have not a hæmolytic process. I have ventured to take up some considerable space in giving the principal clinical facts of this case, because it seemed to throw some light upon the disease, and to strongly support the conclusions arrived at by Dr. Hunter in his very valuable contribution to our knowledge of this subject.

THE PHYSIOLOGY AND PATHOLOGY OF SOME OF THE VARIATIONS IN THE HUMAN SKELETON.

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WE have for a long time been hampered in our progress of the study of abnormal conditions by the use of terms to which various observers give different meanings, and which others, again, use to signify conditions which do not resemble one another. The two words to which I would now take objection are *disease* and *inflammation*, both of which are used very indiscriminately. The task of drawing any definite line between what is physiological and what is pathological in the case of the skeleton, and between the changes that are inflammatory and those that are not inflammatory, seems to me to be one of the greatest difficulty. I will attempt to illustrate my point in the following statements of what I believe to be facts, each of which will be discussed under a separate heading.

1. The so-called normal anatomy of each race is dependent for its existence upon a certain fixed relationship between the normal organism and its surroundings. If that normal mechanical relationship, or, in other words, the physiology of the organism, be altered, a corresponding change is produced in its anatomy in order that it may be able to accommodate itself to its surroundings and to perform its novel functions. We understand that each portion of the skeleton, its muscular coverings and appendages, performs a definite physiological function, the character of that function varying not uncommonly at different periods of the lifetime of the individual, in correspondence with changes in its relationship to its surroundings. Take, for example, the brain of the fœtus, which can be subjected to very considerable local or general pressure without suffering any injury. We see how the structure of the skull is adapted to retain the integrity of this organ in its first most important mechanical relationship—namely, to the true pelvis of the mother—as it makes its transit into the outer world. Later, as the brain acquires a much more sensitive structure, the skull-cap is rendered just sufficiently strong and firm to shield the most sensitive organ in the body from such local and general pressure as it is exposed to under average circumstances. In the human subject, that bears to its surroundings the ordinary relationship of adult life, we obtain by these mechanical means a skull of a thickness and texture which we are accustomed to regard as normal. As the mechanical relationship which this portion of the organism bears to its surroundings differs very considerably in the several races of man and quadrupeds, we find that in each the amount of thickness and density of the vault of the normal skull varies in proportion as the necessity for such protection exists.

2. If the physiological relationship of the normal organism to its surroundings be altered in such a manner that the brain and its bony case be exposed habitually to the influence of forces other than those which affect and determine its normal structure, we see that the form, texture, and thickness of the skull is altered in order that the skull may perform satisfactorily its new functions or physiology. Such experiments are being continually performed for us owing to the necessity which exists in large communities for the subdivision of labour. If we examine the skulls of those who have been engaged for many years in carrying heavy loads upon the head or upon the back of the head and neck, and compare them with the normal or average skull of the same race, we find that the abnormal forces to which the skull has been exposed have produced changes in its several characters which vary

in type and locality with the direction and degree of the forces which have exerted their influence upon it, and also with the age and vigour of the individual.

3. I do not think that anyone would call such a skull diseased, or would assert that the abnormal characters which it presents are the result of chronic inflammation. The influence of the same heavy load that produced the alterations in the skull determines in the other portions of the skeleton through which its weight is transmitted changes whose character and causation are identical with those present in the skull, yet pathologists almost without exception insist on describing the more obvious and local changes in the former as inflammatory and not physiological. Up to a very recent date they were regarded as the product of the disease called rheumatoid arthritis. The changes to which I refer are a general increase in the density of the structure of the bones, and a more marked local increase along the lines of greatest pressure; a compression and induration, or a partial or complete removal of the soft structures intervening between adjacent bones; the formation of large bosses or margins of dense bone in the seats of greatest pressure, where they perform a definite physiological function; a frequent partial or complete ankylosis of adjacent bones, together with the occasional conversion of an amphiarthrodial joint into one of an arthrodial type, &c. It seems to me absurd to argue that, because the bones and the bosses or margins that project from them have acquired a density of structure and a power of resisting pressure which is necessary to them in order that the organism may continue to perform its abnormal functions, and which are enormously in excess of the average or normal, the condition is inflammatory and not physiological. The skeleton of such a labourer is brought into a remarkable and unusual relationship to its surroundings, which entails its habitually and constantly performing certain special functions which are somewhat similar to those performed by the normal subject, but demanding the expenditure of an enormous amount of muscular energy in the support of an immensely heavy load. Similar changes ensue in animals other than man which have to draw or carry heavy loads. Practically it is impossible to pursue any occupation entailing the habitual performance of any sequence of movements or the carrying of any weight without the several portions of the skeleton influenced by it rapidly undergoing distinct and characteristic changes.

