on behalf of the Association, our most grateful thanks for their excellent, suggestive, and practical discussion of the question.

Dr. CLOUSTON—I shall not venture to say anything more than that I am sure Dr. Tuke and myself are well satisfied with the results of the debate; and I would only add this one remark. I am very much disappointed that the officers of asylums who adopt a routine treatment of putting their patients to bed for a few days or weeks have not defended their practice during this discussion. I think that the practice goes by default, and probably will not be longer continued. With regard to the various scientific questions involved, I am certain that they have been thoroughly thrashed out, and that almost everything I was to have said in answer to Dr. Tuke has been better set forth by other speakers. It only remains for me to thank you, gentlemen, for your kind attention.

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*The Pathology of Milkiness, Thickening, and Opacity of the Pia-arachnoid in the Insane.** By W. F. ROBERTSON, M.D., Pathologist, Royal Asylum, Edinburgh.

There is at the present time great need of more complete and definite knowledge as to the pathology of the very marked structural changes that so commonly affect the pia-arachnoid in the insane. The subject is one of much importance to all of us as medical psychologists, for not only is the condition in question one of the most conspicuous lesions associated with mental disease, but it implicates a structure of primary importance in the economy of the central nervous system. It is by way of vessels that course through this membrane that nutriment is conveyed to the brain cortex, and the waste products resulting from metabolism in the cerebral tissues are mainly conveyed away in the fluid that circulates in its lymph spaces. Therefore it is evident that these morbid changes may very seriously interfere with the functions both of nutrition and excretion in the brain.

The subject has quite recently been fully gone into in two papers of a series published in conjunction with Dr. James Middlemass in the "Edinburgh Medical Journal" (1), but I am now able to add a number of new points to the statements there made.

I shall not describe the various naked-eye appearances that the condition presents. With these you are all already perfectly familiar, as well as with the different forms of mental disease with which they are specially associated. As you also know, the change is not confined to the insane. It usually occurs in some degree in people dying after middle

* Read at the Annual Meeting of the Association, 1895, and illustrated by a microscopic demonstration.

age, but, even apart from conditions of senility, it is occasionally seen in patients who were not mentally affected. Beyond all question, however, it occurs far more frequently in the insane than in the mentally sound, and it is commonly developed in the former to a degree practically never seen in the latter, except occasionally in drunkards.

Before passing to the description of the microscopic changes it will be well to briefly state the various opinions that have been expressed as to the pathology of milkiness and thickening of the pia-arachnoid. Bayle (2), writing in the early part of the present century, regarded the condition as a chronic meningitis, which he believed must play the principal *rôle* in the etiology of insanity. Bevan Lewis (3) is of opinion that in its extreme degrees "we must infer an inflammatory agency." In its slighter manifestations, and especially in senile atrophy of the brain, he thinks it may occur apart from inflammatory action. In all cases he attributes much importance to the effect of frequent congestive conditions or chronic hyperæmia. Ziegler (4) describes two separate conditions, one affecting mainly "the arachnoid and sub-arachnoid tissues," and the other involving chiefly "the pia and underlying nerve tissue." The former he terms "chronic arachnitis or external leptomeningitis," and the latter "atrophic meningo-encephalitis." Though thus committing himself in his terminology to an inflammatory theory, he states that he doubts if the first form is always inflammatory, and that the second in its inception is mainly dependent upon degenerative changes. Batty Tuke and Woodhead (5) also practically adhere to the inflammatory theory of Bayle, though they attach considerable importance to "occasional pathological congestion superadded to the normal mechanical obstruction produced by the peculiar anatomical relations of the vessels to the longitudinal sinus." Dr. Batty Tuke, in his more recent work on "The Insanity of Over-exertion of the Brain" (6), further attributes the morbid change to "a deposit of waste and plastic exudates. As these accumulate and diffuse the membrane becomes thick, tough, and on section is found to consist of a mass of material which looks like an immense increase of the normal trabeculæ." The best account that we have of the microscopic changes is undoubtedly that of Ziegler. The condition that he terms "chronic arachnitis" is due, he says, to fibrous thickening, endothelial hyperplasia, and more rarely to cellular infiltration. In early cases of "atrophic meningo-

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encephalitis," in which he thinks that the changes may be degenerative only, the white turbidity is due, he says, "to accumulation of small globules and granules of fat, fatty and broken-down cells, and occasional fat granule cells." In many cases from the first, and in all advanced cases, the most important change is the small-celled infiltration that pervades the pia mater, and, to a less degree, the subarachnoid tissues. Other authorities are pretty generally agreed in describing the microscopic changes as consisting in an infiltration of the tissues with round cells and an increase in the fibrous elements.

