

tissue; for when unrestrained, this material, as in the suprapatellar pouch, may measure an inch and a half in thickness.

As the above may seem but a contradiction and an opinion, though substantiated by numerous specimens both in my own possession and in the museum at this hospital, it is possible to show that the same holds true in other parts of a bone. For example, in central osteitis resulting in suppuration, as in the extremities of the tibia, it is well known that there is at first no enlargement; and if the inflamed area be small, the pus may remain for a long period enclosed within the bone, without any increase in size taking place. When, however, the inflammatory process has reached the periosteum, then, and I would say only then, is there any enlargement.

The example of enlargement usually explained by chronic osteitis is that where on section the bone is found uniformly cancellous to the outer compact layer. Such a bone is said to be "expanded" by an enlargement of the normal spaces, or to be "swollen." Thus Sir Jas. Paget,<sup>1</sup> after describing the process of inflammatory softening in bone, proceeds to show the subsequent changes that are permitted, "especially the swelling and expansion." In the first specimen used to illustrate the subject, that of a humerus removed from an arm four months after excision of the elbow, 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." In describing the next illustration, which exhibits an increase in diameter, due to the presence of regular laminae outside a compact layer, Sir James says, "The most striking change is a more or less extensive and wide separation of the concentric laminae of the walls of the bone, so that, as in the section of this femur (Fig. 46), the longitudinal section of the enlarged wall appears composed of two or more layers of compact tissue with a widely cancellous tissue between them, and these layers may sometimes be traced into continuity with those forming the healthy portion of the wall." In a previous paragraph he describes bone as "yielding and extending as the naturally softer tissues do in an inflammatory swelling."

I venture, in opposition to this explanation, to submit the following, founded upon the observation that all enlargement seems, from the examination of specimens, to be periosteal. The uniformity on section is explained in two ways. In some specimens, as in Sir James Paget's first case, I would suggest that there is periostitis at the same time as the osteitis. That the former process lays down new bone and increases the diameter, while the latter leads to absorption of the bony framework, and to widening of the cancellous spaces and of the smaller cavities in the compact layer. The new bone being itself cancellous, and the compact having become so, there is an unbroken area of this material from the medullary canal to the thin compact layer bounding the new periosteal bone. The result is that expansion appears to have taken place. When the inflammatory products have organised and the bone becomes sclerosed, and when the dense periosteal layer is blended with the original compact tissue, the latter can still be traced by its greater whiteness, and usually greater density, an additional proof of the periosteal increase. In the same way it seems to me that the narrowing of the medullary canal may be explained by the formation of new bone, by the endosteum, and not, as Sir James Paget says, by osteitis alone. On this point he writes, "The inner layers of the wall are pressed inwards, and encroach upon the medullary tissue."

In other cases the periostitis is more chronic, and the osteitis superficial only, so that more or less of the old compact layer remains after all inflammation has subsided. Such a specimen, I fancy, is Sir James Paget's second illustration above referred to (Fig. 46). In such I would suggest the following interpretation. New bone is deposited by periostitis, the associated osteitis is superficial, and hence there is little cancellation of the compact layer from this cause. If the periosteal bone become permanent, with or without passing through the sclerotic stage, then the original compact layer becomes cancellous. This seems to be effected by a physiological process independent of inflammation. The compact layer now occupies the interior of the bone, it no longer has the function of compact tissue, and so slowly assumes the character of the surrounding material. This may actually be seen taking place, as in a specimen in the

Guy's Museum (No. 1153), in which the sclerosed periosteal bone has become blended with the compact layer. The cancellating process has in one part removed all distinction between the two. It seems, in fact, to follow from the functions of the several parts of a bone that, given a permanent increase in diameter from periosteal deposit, the surface of that deposit will become compact and blend above and below with the original compact layer of the bone; while the deeper part, though having become sclerosed, will, with the now embedded compact layer, ultimately become porous, and the two be indistinguishable from each other and from the original cancellous tissue.

In support of this view, it may be mentioned that the impacted compact layer in fractures becomes cancellous, suggesting that all parts within the outer compact layer must in a healthy condition of bone assume this condition.<sup>2</sup>

Smith also, in his work on Fractures (p. 161), in endeavouring to disprove the existence of impaction in Colles' fracture, says:—"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."

This functional modelling in new bone is seen in the union of fractures with overlapping of the fragments. Here the periosteal formation is the bond of connexion, and where it passes from the end of one fragment to the surface of the other it has an outer compact and an inner cancellous part.

