An address on chronic inflammation of bone : read in the Section on Pathology / by Charters J. Symonds.

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AN ADDRESS ON CHRONIC INFLAMMATION OF BONE.

Read in the Section of Pathology.

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I MUST offer some apology for the title of the subject, the discussion upon which, I am about to open. "Chronic Inflammation of Bone" sounds somewhat vague, and suggests a dissertation upon almost every osseous affection. That it is not my intention to weary you, or the members gathered here, with a ramble over so wide a field, I will first say that it is rather my object to inquire into the causes which produce enlargement and hypertrophy of bone. It was this notion I wished to convey when asked, some time ago, by our Secretary, Dr. Goodhart, to suggest a subject; but the slight confusion which often attends such arrangements, particularly amongst people otherwise busily engaged, must be my apology. I trust, however, that the subsequent note, inviting communications upon the various diseases in which bones are enlarged, has been sufficient to give a general outline of the drift of my paper.

The conclusions at which I have arrived are drawn chiefly from the study of museum specimens, especially those in our own collection at Guy's Hospital, which is particularly rich in examples of thickened bones. I have also examined the specimens in the Museum of the Royal College of Surgeons, and those at St. Bartholomew's Hospital, where many of Mr. Stanley's preparations are to be found. From these, as well as from other sources, I have been led to certain conclusions, some of which differ considerably from those generally received. These I will put forward in a tentative way, desiring to suggest that the ground is still open to inquiry, rather than to make any dogmatic statement.

Another object I have in view is to single out from these numerous forms of thickened bone—variously described as hyperostosis, osteoporosis, hypertrophy, etc.—such as can be unmistakably referred to recognised causes, and to use the information thus derived for the elucidation of the more obscure affections.

In pursuing these objects, though I shall be found sometimes standing in opposition to long-established explanations, some of which bear the weight of our greatest authorities, I trust that my views may serve the purpose at least of reopening an old subject, and that, as an outcome of our discussion, we may find ourselves nearer the truth.

I must anticipate, however, the disappointment that many, who may hope that this obscure subject will be cleared up will feel; for it will appear that still I leave a considerable amount of ground uncleared. This, I admit, will be found to be the case; but, after all, if one be successful in lifting but one morbid process into its proper position, or in removing another from a position falsely attained, it will, I imagine, be allowed that some advance has been made. This much, at least, I hope to advance.

Our inability at present, in referring many of these thickened bones to their correct position in pathology, is the result of our ignorance of the lesions that have been associated with them. It will be another of my objects to indicate the direction in which we ought to work, with the greatest probability of attaining the truth.

For illustrating this subject, I have, through the kindness of our curator, Dr. Goodhart, been able to show a series of specimens from our museum.

In examining the various bones that undergo enlargement, it is convenient at first to separate them into two groups, and to consider first the long bones, for in these the processes are fewer and the explanations simpler; next, to take the cranial bones, placing these aside for separate consideration, since the whole subject is more obscure, and a greater number of morbid processes are in force.

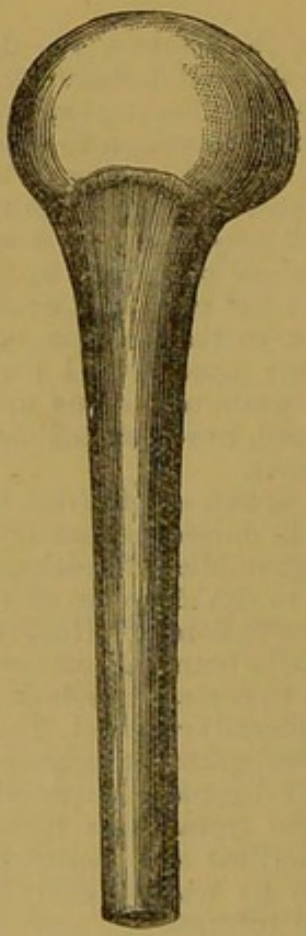

Fig. 1.—Normal humerus.

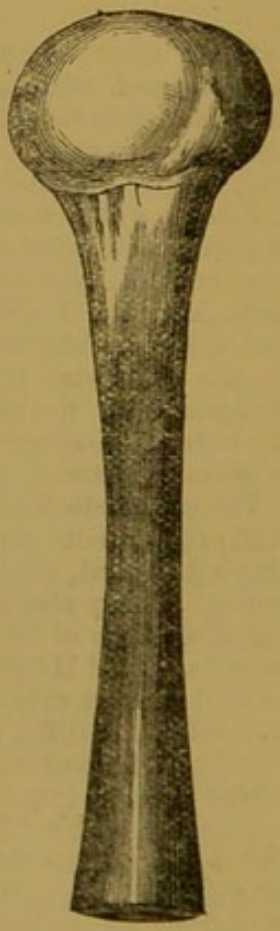

Fig. 2.—Humerus enlarged.

Now, amongst the causes known to produce enlargement in long bones, there is no doubt that chronic inflammation is the most frequent. I will first, therefore, ask you to consider the manner in which this process effects the change in bone, and next inquire into the various causes upon which the process depends; and afterwards

to consider what other processes than inflammation may produce this enlargement in bone. In this way, I hope to obtain information, which may be applied to the elucidation of the more obscure class of thickened skulls.