4. Let us go one step further in our research, and suppose that the labourer continues to perform his heavy work under disadvantageous circumstances, such as diminished physical energy due to want of sufficient food, exposure, or disease, disproportionately heavy load, &c. We find that, while very extensive ankylosis ensues between certain bones, in other joints, as that of the hip, the organism may be no longer able to render the upper margin of the acetabulum sufficiently dense and strong to support the pressure transmitted to it through the upper surface of the head of the femur. In consequence of this the margin yields, and the head, while itself undergoing changes in form, is gradually displaced further and further upon the dorsum ilii. During the displacement of the head the organism makes continuous vigorous efforts to stay its ascent by the formation of bone above it and in the ligaments. Are we to regard this condition as pathological, and the manner in which the changes have been brought about as inflammatory and not physiological? I maintain that we are not. The organism has placed itself in a very abnormal relationship to its surroundings, which differs very greatly from that which determined and serves to retain unimpaired the normal form of its skeleton; in fact, it has attempted to perform a physiology for which it was structurally unfitted without its several portions undergoing such extensive changes as finally to unfit it for performing perfectly their original functions. These are the changes that are regarded as inflammatory in origin and the product of disease.

5. In the performance of many forms of heavy labour, and not unfrequently as the result of deformity, enormous strain is sustained habitually by certain muscles—as the deltoid, subscapularis, gluteus maximus, psoas, soleus, &c. The presence of this strain determines the extension of bone into the tendinous attachments of these muscles or along the direction of the traction which they exert. I allude to the development of the third trochanter on the femur, to the elongation of the lesser trochanter, to the separate formation of bone in the psoas tendon, to the increase in

breadth of the acromion, to the occasional enlargement of the prominence in the insertion of the deltoid, to the enormous increase in amount of tendon in the several muscles, &c. I mention these conditions to show that they are caused in a definite mechanical manner and to perform an obvious physiological function, and that *their origin is not inflammatory*. They are sometimes, though obviously wrongly, regarded as retrocessions to a preceding type, and arising without any cause, if one could imagine such a possibility.

6. If the relationship of the growing organism to its surroundings be altered so as to prevent it exposing the various portions of its skeleton to the influence of those factors which originally determined and now serve to maintain their normal form and the character of the several muscular and ligamentous tissues, we observe that it is wanting in vigour and that it assumes habitually *attitudes of rest*. This results first in the fixation and subsequently in the exaggeration of these postures, which are simply physiological, the bones, both in their growing lines and in their points of impact, and the ligaments and muscles, undergoing such gradual and progressive changes as finally to render the organism unable to perform functions which were originally normal, but which have become abnormal. I allude to the simple acquired deformities of young life. Have we any right to regard the manner in which this alteration in the mechanism of the organism is brought about as *disease* and the product of *inflammation*? Many of the processes involved in the earlier stages of the development of these deformities are not commonly regarded as inflammatory, but the later changes, such as the destruction of fibrous and articular cartilage and bone, and the eburnation and osteophytic marginal growth from the latter, usually are. These conditions are obviously of necessity normal to their surroundings and physiological in their function.