The following statements embody the views above explained:—

1. That osteitis, even when central, is only attended with enlargement when the periosteum is involved.
2. That in all cases of primary synovial disease of joints, or of caries of articular surfaces, with secondary synovial disease, there is no enlargement of the bone.
3. That the enlargement of joint-ends in these cases is only apparent, and is explained by the unnatural prominence of the femoral condyles, owing to wasting of the thigh and to the thickening of the ligaments and synovial membrane.
4. That all inflammatory increase in the size of bones is due to periosteal formation, and that the uniformly cancellated appearance is due (a) to the widening from absorption of the spaces in the compact layer, whereby it becomes indistinguishable from the original cancellous tissue and the porous periosteal bone; and (b) to the remodelling of the compact layer when the osteitis has been superficial.
5. That the term "expansion" is misleading as applied to the results of the inflammatory processes in bone, since the widening of the spaces is an absorptive one.

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## ON THE GRANULAR MATTER OF THE BLOOD.<sup>1</sup>

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THE blood may shortly be described as consisting of a fluid or plasma, and a solid or corpuscular portion. The latter is composed chiefly of two varieties: (1) the white corpuscles or leucocytes; (2) the red corpuscles or erythrocytes (Alex. Duane). In addition, there is also a considerable amount of granular matter; and it is to some of these granules that my attention has recently been directed. If a drop of blood be smeared on a slide, rapidly dried, stained, and mounted, after the manner proposed by Koch, as a ready method of examining fluids for micro-organisms, it will be found that certain portions only are stained—viz., the nuclei of the leucocytes, whilst the body as a rule remains clear; sometimes the whole corpuscle becomes stained. The red discs do not take up the colouring matter unless the staining is excessive, and then only to a slight degree. It is somewhat surprising at first how, in a

<sup>2</sup> An impacted fracture of humerus, No. 1111 (50) shows this.

<sup>1</sup> In justice to the author we feel it desirable to state that the MS. of this article came into our hands in the middle of November last year, prior, therefore, to the publication of Bizzozero's researches, commented on in our issue of Jan. 21st.—ED. L.

well-prepared specimen, the characteristics of the individual corpuscle are retained. In addition to the nuclei of the leucocytes, numerous small spherical granular bodies may be seen arranged for the most part in groups of five to twelve, like grapes on a stalk, though occurring also singly; but their depth of staining is, however, less than that of the nuclei of the white corpuscle. To see these little bodies well a magnifying power of at least 600 diameters is required. The question naturally arises, Are these little bodies the result of some change, some breaking up of the corpuscular elements during the process of drying, to which the preparation has been submitted, or do they naturally exist in normal blood? My impression is that they actually do exist, as they are well seen in blood in which the greatest precautions against change have been taken, as in using the warm stage. They can also be seen on the cold stage (0°C) and by the ordinary method of examination. An attempt to estimate their number must necessarily meet with considerable difficulty owing to their tendency to group together, but the result of several enumerations by means of Gowers' hæmacytometer gives from 250,000 to 300,000 in a cubic millimetre of blood. Spherical in shape, their size varies from  $2.5\ \mu$  to  $4\ \mu$ . At times these bodies appear to be fugitive, as they are seen to become rapidly fainter and disappear even whilst under the eye of the observer; this is probably due to the fact that their refractive index varies but little from that of the plasma, and as the plasma becomes tinted with hæmoglobin so does the refractive index of the two become approximated. By far the best demonstration of these racemose bodies can be obtained by allowing a drop or two of Hayem's corrosive sublimate solution or Gowers' salt solution to flow under the cover-glass; it will then be seen that, in addition to the physical properties already enumerated, they possess that of adhesiveness, remaining sticking to the slide or cover-glass whilst the corpuscles are washed away. They also assume a faintly greenish-yellow colour, become somewhat flattened, and lose their granular appearance. In the examination of blood in disease I have not been able to satisfy myself that there is any variation with regard to the shape or number of these racemose bodies; but in a case of pernicious anæmia my observations have varied, in some samples of the blood their number was far below, whilst in others it was very much the same as in normal blood. In observations on the blood of animals the racemose corpuscles can be clearly seen, and in specimens of dog's blood taken from the superior and inferior cavæ, portal vein, and splenic artery and vein, there is no appreciable difference in their number. They are absent from the scrapings of lymphatic glands, spleen, and the medulla of bones in the dog. All these facts, together with their power of staining with methyl-violet, and their similarity to the processes thrown out by a living leucocyte, have led me to the conclusion that these bodies are broken-up white corpuscles, or portions detached from the same.