In the papers referred to Dr. Middlemass and I have been obliged to differ to some extent from these views, both as to the nature of the pathological process and the textural changes that occur. We are unable to see that there is any warrant for Ziegler's classification, and would in the meantime consider all the changes to be observed in typical cases as manifestations of one morbid process, making the reservation that in advanced general paralysis, and probably also in syphilitic insanity, there is superadded a distinct and more active process. To this subject I shall return after I have given a description of the textural changes that occur according to my own investigations. In order to render this description intelligible, however, it will be necessary to make some observations upon the normal structure of the tissues involved.

It is usually taught that there are two distinct membranes —an outer delicate, non-vascular layer of fibrous tissue which bridges the sulci without dipping into them, and an inner vascular membrane which closely invests the whole of the cerebral surface. Between these two layers there is said to be a considerable space (the "sub-arachnoid space") traversed by numerous trabeculæ, a spongy lymph-sac being thus formed which contains the cerebro-spinal fluid. Dr. Batty Tuke (7) has dissented from this commonly-received view of the constitution of the pia-arachnoid. He holds that it should be looked upon as only one membrane, of which the so-called arachnoid is merely the outer layer. If I understand him aright he bases this view upon the belief that over a convolution the two layers are intimately bound together, leaving no spaces containing cerebro-spinal fluid. He is otherwise in accord with the usual descriptions of the microscopic structure, except that he holds that the vessels are distributed between the two layers instead of in the

inner layer. Now, while I think that Dr. Batty Tuke's idea of the pia-arachnoid as one membrane is a step towards a more correct conception of its constitution, yet I venture to maintain that in certain other respects his view, like that generally taught, is a mistaken one. It can be demonstrated that his statement regarding the distribution of the vessels is only correct for the large arteries, and his contention that the two layers of the membrane are intimately bound together over the convolutions, leaving no so-called sub-arachnoid spaces, is also disproved by special methods of examination. By the employment of these methods it can be shown that the membrane has peculiarities of structure that have hitherto escaped notice, and a knowledge of which must, I think, lead to the adoption of a view of its constitution differing from either of those that are at present advocated. According to the present teaching there are three structures composing the pia-arachnoid,—an outer layer of dense fibrous tissue; an inner layer of a similar kind, but differing from it in being highly vascular; and an intervening trabecular tissue, which, according to Dr. Batty Tuke, is absent over the convolutions. I think that it can be shown that there is essentially only one structure throughout, and therefore only one membrane.

The minute anatomy of the pia-arachnoid seems to have been studied almost exclusively by means of transverse sections. These, however, fail to demonstrate the arrangement of the lymph spaces—a matter of the utmost importance. For the satisfactory examination either of the normal structure of the membrane or of the morbid changes that occur in it, it is necessary to use horizontal and oblique sections. Especially useful are superficial horizontal or surface sections, by means of which a high power view may be obtained of an extensive area of the free surface. This form of preparation introduces what I believe is a new and valuable histological method, of much wider application than the present, some of the results obtained by the employment of which are demonstrated before a medical society here to-day for the first time. The facts regarding the structure of the normal human pia-arachnoid that are revealed by the employment of these methods I shall as briefly as possible describe.