7. In feeble old age, the form of the skeleton as a whole, as well as the form of many of its constituent parts, undergo remarkable changes. The attitudes in which the body is retained are such postures of rest as are habitually assumed at that period of life, it being the aim of the organism to throw as much strain as possible upon the ligaments and bones, and to make as little use as possible of the enfeebled muscles. These attitudes are almost identical in character and causation with the similar deformities which sometimes appear in young life. Many of the joints are altered in character by the change in the functions which they now have to perform, or, in other words, the several portions of the organism, like the organism itself, have to accommodate their form to an altered relationship to their surroundings. The texture of the bones is rendered rare, and many alter very greatly in form, notably the sacrum, which yields before any bone in the body. Now I do not think that anyone, recognising the obvious manner of its causation, can regard the destruction of the inter-articular fibro-cartilage of the temporo-maxillary articulation, the eminentia articularis, the articular cartilage and subjacent bone of the glenoid cavity and condyle of the jaw as being the product of any inflammatory process. The same applies to the yielding of the sacrum, the destruction of portions of the fibro-cartilages between the vertebræ, the formation of articular cavities in their substance, and many other changes which I have described fully in the Transactions of the Pathological Society (1886).¹

8. Not very rarely a condition described as mollities ossium develops in feeble old age. The changes presented by the skeleton are the exaggeration of those which I have described as constituting senile degeneration; in fact, it is impossible to draw any sharp line of distinction between the more commonly observed senile change and the more rarely observed malacic alteration of the skeleton. The latter is associated with a deposit of a more or less completely decalcified callus upon the outer surface of the skull, when that structure has become so soft that it is unable to retain the brain securely in position and to shield it from injury. Preceding this deposit upon the outer surface of the vault there is a deposit of a similar material upon the softening lamellæ of the original bone. I do not intend to discuss the causation and pathology of mollities ossium further here, as I have done so in preceding papers,² but I have brought

forward enough to show that where the organism is by some means being deprived of bone-forming power, whether it be due to an inability on its part to make use of food, or perhaps even to obtain it, as in the feeble old subject, or in the case of its occurring at a comparatively early period of life, when it may be due also to some very considerable strain upon the organism, such as frequent childbirth, prolonged illness, &c., the whole, or nearly the whole, of the bone-forming function of the organism is devoted to securing the most important organ in the body from injury. Are we in this condition to regard the wasting of the long bones, or the softening of the skull, or the deposit of a form of decalcified material in its substance and upon its surface as being inflammatory, or as being other than physiological? I think not.

9. We have a similar condition developed in the young subject when it is not supplied with or is unable to obtain suitable food. The bone-forming capacity of the organism is insufficient to form bone strong enough to perform its normal physiological functions. The organism, in a manner aware of such insufficiency, and in the use of a capacity which I might be allowed to call *tissue instinct*, makes vigorous attempts to remedy it by a rapid and irregular growth of the growing line of cartilage, and by a similar rapid deposit of soft and more or less insufficiently calcified periosteal bone. Though the whole condition of the change in the osseous system indicates a *hurry* of normal physiological changes, yet they all follow simple mechanical and functional lines, and are in such a direction as to tend to fit the organism to bear the ordinary relationship to its surroundings. The long and short bones of the body yield and form curvatures. As the skeleton of the rickety subject acquires a normal density of structure with advancing age, which it is enabled to do by the fact that as time goes on the difficulty of obtaining suitable nutriment becomes less conspicuous, the excessive thickening of the skull, being no longer requisite to shelter or retain the brain in its normal form, disappears, though the organism is unable to part with the bone which had been deposited along the concavities of the long bones to strengthen their curvatures. The deformity produced by the curvature is to a considerable extent diminished by the removal of bone from the convex surface of the shaft, and by the further deposition of new bone upon the concavity.³ To how much of the condition of the skeleton which we describe as rickets, and to which of its stages, are we justified in applying the term *inflammation* as a factor in their causation? and, if we do apply it, what do we mean by it? To assert that in mollities ossium, as in rickets, the vault of the skull is thickened, because it is formed in membrane and not laid down primarily in cartilage, is begging the question and not explaining the causation of the hypertrophy.