It is, I think, at the present day generally admitted, that inflammatory enlargement of bone may be due either to "expansion" of its tissue, or to a periosteal deposit upon its surface. As the proportion which each of these processes takes in producing enlargement is a debatable point, I may premise that I hope to show that the first, viz., "expansion," so-called, arising from osteitis, does not of itself lead to an increase in diameter in a bone, but that all such enlargement, so far as simple uncomplicated inflammation is concerned, is due to the second cause, viz., periostitis; and that chronic osteitis never increases the diameter of a bone until the periosteum is reached, when the latter lays down, where healthy, a variable amount of new bone.

It is unnecessary for me to dwell upon the effects of simple periostitis in producing thickening of bones; the specimens are numerous, and admit of no cavil. But the effect of chronic osteitis, in producing enlargements by expansion of the bone, seems quite open to objection.

The specimens upon which this effect is founded are those in which, on section, an enlarged bone is found uniformly porous to its outer compact layer, no trace of the original compact tissue being visible. Such a bone is said to be "expanded" by an enlargement of its spaces, or to be "swollen."

The historic specimen upon which this observation is largely founded was one communicated by Mr. Arnott to Mr. Stanley, and published by the latter in his work on *Diseases and Injuries of the Bones.* (Plate I, Figs. 4, 5, 6.) These drawings I have had copied from Mr. Stanley's work. The bones were re-

Fig. 3.—Transverse section of Fig. 2, showing "Expansion from Separation of Lamellæ."

moved from a boy, aged 14, who died four months after excision of the elbow for old joint-disease. The humerus is much larger and lighter than its fellow, and, by the transverse section, it is seen to be porous. This effect is said to be produced by expansion of the natural cavities of the bone, due to a separation of the lamellæ by inflammatory products.

Stanley (page 19) writes: "Simple swelling of a bone, from expansion of its tissue, is one of the most frequent alterations to which it is liable. The lamellæ are separated, the vascular walls are widened, and the bone is softened; not that its lamellæ and the walls of its cells are thinner, but that, by the separation and the widening of their interspaces, the bone loses so much of its compactness and power of resistance, that it readily yields to compression." He further says: "I have seen many instances of expansion of articular ends of bones, especially of the inner condyle of the femur, and of the head of the tibia, ensuing from a blow or other slight injury." Again, he observes, that "a bone, once enlarged by expansion of its tissues, will permanently remain so."

That I may quote no antiquated opinion, I may read the views of

Sir James Paget, also quoting Stanley, in the latest as well as in the first edition of his *Lectures on Surgical Pathology*. After describing the softening effects of inflammation in bone, he proceeds to show the subsequent changes that are permitted, especially the "swelling and expansion." Referring to this humerus of Mr. Arnott's, he says "the lower end was red and swollen, with expansion or separation of the layers of its walls, and the case showed well the coincidence of absorption and of enlargement by expansion"—a remark indicating his adoption of Stanley's explanation.

Sir James also refers to another specimen, his illustration to which I have cut from his "Lectures," and pass round with the description appended.

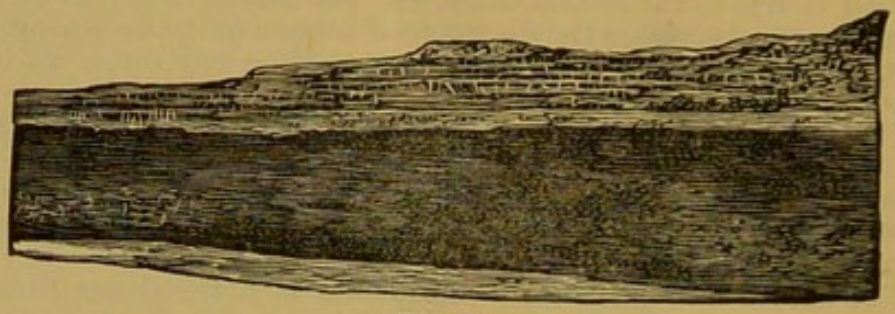

Fig. 4.

It is a section through the shaft of a femur exhibiting an increase in diameter due to the presence of regular laminæ outside a compact layer. "The most striking change," he says, "is a more or less extensive and wide separation of the concentric laminæ of the walls of the bone, so that, as in the section of this femur, the longitudinal section of the enlarged wall appears composed of two or more layers of compact tissue with a widely cancellous tissue between them."

I have not succeeded in finding this specimen in the St. Bartholomew's Hospital museum, but there exist amongst the excellent collection of bones many similar examples; and in the new catalogue such bones—I mean those exhibiting a new periosteal deposit with a porous interior—are described as showing thickening from "expansion and deposit."

A later authority, Mr. Lister, says, in describing a case of disease of the knee-joint, that "sympathetically the bones also had become affected, and the ends of both the tibia and the femur were greatly enlarged." It is, therefore, fair to assume that both a temporary, as well as a permanent enlargement of bone, due to expansion of its texture, is admitted at the present day.