Various observers, both English and foreign, in writing on the blood, have described small bodies as to the origin and ultimate destination of which different views have been held. Zimmerman<sup>2</sup> described small vesicles which, he believed, ultimately became fully developed red corpuscles. Virchow also states that little corpuscles do undoubtedly occur in the blood; he thinks, however, that they are not destined to become red corpuscles, but are, on the contrary, old ones breaking up. Riess also mentions the existence in the blood of small corpuscles, and cautions the observer not to mistake them for micro-organisms. Cornil and Ranvier, as well as Marius and Vanlair, describe, under the head of "microcytes," small spherical, highly refractive, red granules. But, perhaps, the most elaborate description is that of M. Hayem.<sup>3</sup> In a series of papers he brings forward evidence to prove the existence in the blood of man of elementary red corpuscles, which he has called hæmatoblasts. He states that Marius, Vanlair, and others are inaccurate when they describe the existence in blood of small spherical granules, and that his own physiological and clinical researches show that these so-called microcytes are only globules modified by external agencies—in other words, that they do not exist in the blood. His further description of the hæmatoblast is very similar to that described by myself above, with the one great exception that he regards their shape as discoidal instead of spherical. To demonstrate

this clearly the blood is irrigated with a corrosive sublimate solution. It seems to me that blood irrigated with any fluid can hardly be called in a normal condition, and I venture to think that his statement with regard to their discoidal shape is true only of blood that has been placed under abnormal conditions by irrigation. I myself have not been able to verify his conclusions though the granules lose their crenation and become somewhat flattened as mentioned above. More recently Dr. Norris<sup>4</sup> of Birmingham, in a series of papers which he has written on the existence in the blood of a third corpuscular element, alludes to the presence of small granular bodies which he recognises as Hayem's hæmatoblasts, but affirms that these hæmatoblasts are simply due to the breaking up of his third corpuscle. My observations may be summed up as follows:—

1. That there exist in the blood small spherical granular bodies arranged either singly or in groups.
2. That these small bodies stain with the aniline dyes in a similar manner to nuclear matter.
3. That they are fragments of white corpuscles, or portions detached from the same bodies.

## TROPHIC SKIN CHANGES OF NEUROTIC ORIGIN.

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### I. HERPES ZOSTER.

SINCE the eruption on the skin known as Herpes Zoster has been recognised to be a manifestation of nerve trouble, observers have been on the *qui vive* to identify other lesions of the skin as due to a similar cause. The list of trophic disorders of the skin in immediate relation with nerve lesions is already a long one, and it is even probable that it will be added to in future, not so much by the recognition of fresh types of skin affection, as by the inclusion of some of those with which we are already familiar, but which have been thought to be due to constitutional and other causes.

Any association which may exist between particular types of skin eruption and special varieties of nerve lesion is at present almost entirely matter of surmise.

I propose to collect together under convenient headings cases bearing on the subject, most of which have already been recorded, not narrating them in detail, but merely stating illustrative facts. References will be given as a help towards the bibliography of the subject. It will be convenient to deal with those eruptions first which are associated with local nerve lesions. Having shown what is possible under such circumstances, the ground will be somewhat cleared for conjecture with regard to nerve conditions of a more general character in their relation to cutaneous phenomena.

The eruption of herpes zoster is in its full development altogether *sui generis*—no one, having once seen it, fails to recognise it again. The clusters of various-sized vesicles following the distribution of the cutaneous nerves, the confluence of some of the vesicles to form shallow bullæ, are too well known to need minute description. In addition there is the definite clinical course pursued by the eruption, which is in itself quite characteristic. The symptom *pain* is such a prominent one in herpes zoster, and corresponds so closely to that which is described by patients suffering with neuralgic affections, that authors soon began to describe it as neuralgia. It may be noted, however, that intermittency or remittency is not laid special stress upon by patients suffering with herpes zoster; nor do they often complain of the violent paroxysms such as accompany ordinary neuralgia of the fifth or sciatic. Entire lack of proportion between the severity of the pain and the cutaneous symptoms is constantly observed, and their relation as to time is so inconstant, that the pain may precede the eruption for days or continue after its disappearance for months. It is asserted by some observers (e.g., Bärensprung) that herpes zoster sometimes occurs without any neuralgia being present at all; indeed, this may be said to be the rule with children amongst

<sup>2</sup> Archives f. Path. Anat. und Phys., vol. xviii., pp. 221-242.

<sup>3</sup> Comptes Rendus de l'Académie des Sciences, vol. lxxxv.

<sup>4</sup> Birmingham Phil. Proc., 1880.