10. Lastly, we come to the most difficult problem of the lot, and that is osteitis deformans. It was supposed that only certain bones of the skeleton exhibited the changes that characterise this abnormal condition; but I have been able to satisfy myself, by careful dissection of well-marked examples, that, like rickets, it is a condition that affects every bone in the body; that the changes, such as the thickening and sclerosis, are, as in rickets, mollities ossium, and labour conditions, most marked where it is necessary that they should be, or, in other words, where the necessity for their existence arises. The mechanical aspect of osteitis deformans, like that of rickets, is an inability of the osseous system to bear its normal relationship to its surroundings; and the organism, by the exercise of its tissue instinct, attempts to obviate this progressive insufficiency by the abundant and rapid deposit of new material, the mode and locality of its deposition being based on the principle of demand and supply. It differs from rickets in that the former condition is a progressive one and is not recoverable, while in the latter such thickenings as are no longer requisite for the maintenance of a satisfactory relationship between the organism and its surroundings disappear. Exactly the same mechanical factors determine and control the progress of osteitis deformans as do that of the skeleton, whether normal or displaying senile, malacic, or rachitic changes, the similarity to the last condition being perhaps the most obvious.

¹ The Causation and Pathology of the so-called Disease "Rheumatoid Arthritis," and of Senile Changes.

² Mollities Ossium, Rheumatoid Arthritis, and Charcot's Disease (Trans. Path. Soc., 1885). The Factors which determine the Hypertrophy of the Skull in Mollities Ossium, Rickets, Osteitis Deformans,

and Congenital Syphilis (THE LANCET, April 28th, 1888, p. 815). An important Factor in the Causation of some of the Deformities which develop in Rickets, Mollities Ossium, and Osteitis Deformans (Journal of Anatomy and Physiology, 1887).

³ The Anatomy, Pathology, and Physiology of the Deformities that Develop in Young Life. (Guy's Hospital Reports 1887.)

If we are to regard osteitis deformans as a sarcomatosis, we must remember that it affects the whole of the osseous system, and that the laws that govern its growth and determine its most obvious characters, and to which the skeleton reacts, are identical with those that govern the growth of the normal skeleton, so that it must form a very remarkable exception to the mode of extension of sarcomatous tumours generally. Is osteitis deformans a universal inflammation of the osseous system? If so, its form is not exactly that of rickets or of mollities ossium. If we look upon the thickening of the skull in osteitis deformans as being the result of inflammation, I think that we must also regard the similar condition of the skull in mollities ossium and rickets as being also inflammatory. Again, if the thickening of the skull in mollities is inflammatory, must we not consider the rarefaction of the long bones of the extremities and the bones of the trunk as being also inflammatory? To answer the last question in the affirmative seems to be to assume a conclusion for which we have not sufficient grounds.

IS "GENERAL PARALYSIS OF THE INSANE" NECESSARILY AN ANOMALOUS AND HOPELESS DISEASE?

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DR. HANDFORD'S article on certain atypical cases of general paralysis¹ has encouraged me to record my difficulties in accepting the axioms which have been laid down in regard to this disease, and the somewhat unorthodox conclusions at which I have consequently arrived.

General paralysis has on high authority been defined as an anomalous and inevitably fatal disease; and, indeed, from the way in which it has even lately been spoken of, one might almost suppose it to depend on some virus as definite as the bacillus anthracis. I cannot find, however, from a study of the literature of the subject, and from my own observation, that the requisite positive proof of such assertions has ever been satisfactorily afforded. Post mortem, the macroscopical and microscopical appearances are such as might result from ordinary morbid processes, modified (as they are everywhere) by the normal constitution of the affected part. Although remarkable and often characteristic, I do not know of one that may be called pathognomonic.

The prognosis in general paralysis is so dependent on its possible affinity with acute cerebral affections otherwise named, that the two questions may be considered together. In the central nervous system the symptoms attending "functional" disorders may closely resemble those of downright inflammation and degeneration, from which, indeed, these states probably differ only in degree. As a result of acute fevers &c., hyperkinesis and ankle-clonus may for a time be as well marked as in lateral sclerosis, or absent knee-jerk as in locomotor ataxy.² Between early general paralysis and early intoxication the likeness, pointed out specially by Dr. Wilks, is so well known that I need only recall it. The effect of hasheesh has been thus described: "After a few minutes I seemed to become two persons; I was conscious of my real self reclining on a lounge, and why I was there; my double was in a vast building made of gold and marble, splendidly brilliant and beautiful beyond all description. On another occasion my double seemed to be a sea, bright, and tossing as the wind blew; then a continent. I lost all conception of time. I arose to open a door; this seemed to be a million years.....I felt an unusual fullness of mental impressions. I understood clairvoyance, hypnotism, and all else. My thoughts flew with enormous rapidity." So after taking opium or inhaling chloroform. We may suppose that, in Dr. Hughlings Jackson's language, there is in all these states a temporary dissolution at the highest level with super-activity below. But after prolonged disturbance a return to the normal may occur, as in ordinary "mania," without physical symptoms. It now and then

happens, however, that such cases lapse into general paralysis. Again, a more or less permanent recovery may take place after a disorder, not only prolonged, but extending to lower levels, as in the following cases:—