Taking first the membrane over a convolution, suitably stained, superficial horizontal sections show on the outer surface a single layer of flattened endothelial cells with large

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oval nuclei. On the inner surface of the membrane in apposition with the cortical tissue it is generally taught that there is a layer of cells of the same character. Its existence, though questionable, may be provisionally accepted. Between these two endothelial layers there is a structure, the basis of which is delicate white fibrous tissue. This tissue is collected into bundles or trabeculæ of varying thickness and length. The arrangement that these have is that of a large number of inaccurately superimposed and partially united, slightly flattened networks, lying for the most part parallel to the cerebral surface. The meshes of these networks, which are quite microscopic in size, form freely communicating spaces, which contain cerebro-spinal fluid. On the surface of the trabeculæ lining every individual space there is a continuous layer of flattened endothelium. The spaces vary greatly in size. They are largest in the centre of the membrane, a circumstance that explains its seeming division into two separate layers. In the sulci some of the spaces are specially large, and about the base of the brain and along the upper surface of the corpus callosum there are some still larger cavities which form the arachnoid cisterns. Below the endothelium of the outer surface there is no distinct horizontal layer of compact fibrous tissue that can be properly regarded as a separate membrane. What is typically found is simply a layer of connective tissue of the same thickness as the subjacent trabeculæ, and formed by their arches. The same arrangement of trabeculæ and intervening lymph spaces is maintained to the inner surface immediately external to the cortical tissue. Thus the membrane has throughout the structure of a spongy lymph sac. Though most of the vessels lie in the deeper portions of the membrane, they may occur in any part of it. In almost every superficial horizontal section they may be seen immediately below the outer endothelial layer. The veins especially tend to lie near the outer surface. Thus the statement that the so-called arachnoid is a non-vascular structure is quite an erroneous one. The majority of the arterioles are large, being for the supply of the subjacent cerebral tissues. Capillaries occur chiefly in the deeper parts, but may occasionally be seen near the outer surface. They are, however, always remarkably few in number throughout the membrane. They are evidently little, if at all, required for its nutrition, which seems to be maintained by the cerebro-spinal fluid. Around the large vessels near the inner aspect the connective tissue

cells are more numerous, and the lymph spaces smaller and more elongated than elsewhere, so that the tissue seems to be of a denser structure. It is the modified appearance that these slight differences produce in transverse sections that has doubtless led to the belief that there is an inner layer of a distinct structural character. Horizontal sections, however, prove that the structure is essentially the same throughout.

I admit that in some, even horizontal preparations, it is difficult to see the lymph spaces in the external and internal denser layers of the membrane. But as a rule they are quite distinct, and when they are not their existence is attested by the presence of endothelial cells, the outlines of which can be seen in silver preparations. At most these relatively dense portions of the membrane are but loose areolar tissue in close proximity to quite evident lymph sinuses. They must therefore be freely permeable by the cerebro-spinal fluid, which is the important point, and one that is amply confirmed by pathological states in which such tissue may often be seen to be clogged with *débris* in common with the larger and easily recognized sinuses. There is a fact regarding the arterioles of the pia-arachnoid that, I think, helps us to understand the true constitution of the membrane. It is that they have no proper adventitial coat. Immediately outside of the muscular wall there is a single layer of endothelium, which is continued down to the capillaries as is pointed out by Klein. Obersteiner (8) believes that this layer forms the outer wall of a lymph sac, a point that must be regarded, I think, as doubtful. Beyond this endothelial layer lying upon the muscular coat, the vessels, with the exception of some of the very largest of them, have no special investment of longitudinally disposed fibrous tissue, such as is found in the vessels elsewhere. They are, as it were, naked vessels surrounded by trabeculæ and lymph spaces. The trabecular tissue, however, practically forms for them a common adventitia. From a consideration of these features of structure, Dr. Middlemass and I have advocated the view that the whole extra-vascular structure of the soft membranes may be looked upon as the conjoined and hypertrophied adventitial coats of the pial vessels, the lymphatic spaces of which have undergone a special development so as to form a spongy lymph sac. The main object of this special development is doubtless to give to the brain the protective advantages afforded by its envelopment in a thin water-cushion.

