
KPCL	-•1273	-•8773	-•1138	-•0724	-•1845	•0822	-•0692
KNSO	•1210	•8593	•0242	•1329	-•0973	•1320	-•1417
ADPO	•1723	•8364	•0453	•1777	•0306	-•0670	•2029
TOPL	•0242	•8226	•0034	•1444	•1683	-•3683	-•0869
KNOF	-•1696	•8056	•1438	•2276	-•2970	-•0648	•1646
KNNH	-•2284	-•7984	-•4197	•0674	-•2131	•1082	-•1438
% x max combined Cervical	•4015	•1272	•8532	•1725	•1587	•0681	•0937
% x max Cervical facets	•2139	•0908	•8244	•2439	•2200	-•0030	-•0955
% x max Cervical disc	•5043	•1152	•7066	•1101	•0626	•1160	•2360
% combined Cervical	•4595	•2099	•7057	•3180	•1960	•0733	•1089
% Cervical facets	•1748	•1583	•6848	•3958	•3091	•0393	-•1119
% x max Column facets	•1966	•0259	•6800	•4142	•5074	•0137	-•0011
% x max Combined column	•5860	•0971	•6332	•3222	•2674	•0389	•1648

Table 8 contd.

Variable	FACTOR 1	2	3	4	5	6	7
% combined Lumbar	•4689	•1677	•2539	•7817	•1250	-•0719	•0806
% x max Lumbar facets	•1267	•1071	•4198	•7624	•1340	-•1817	-•1054
% x max combined Lumbar	•3964	•1216	•4028	•7568	•0277	-•0882	•1528
% Lumbar facets	•1539	•1583	•3207	•7556	•2201	-•1451	-•1706
M_3	•2171	•1290	•1236	•6584	•2895	•4682	•1350
M_2	•3896	•2560	•1555	•6295	•2451	•3744	•1676
M_1	•3754	•3673	•1409	•5814	•1936	•3134	•1670
% Thoracic facets	•2068	•0085	•2444	•1708	•8798	-•0338	•0753
% x max Thoracic facets	•1514	•0096	•3020	•1664	•8289	•0911	•1920
% column facets	•2068	•0790	•5103	•4896	•6369	-•0247	-•0866
TMOPL	-•0686	•0228	-•1082	•0061	•0689	-•8158	•0672
CTHICK	-•2306	-•0464	-•0819	-•0860	•1311	•4042	-•3012
AMOD	•1202	-•0354	•0424	•0217	•1805	-•1102	•7831

Table 9 Correlations of Continuous Age-Related Measurements with the Rotated Factors in Males.

Variable	FACTOR 1	2	3	4	5	6	7
% x max Column discs	•8642	•2435	•2329	•2011	-•0968	•1363	•1105
% x max Cervical discs	•8309	•2557	•0877	•1132	•2870	•0041	-•0071
% x max combined Cervical	•8262	•3495	•0731	•1415	•3533	-•0831	-•0477
% Cervical discs	•8120	•1926	•1976	•2092	•1929	•0516	-•0594
% Column discs	•8113	•1849	•2861	•3981	-•0999	•1564	-•0145
% combined Cervical	•7912	•3615	•1532	•2780	•2523	-•0402	-•0726
% x max combined Column	•7782	•5149	•2028	•1944	•0069	•0331	•0898
% x max Thoracic discs	•7540	•2463	•1470	•1410	-•2589	•2518	•1819
% Thoracic discs	•7281	•2122	•2143	•3538	-•1613	•2732	•0553
% combined Column	•7063	•5123	•2537	•3928	-•0458	•0247	-•0209
% x max combined Thoracic	•6677	•5238	•1362	•1285	-•1753	•2032	•2346
% x max Cervical facets	•6653	•4144	•0233	•2048	•3160	-•2471	-•1544
% x max Lumbar discs	•6326	•3023	•2953	•4114	-•2039	•0653	-•0106
% combined Thoracic	•6293	•5616	•1754	•3215	-•0626	•1641	•1338
% Lumbar discs	•6186	•2191	•2883	•5459	-•2780	•0466	-•0355
% Cervical facets	•5998	•4919	•0635	•3142	•2209	-•2169	-•1479
% Thoracic facets	•2747	•8353	•0099	•1882	•1374	•0156	•2084
% x max Lumbar facets	•2726	•8083	•0788	•2114	-•1171	•0245	-•1598
% column facets	•4318	•8062	•0761	•2864	•1015	-•0947	-•0218
% x max Thoracic facets	•2515	•8053	•0649	•0448	•1314	•0784	•2670
% x max Column facets	•4919	•7865	•0697	•1350	•2079	-•0743	•0419
% Lumbar facets	•2519	•7540	•0657	•3444	-•0712	•0078	-•2561
% x max combined Lumbar	•5388	•6026	•2173	•3577	-•2148	-•0147	-•0691
% combined Lumbar	•5021	•5439	•1826	•5358	-•2408	-•0487	-•1431

Table 9 contd.

Variable	FACTOR 1	2	3	4	5	6	7
TNSO	•0807	•0577	•8800	•1184	•0925	-•1219	-•2299
KNSO	•1158	-•0187	•8465	•1327	•1079	-•1512	•1523
ADPO	•1382	•1035	•8378	•0500	-•0719	•0433	•0686
KPCL	-•4326	-•3035	-•8034	•0154	•0325	-•0593	•1092
TOPL	•0911	•1253	•7938	•1991	•1425	•0521	•2086
CNSO	•2662	-•1368	•7806	•0806	•0701	-•4479	•2044
KNNH	-•4735	-•0924	-•7014	•0382	-•4683	-•3833	-•2452
M_3	•2556	•2691	•0575	•8370	•0840	-•0854	•1558
M_2	•3016	•2917	•2922	•7957	•1394	-•0130	-•0180
PS	•3827	•1495	-•0303	•7312	-•0299	•1312	•1066
M_1	•3026	•3811	•1986	•7178	•1613	•0303	•0155
KNOF	•0544	•1151	•4080	•1013	•8053	•1333	-•0775
AMOD	•1156	•0980	-•1044	-•1063	-•0148	•8002	•0053
CTHICK	•0831	•1147	•0506	-•1378	-•0948	-•4437	•1121
TMOPL	•0287	•0361	-•0202	•1227	-•0503	-•1344	•8648