In attempting to refute such an interpretation, I may first say, that a temporary swelling of a bone will, I imagine, from the nature of things, be allowed by the majority to be impossible. If such occur in the articular ends, as is asserted, I would ask what becomes of the articular cartilage, itself a brittle and non-vascular structure. Though bone does resemble in its behaviour under inflammation in many respects the soft parts, in this it seems different, that no swelling of its tissues is permitted, except of the periosteum.

Both Stanley and Lister have referred to the enlargement of the end of the femur and tibia, the former writer after injury, the latter in association with pulpy disease of the knee-joint. On this point I do not hesitate to say, that caries and necrosis of articular surfaces do not lead to enlargement by expansion of the articular extremity, but that such appearances, clinically, are fallacious, and that, when examined after excision or amputation, the bones will be found of normal size, however advanced the disease. These specimens, the one from the bones forming the knee, and the other from those forming the elbow-joint, afford a good illustration of this statement.

So often have I examined such bones, more than one of which has been put down clinically as expanded, that I feel upon tolerably safe ground in saying that there is no increase in size from such a cause, but that, in the case of the knee, the greater prominence of the bones is due (1) to the wasting of the thigh ; (2) to the thickening of the synovial membrane, and (3) to the backward displacement and outward rotation of the tibia, the last points throwing the inner condyle particularly into prominence.

I must here except those cases occurring in the middle-aged, where irregular spicula and masses of bone are formed round diseased joints, as may be seen in the elbow sent round. Such are evidently surface-formations, as is the slight superficial deposit upon the condyle of the femur, in the other specimens. For introducing such clinical details, I ought, perhaps, to apologise to those to whom such points are well known ; but the prevalence of the impression of the existence of enlargement, under such circumstances, must be my excuse.

Having objected to the generally received interpretation of such specimens as Mr. Arnott's, I now propose to offer a different reading, to account for these enlarged and uniformly porous bones. Adhering to my statement that all enlargement is a superficial or periosteal addition, the uniformity in structure may be explained in two ways.

In some, as in the humerus referred to, I would suggest that an organising periostitis has occurred at the same time as the osteitis, that the former process leads to a deposit of new bone, and increases the diameter, while the latter renders the compact tissue porous by absorption of the bony framework ; so that, the new bone being itself porous, and the old bone having become so, there is an unbroken area of cancellous tissue from the medullary canal to the thin compact layer bounding the new periosteal bone. Thus the appearance of expansion is explained. When the periosteal new bone has become sclerosed it resembles the compact, and again an uniform section of dense bone results. But in such specimens a white dense line may sometimes be seen, indicating the original limits of the compact layer, as in this femur (No. 1153 Guy's Hospital Museum). Again, in other specimens, as in the figure just handed round from Paget's work (Fig. 4), the osteitis has affected the superficial layer of the compact bone only, much of the original remaining compact, the outer part having become cancellous, and blended with the new porous bone.

The other way which I would suggest, in which uniformity in structure is produced, is more physiological than pathological, and is this:—If the periosteal deposits become permanent, and the compact layer have not been rendered cancellous by osteitis, it seems that, being now in the interior of a bone, it must assimilate itself in structure and function with the parts around it. The process by which this is effected seems to be atrophy, brought about by a loss of function, and must be very slow. This may be seen occurring in this specimen, where the sclerosed periosteal bone is blended with the original compact layer (No. 1152, 85 Guy's Hospital Museum). At one point the cancellating process has removed all distinction between the two parts, while, at another, the line of the old compact layer can be made out still.

The same cancellation of embedded compact tissue is seen in fractures, as in this humerus, where the new bone has formed around the displaced and partly impacted fragment. The compact tissue of the latter is becoming cancellated, while a well-marked compact layer limits the new bone. A specimen of a fractured femur in the College of Surgeons shows this well.

Again, when the fragments override, as in this femur, the uniting material has a thick compact layer externally, is porous internally, and the compact tissue of the shaft, where embedded, is seen, to be much thinner than elsewhere, in fact, is undergoing cancellation.

Smith, in his work on *Fractures*, calls attention to this point in endeavouring to disprove the existence of impaction in "Colles' fracture." He says (p. 161): "At a still more remote period, the enveloped portion of compact structure is frequently removed by absorption; it becomes, as it were, resolved into cancellated structure, the appearance of penetration is effaced, and the whole interior of the bone presents a cellular aspect."

It seems, indeed, to follow from the functions of the several parts of a bone, that all within the outer compact layer must be cancellous; so that, given a permanent periosteal increase, the surface of that deposit will become compact, and blend above and below with the original outer surface of the bone, while the now embedded compact layer will become cancellous; a result which again will give the appearance of expansion.