1. A big man, aged thirty-two; much alcoholic and sexual excess. Boisterous excitement, varied by emotional depression. Says repeatedly that his brother has millions, and he himself can have as much as he likes. Pupils equal; knee-jerk brisk; tongue tremulous; slight slurring of syllables. He recovered in six months, and has had no relapse (two years).

2. A temperate man, aged forty-five; hard worker and keen politician. Stupor, with slight indications of general paralysis. Well in six months and returned to work. Readmitted "a typical general paralytic" three months later. Died, worn out, after a few weeks' intense excitement.

3. A man aged thirty-eight; irregular life and recent domestic calamity. Extremely ill; large syphilitic ulcers; sordes; tongue raw and tremulous; pupils unequal. Sleepless and restless, shouting that he is worth millions, the greatest man in the world, &c. In two months there was general improvement; no delusions, slight emotional depression; still tremor and unequal pupils; retinæ healthy. Discharged in four months, being quite rational and intelligent; knee-jerks a little brisk; no tremor or inequality the last six weeks.

Amongst other similar cases, a remarkable one is that of a man aged thirty-one, who, after some eccentricities, became maniacal, with much emotional exaltation, extravagant boasting, letter-writing to the Queen, masturbation, self-decoration with trumpery, &c. As this subsided (after close on two years) he became taciturn and hypochondriacal with loss of expression, thickness of articulation, fibrillar tremor, and incapacity for exertion. These threatening symptoms gradually vanished, and since (nearly three years) he has been in constant and responsible employment.

These disorders seem to form a series in which the prospect of recovery partly depends, as in other organs, on the intensity and persistency of the irritation, and the consequent amount of structural change, as well as the constitutional state. It must be remembered that in the cerebral cortex morbid processes are peculiarly modified by anatomical conditions, by extreme instability, and by intricate connexions with the rest of the system. There are many diseases which are apt to have more serious results when occurring for the first time after adolescence; and the changes which have taken place in the brain of even a healthy adult may have much to do in determining the usual progressive degeneration. Nor does speedy relapse after apparently complete recovery seem so remarkable when we consider that not only do all predisposing causes remain as before, but that in very few instances can the brain be guarded like a recently inflamed lung or sprained ankle. Indeed, the unlucky convalescent is in most cases beset as soon as he leaves the asylum by domestic and business worries, temptations to excess, and all the other disturbing influences of modern civilised life.

Even if the possibility of some specific process be admitted, it does not follow that in general paralysis recovery is altogether out of the question. The same has been said of such a specific disorder as acute tuberculosis, but there is good evidence to show that this is not the case.³ In the present state of our knowledge, however, may not the phenomena be at least fairly well accounted for upon general considerations, without recourse to a hypothesis difficult to reconcile with ordinary rules of pathology? I cannot help thinking that we have here an instance of that unfortunate property which names and definitions seem to possess, of causing endless confusion and futile discussion. "General paralysis" was probably originally used merely as a clinical term, and applied to those later stages in which there is obvious, general, and permanent muscular affection. As it was found that such a condition was often preceded by acute brain disorder, the term would be extended to include the whole series of events. Next arises the theory that general paralysis is a hopeless disease, and that no case which permanently recovers is pathologically akin to true general paralysis, however close the resemblance up to a certain stage. Arrest of general paralysis cannot, of course, be expected when such progress has been made as to interfere with general

¹ THE LANCET, Dec. 15th, 1888.

² Angel Money, Brit. Med. Jour., Oct. 29th, 1887. Bristowe, *ibid.*, Feb. 4th, 1888.

³ Westminster Hosp. Reports, vol. i.; Erichsen's Surgery, vol. ii., 1888.