It seems, at first, that the explanation will scarcely explain some of the specimens of enlarged bones, such, for instance, as one figured in Stanley's work, where the cancellation is extreme, and strongly suggests expansion. It was, therefore, necessary to procure a specimen showing an earlier stage, and, curiously enough, no better could be found than one figured by Stanley himself in the sixteenth plate of his *Atlas*. A copy of this I have placed on the same sheet with the illustration before referred to. It shows a

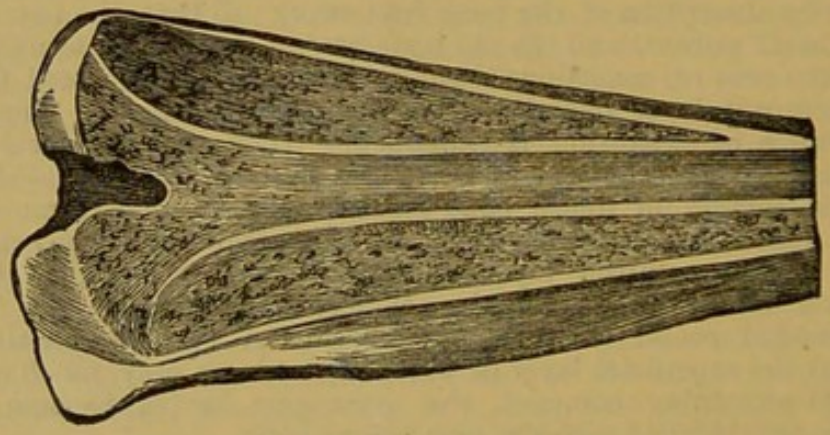

Fig. 5.

thick and smooth-surfaced periosteal deposit upon the old compact layer of the humerus. A new dense layer limits this new bone. The radius and ulna showed the same change. This specimen was obtained from a boy aged 10, long suffering from disease in the elbow. It is thus commented upon by Mr. Stanley: "Inflammation extending to the adjacent bones has occasioned the formation of a perfect wall of new bone around the adjacent portions of the humerus and ulna. The wall of new bone consists of an outer compact and an inner cancellous texture, and is, in each instance, formed between the periosteum and the walls of the original bone, to both of which it is united."

The outer surface of the specimen is fairly uniform; and had the case lasted long enough, I believe that the included compact tissue would have become cancellous, and the whole bone presented the uniform appearance seen in the first specimen.

I may also refer to abscess in bone as showing the absence of enlargement with central osteitis; for I believe I am correct in saying that no increase in the size of the affected part takes place until

the periosteum is reached, when this structure, on irritation, deposits a variable amount of new bone.

The authors I have quoted attribute the narrowing of the medullary canal to the reaching inwards of the laminæ surrounding it, owing to expansion of the spaces; but I imagine that, for the reasons given above, the obliteration of this canal is to be attributed rather to an endosteal formation.

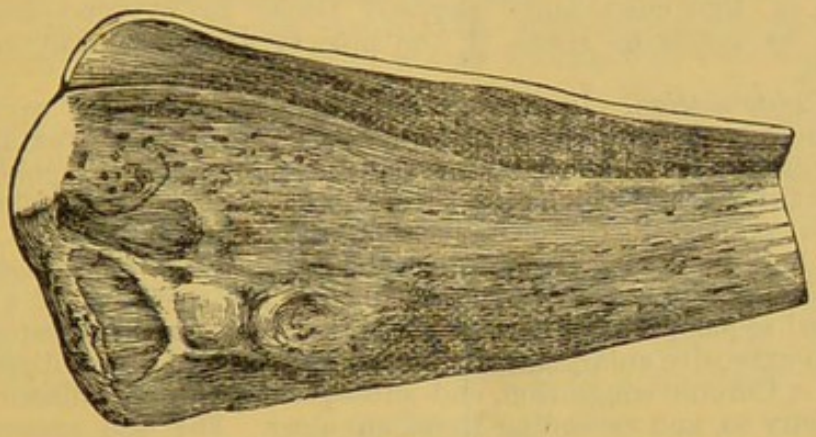

Fig. 6.

Besides the evidence derived from the examination of specimens in support of the view I have brought before you, it seems to me that the physical nature of a bone, negatives the possibility of expansion. Even if a previous softening be admitted, so as to meet the objection of rigidity, there ought to be evidence of a crowding together or compaction of the lamellæ, for example, round an abscess. But such an appearance I have never myself met with, and, so far as I know, has not been described.

The effect of inflammation is to produce absorption of the bony laminæ until the periosteum is reached; and, if flakes of bone be found in the wall of an abscess projecting from the bone, or, as is common enough, in the capsule of a myeloid tumour, I would prefer to say that such flakes have either been carried out with the periosteum, or, as is more probable, that new bone has been formed by the stretched and irritated periosteum.

Before passing to inquire into the cause of thickened crania, I will, for the purposes of discussion, formulate in a few words so much ground as has been already covered.

1. All enlargement from simple inflammation is due to periosteal formations, and the uniformly cancellous appearance is due to (*a*) the widening of the canals in the compact tissue through absorption of their bony walls, or (*b*) the atrophy or remodelling of embedded compact bone.

2. The term "expansion," as applied to bone, is misleading, as the widening of the spaces is due to absorption.

3. In central and superficial osteitis, no enlargement is produced till the periosteum is reached.

4. In simple articular caries and necrosis, there is no enlargement of the extremities of the bones from "expansion," this appearance being fallacious.

The causes producing enlargement in long bones are, so far as I can find, the following.

1. *Hypertrophy.*—Sir James Paget gives several examples of this, chiefly in the skull. So far as regards long bones, with which we are now concerned, few examples appear to exist, except those of *elongation* of bone recorded by Stanley and Paget. The latter suggests that inflammatory changes in bone produce this effect; but I would suggest that, as the elongation generally occurs in bones

when there is a cause of congestion near the epiphysis, as, for example, in the femur, the increase is due to rapid growth of the epiphysal cartilage from the attendant congestion. Besides these cases, which appear to be examples of true hypertrophy, one of two parallel bones—*e. g.*, the fibula—is said to become hypertrophied when the other, having been broken, has remained ununited. This term hypertrophy ought, I think, to be distinctly limited to these rare cases, so far as long bones are concerned; and that, rather than speak of thickened and sclerosed bones as hypertrophied, it would be better to speak of the process which has induced the change.

2. *Inflammation.*—In a very large number, as Paget says, enlargement is the consequence of inflammation in bone. Taking the explanation offered above as to the mode in which this change is brought about, and which differs from Sir James Paget's, the following may be taken as the causes under this head: syphilis never, so far as I can find, producing an uniform enlargement of an entire bone. *a.* Scrofula. *b.* Simple inflammation due to the irritation of a long-retained sequestrum. It may be noted that very small sequestra may lead to extensive enlargement. A specimen handed round illustrates this. *c.* Chronic congestion, and subsequently chronic inflammation secondary to, and extending from, an ulcer. The best example of this is seen in the tibia, a preparation of which is before you. The inflammatory process may extend deeply, changing entirely the appearance of the compact layers, obliterating all distinction between it and the new periosteal bone. Coming under the same heading is the enlargement occurring where chronic œdema exists, an increased supply of blood or lymph being furnished to the bone. In Dr. Day's case of obstructed lymphatics, in one lower extremity the bones were much larger than those in the sound limb. One other example of this I have seen in a patient who had chronic suppuration in the foot for many years, associated with an elephantoid condition of the leg; but the tibia and fibula were here much increased by periosteal deposit; while the bones of the foot, though in the very focus of inflammation, were unaltered. The fibula in the jar, and the tibia, illustrate this to some extent.

3. *Osteitis Deformans.*—It is unnecessary to dwell upon this malady, so fully and so recently described by Sir J. Paget, unless to remark that in it alone (so far as our present knowledge goes) is a long bone, ever enlarged uniformly and throughout. An example is before you in this tibia. The increase in diameter of this tibia is not difficult to explain in the way I have suggested, but it is much more difficult to account for the increase in length of the bones in this disease. That elongation undoubtedly takes place, this illustration of a much curved and elongated radius will be sufficient proof. That the morbid process affects the articular lamellæ, as well as the rest of the bone, is scarcely sufficient to account for the great increase that occurs. Hence, I am at a loss, for the present, to account for the change; unless, indeed, the whole process displayed in this affection be different from that of simple inflammation, and own a method of increase peculiarly its own.

4. *Rickets.*—It seems doubtful if rickets ever produces permanent enlargement of a long bone, except the greater development seen at the point of curvature, which I imagine may be looked upon more as a result of repair than of disease. This fibula well illustrates the condition to which I refer. Paget, in speaking of this disease as it affects bones, says they are "too short, not too long; too small, not too large."

5. *Osteomalacia* does not lead to increase in the size of the long bones; and I hope presently to show that neither does it do so, as has been alleged, in the skull.

Turning, now, to the cranial bones, several new causes of enlargement present themselves for consideration; and these I now propose to investigate.

Localised thickenings of the cranial bones may, as in others, be due to syphilis and struma, but neither of these produces an uniform enlargement. These uniformly thickened skulls, examples of which occur in almost every museum, are still ill-understood.

Several distinct appearances are presented by these specimens.

1. In some, the outer and inner tables are distinct, compact, and of no great thickness; the diploë is well-defined, with a regular open texture. It is, indeed, normal in all respects, except for its great thickness. Here is an example of this form, which, I think you will see differs from all the others. It is part of a skull of a man aged 30, who committed suicide, many years ago, at the London Hospital. A portion of the same is in the museum of the College of Surgeons.

Writing on hypertrophy of bone, Sir J. Paget attributes to concentric hypertrophy, from shrinking of the brain, many thick skulls.[1] He does not describe the appearances in such cases, but gives an illustration—which I pass round—taken from a similar section of

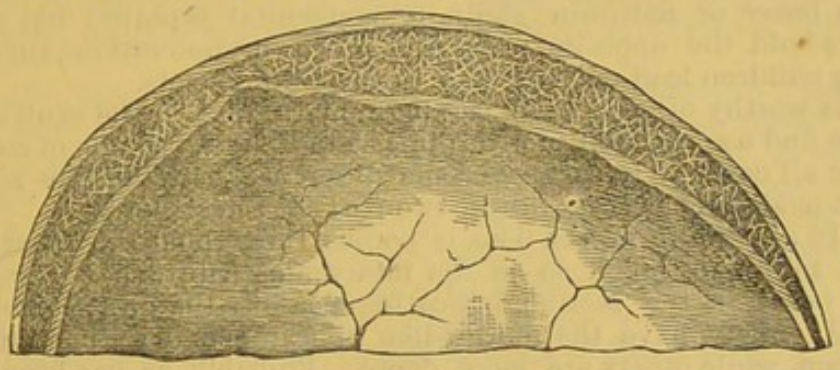

Fig. 7.

this same skull, so that we may take it as representing his idea of the change. He describes it thus: "The thickening of the skull is effected by the gradual remodelling of the inner table and diploë of the bones of the vault; so that, although the exterior of the skull may retain its natural form and size, the inner table grows more and more inwards, as if shrinking towards the retiring and shrinking brain—not thickening, but simply removing from the outer table, and leaving a wider space filled with healthy diploë."

He gives another case of a young lady, intellectually feeble, whose cranial cavity was singularly contracted, the skull having adapted itself to an imperfectly grown brain by a hypertrophy of its deploë, while externally the skull was not below the average female size. It seems to me that this case offers a different explanation of itself, and one that may be applied to the specimen I have handed round, and of all thick skulls presenting similar characters.

So far as I can gather, there is no appearance which suggests the present existence or progress of this "concentric hypertrophy." I am rather inclined, on the contrary, to hold that the shrinking brain is replaced by cerebro-spinal fluid, an appearance frequently observed, and that the thickening in such skulls as these takes place during the period of development only, and that it is not a true example of accommodating hypertrophy. The capacity of the cranial cavity is, I imagine, determined—as indeed Paget has suggested—by the

[1] On this subject of shrinking brain, with alteration in the bone, I may quote a remark from Billroth, who says "that the filling up of the diploë with bony substance is so common in advancing age as to be scarcely pathological." This seems to me to need confirmation. He does not state if, at the same time, the bone is thickened.

growth of the brain, or in some perhaps by an early ossification of the bones with closure of sutures. Admitting this, two explanations suggest themselves.

First, after the lateral increase, or increase in area, has been checked, the process of growth continues and leads to a thickening of the bone; the new material being added to the surface instead of to the margin. In this way, the size of the head may not be diminished.

Secondly, it is not unlikely that rickets may produce this appearance. In this disease, as is well known, the cranium becomes enlarged, often with remarkable elevations round the centres of development, producing the so-called natiform skull. A section through such a skull of an adult, in the College of Surgeons, shows exactly the appearances presented by this piece of bone and by Paget's drawing. I have not met with any description of an adult rachitic skull where enlargment had persisted. Sir J. Paget's description applies, without doubt, to the early stages, specimens of which exist in the College museum, and are described as "very light, almost friable, not porous, but like fine cloth or felt."

I must not omit to mention here that M. Parrot has attributed these bossy or natiform skulls to congenital syphilis; but many others hold the opposite view, and my own observations amongst living children lead me to decide in favour of rickets.

It is worthy of more than passing remark, that in the skull alone do we find a permanent increase in size in rickets, though, of course, not in all cases. It suggests some difference between the subsequent modelling process in the two parts of the skeleton.

2. In another form, the bone is porous throughout the inner and outer tables, as much so as the intervening substance. Irregular gaps occur in the section, indicating possibly the situation of the diploë. Many have the mortar-like appearance described by Mr. Durham, while others are more dense. Probably, as has been suggested, these two are but stages of one process. Mr. Durham, however, regards the two as distinct, the porous form—at least some of them—he considers examples of cured osteomalacia; the denser ones as rachitic. The majority of these skulls are no doubt examples of osteitis deformans, in which disease, as is well known, the bones are uniformly thickened. No trace of the compact tissue is left at all. Several of these specimens illustrate this appearance.

Another disease said to lead to the production of this appearance is osteomalacia. This has generally been stated since the publication of Mr. Durham's paper in the *Guy's Hospital Reports* for 1864, and since Mr. Solly's communication to volume xxvii of the *Medico-Chirurgical Transactions*. Paget alludes to it upon the authority of the above writers. I have examined, with regard to this point, the specimens upon which the statement of enlargement has been based, and I cannot but think that a different and truer explanation can be given, and indeed, that in this malady there is no increase in the thickness of the skull. Here is Mr. Durham's specimen. The calvaria is seen to be much thicker than normal, and to show all the appearances described in osteomalacia. It is by no means so thick as in the dry specimens just exhibited, and it is far more porous.

Mr. Solly's specimen may be seen in the museum at St. Thomas's Hospital. It is a dry preparation of about the same thickness as this one, and shows the same degree of porosity.

In the same bottle with Mr. Durham's specimen are portions of several other bones from the same patient, none of which show any thickening, and it will be found to be true that in no other bone does an increase in size take place. Surely, therefore, unless we admit that the effects of disease are entirely different in the cranial

bones from what they are in the long bones, this is almost sufficient to negative the conclusion of enlargement.

Again, in some well-marked examples of the disease, the cranial vault is, if anything, thinner than usual; at least it is not increased in thickness, though showing well the characteristic changes. Mr. Solly records one such case, and explains the absence of thickening by the short duration, which was four and a half years, while that in which the enlargement occurred had existed eight years; and he says he has no doubt that, had his second patient lived, an increase would have taken place. In opposition to this opinion, it must be said that, though the skull was not thick, the other bones exhibited the softening effects of the malady in an extreme degree, the cranial bones themselves being deeply affected.

Mr. Durham's patient in whom the skull was thick had been ill but three years; so that, upon the ground of duration of the disease, little support can be given to Mr. Solly's conclusion. Mr. Durham founded his statement also upon certain microscopical appearances, which he found to be alike in his case of osteomalacia, and in a soft spot in one of these thick skulls. He concluded that the latter was an example of cured osteomalacia. I am unable at present to dispute the observation, for it would require minute examination of several of these osteoporotic crania, and this at present I have not made.

Again, here is a specimen showing all the changes of osteomalacia, without any increase in thickness—in fact, it is rather thin. This is the ground upon which, so far as I can find, the statement is based; and, in opposition to it, I will suggest, believing I am nearer the truth, that osteomalacia does not give rise to any increase in thickness of the cranial bones, but that the occurrence of this appearance is a coincidence of a normally thick skull made porous by the disease. Moreover, neither Mr. Solly's nor Mr. Durham's specimens are by any means so thick as the bone is in osteitis deformans. Finally, I may add that Mr. Durham's conclusion that some of these porous skulls are due to osteomalacia, infers that the disease is curable. The rarity of this result in well marked examples—if, indeed, it ever occur—is an argument against his deductions.

If we remove this disease as a factor in producing examples of these porous skulls, but one cause is left, and that is Osteitis Deformans. I do not, for a moment, mean to state that this is the only cause, but that, so far as we can say at present, it is the only one we know.

3. A third group, illustrated by this example, exhibits, besides a general increase in size, numerous nodular projections from the inner surface, varying from small rounded nodules to the large irregular agglomeration of bony ridges seen in this specimen. The bone besides is heavier and more dense than in the previous class. The cause of this change is obscure, but it may possibly be an earlier stage of the next group, in which a more definite tumour-formation occurs. They have been found in lunatics; and the smaller nodules are said to occur in puerperal women.

4. In the next class comes the disease known as leontiasis ossea. This differs, however, in so many respects from the previous group, that there is not much difficulty in associating examples of this affection. The bones of the face are affected as well as the vault of the skull, and, moreover, the change is more irregular and nodular, at least, so far as the vault is concerned. Again, some of the specimens of this disease show definite growths, which are evidently exostoses, indicating probably the association of the affection.

Coming next to these specimens, are those skulls covered with smooth bossy masses of bone, and, at the same time, showing

considerable increase in thickness. The most notable example is Mr. Bickersteth's well-known specimen, now before you.

Through these last two groups, we are led easily into tumours; and I must not omit to mention that several of the cases of osteitis deformans have been associated with malignant disease of the bones; and the suggestion, I believe, originally made in the discussion on Sir J. Paget's paper, by Dr. Goodhart, that that affection might, after all, be a diffuse form of new growth, may be true. Here is an example of a thick calvaria, with cancer in the vertebræ.

Returning to these osteoporotic skulls, there is yet the important question to answer: How is the enlargement produced? Is the new material laid down under the pericranium or dura mater, or is it due to a formation in the diploë whereby the two tables are pushed aside?

So universal is the change in all the specimens examined, that I have been unable to discover a point where the change is in progress. So far as simple and well-known inflammatory processes are concerned, I have no doubt that all additions in thickness are surface-formations, as in other bones; but, with regard to osteitis deformans, I must admit the explanation is more difficult. That an increase occurs externally, is manifest, from the size and shape of the head, and the increased depth of the grooves for the meningeal arteries is as good an evidence of an enlargement internally. The irregular gaps, by indicating the original position of the diploë, show in some that greater increase is external to this point, while in others it is internal to it; so that little can be gained from this appearance.

As mentioned before, I think it probable that osteitis deformans differs in its mode of effecting changes in bones from ordinary inflammatory affections; and that this may account for the peculiar appearances in the cranial bones, and may be another reason for separating it as a special pathological change.

Mr. R. W. PARKER (London) said, if he had understood Mr. Symonds's argument aright, that all enlargement of bone was due to changes in the periosteum, and that it did not depend on changes in the bone itself, he should be obliged to join issue. In his own opinion, uniform enlargement of bone depended on changes in the bone-substance itself, and might run its course with very little implication of the periosteum. He thought that, just as chronic inflammation of soft tissues led to thickening, and to a kind of spurious elephantiasis, so chronic inflammation would probably lead to a similar enlargement in bone. He showed some specimens illustrating these views, which were quite at variance with those Mr. Symonds had just laid down. His first specimen showed a femur uniformly enlarged, due to a small central necrosis; the femur retained its normal shape; it was larger in all its dimensions, and heavier; its periosteum had peeled off with the greatest readiness, except at one spot, and this corresponded exactly to where surgical means had given rise to a local periostitis, and so caused thickening and roughening on the surface of the shaft. The second specimen showed a central necrosis, with great and (as usual) very irregular deposit on the surface of the bone, due to periostitis; the bone itself was not much, if at all, enlarged. The one specimen was evidently an ostitis proper, and the other a periostitic deposit. As regards the statement that syphilis never produced a uniform enlargement of a bone, he also joined issue. He had seen many such specimens, and handed one round, illustrating this point as he thought, for the inspection of those present.

Dr. JOSEPH COATS (Glasgow) agreed with Mr. Symonds that thickening of bone in connection with inflammation was usually due to new formation from the periosteum. He differed from him in

his contention that no expansion of the bone itself occurred. There was no such thing as expansion in the rougher sense, by shifting of the dense bone and the lamellæ, but there was such a thing as an expansion entailing absorption and new formation in the substance of the bone itself. Dr. Coats referred to the conditions observed during development as throwing light on the subject. Mayer pointed out that the cancellated tissue of bone had a definite architectural arrangement, and that this arrangement was preserved during the growth of bone. This must be effected either by expansion of the various lamellæ, or by absorption and new formation. Wolff asserted that it took place by interstitial expansion, but this view could not be accepted. There was in the growth of the individual bones an abundant absorption and new formation of bone, so as to adapt them in shape and internal architecture to the altered conditions. Turning to a definite inflammatory process, the union of fractures, there was here also, when the fracture was united, a rearrangement of the bony lamellæ, so as to adapt the bone to the different directions of pressure; and, in order to do this, there must be absorption and new formation. Again, in cases of concentric thickening of the skull, Mr. Symonds supposed that this mostly occurred during the period of growth. This was not consistent with the fact that, in atrophy of the brain, as in general paralysis occurring in old or middle-aged persons, there was frequently considerable thickening. This could only occur by what might be called an expansion of the bone, a process of absorption and new formation, so as to give the appearance of a displacement inwards of the internal table by increase of the diploë.

Dr. G. Sims Woodhead (Edinburgh) agreed with Mr. Symonds as regards the absence of distension of bone as a mechanical condition. In the soft tissues, any increase in tension was followed at no great date by formation of abscess, even when in a chronic form; and, in the case of bone, it would be expected that, where there was increased tension sufficient to cause distension, abscess would rapidly be formed. Distension must be looked upon rather as a "vital" process, and a process in all respects similar to what took place in inflammation of soft tissues, as in ordinary tissue or in cartilage, where there was final increase in the number of cells, and then an absorption of the formed material. At no distant date, this was followed by a new formation of the formed material, which might be moulded afresh, and then distension might take place, as undoubtedly it did in many cases. He agreed with Mr. Symonds that periostitis played the principal part in the formation in bone, or where there was great increase in size. In the majority of cases of periostitis, the formation of new bone was modified considerably by various external conditions, and by the degree rather than the kind of the inflammation. He considered that, until bone was looked upon as a form of connective tissue, little progress would be made in our knowledge of the study of bone-disease. It could be looked upon as amenable to modification during growth; but, when once formed, no change could take place until the rigid tissues were removed, and the way was left clear for a remodelling of the bone. In inflammation and formation of new bone, the whole of the changes pointed to absorption, followed by new formation when the process was within the bone. On the surface of the bone, or under the periosteum, the changes which took place were now pretty generally agreed upon. In many cases, undoubtedly, the two processes, "endostitis" and "periostitis" were combined.

Mr. Paul (Liverpool) said that, in his experience, malignant infiltration of bone, whether osteoid or otherwise, invariably induced absorption of the original bone, though the former growth was accompanied by the formation of fresh bone. He thought that, in

fractures and many other conditions, bone-formation was not entirely due to the periosteum, but often depended, to a large extent, upon the bone-tissue.

Dr. GOODHART (London) agreed with Mr. Symonds in very much that he had said. He had perhaps laid himself a little open to criticism by his way of stating the case, but that he would be able to deal with, in his reply; as it was sufficiently obvious that, seeing that it was necessary to admit a remodeling of the bone to a larger scale, the changes in the central parts must be an essential part of the process; and, of late, Dr. Goodhart had come to think that, while the periosteal changes were the more important, all inflamtions were inflammations of the whole bone. One of the difficulties of the subject was that specimens admitted interpretation to suit different views; and a better illustration of this could hardly be found than the specimen of an infant femur, passed round by Mr. Parker, to illustrate just the opposite view to that of Mr. Symonds. Dr. Goodhart, looking at this, thought that it supported Mr. Parker's contention; but Mr. Symonds and he had seen others like it, where, as the result of injury in children, the old shaft still remained enclosed by a new shaft derived from the periosteum. There were many other points in this very interesting paper upon which he would have been very glad to say something, but time would not allow.

Dr. TURNER referred to specimens of enlargement of the tibia in connection with an old central sequestrum, in which, on section, cancellous tissue alone was seen, with filling up of the medullary canal, in which an indication of compact bony wall was traceable. In such a case, it could not be said whether the enlargement of bone was due to periosteal or osteal growth. Where a sarcomatous growth was developed in the centre of a bone, the outer shell of bone, left by absorption of the inner layer, might be widely expanded; and it seemed possible that, in the case of the cranial bones, an inflammatory hyperplasia of the diploë might lead to a separation of the inner and outer tables, and, subsequently becoming ossified, might lead to true osteal enlargement of the bone. The occurrence of similar expansion of the thick walls of the shaft of the long bones seemed more doubtful.

Mr. SYMONDS replied.