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The same arrangement of trabeculæ and lymph spaces may be demonstrated in the layer of membrane that covers the arachnoid cisterns, and in the arachnoid of the spinal cord. These facts regarding the microscopic anatomy of the pia-arachnoid lead to the conclusion that it can only be correctly looked upon as consisting of one membrane. To meet the requirements of this view Dr. Middlemass and I have suggested the following modifications in the present terminology. "The term 'pia mater' in its usual acceptation is indefensible, but we would suggest that it may conveniently and without risk of confusion be applied to the whole membrane, including its vessels, synonymously with 'pia-arachnoid,' in which sense it is, indeed, frequently at present used. We would not dispense with the term 'arachnoid' as has been advocated by Dr. Batty Tuke. It seems to us a most useful and almost indispensable one. Its meaning, however, should be extended, so as to include all the trabecular tissue which stretches from the external to the internal endothelial layer. The word '*sub*-arachnoid' is anatomically inaccurate, and should therefore be disused, the terms 'arachnoid trabeculæ,' 'arachnoid spaces,' and 'arachnoid fluid' being employed instead."

I shall not detain you with a description of the peculiar dense fibrous tissue bands that may occasionally be observed, nor of the normal pigment cells and cell nests of the arachnoid. They are shown under the microscopes.

I come now to the description of the microscopic changes that occur in this structure when affected by the milky and opaque condition that is so common in the insane. These changes in typical cases consist briefly in a slow hyperplasia of the connective tissue, and of marked proliferative and degenerative changes in the endothelial cells lining the arachnoid spaces and in those of the outer surface. The fibrous tissue may also be affected by retrograde changes. The connective tissue overgrowth is in direct proportion to the degree of milkiness and thickening. It affects the whole membrane, but the outer and less vascular parts chiefly The new fibres tend to be thicker and coarser in structure than normal. Opacities are due to an extreme degree of this overgrowth, resulting in more or less complete obliteration of the arachnoid spaces. The endothelial proliferation may be very marked, slight or entirely absent. Like the fibrous hyperplasia it is, when present, usually most pronounced in the outer portions of the membrane. In the great majority

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of cases it is a well-developed condition, and its absence is quite exceptional. The proliferated cells frequently form dense aggregations in the arachnoid spaces. As observed in transverse sections they have been commonly mistaken for the small round cells of an inflammatory exudation. Their endothelial character, however, is, I maintain, beyond question. The nuclei, though often somewhat smaller, are otherwise morphologically identical with those of the normal endothelial cells of the trabeculæ and of the outer surface. This point is admitted by Ziegler for the earlier stages of the morbid process in a certain number of cases. In the more advanced stages, and in many cases from the first, he believes that there is also a small round-cell infiltration. As the result of my own observations I differ from the latter view. In many cases presenting an extreme degree of milkiness and opacity, I have found that the cellular elements preserve the endothelial type, and that areas of small round-cell infiltration, upon the vessel walls or elsewhere, very seldom occur. It is only in advanced general paralysis, in syphilitic insanity, and in the very rare and still obscure condition known as purulent infiltration of the pia-arachnoid, that such an aggregation of round cells is added to the other appearances. In several cases of early general paralysis I have found that leucocyte infiltration is absent, a fact that has important bearings upon the question of the nature of the disease. Even in some cases of advanced general paralysis such infiltration occurs only locally, and it may be to a very slight degree.

On the outer surface, in addition to the general proliferation of the endothelial cells, there are usually very numerous minute localized aggregations. They are most pronounced in senile insanity and in general paralysis. They constitute granulations of the arachnoid. These were first described in 1826 by Bayle (2), who speaks of them as "rounded, excessively delicate asperities." Their endothelial character was recognized by Meyer (9) in 1862. In transverse sections they appear as oval masses of cells extending about an equal distance above and below the level of the general surface.

It has been convenient to speak first of the proliferative changes that occur in the connective tissue and endothelial elements, but even more pronounced, and, I think, of even more important significance are the degenerative changes that are found, especially in the endothelial cells. These changes may frequently be observed to affect cells that have

not been undergoing active proliferation. A common degenerative process is one manifested by an infiltration of the cell-plate with small yellow granules, a condition that is frequently accompanied by a degree of vacuolation. These granules are slightly darkened, but not blackened by osmic acid, and are therefore not of a fatty nature. They are lighter in colour, larger and less numerous than the granules in the normal pigment cells, which in addition are only found sparsely scattered in the human subject, while every cell in a large area may be affected in the way described. Therefore, I conclude that this is a degenerative change in these cells, though at the same time it is one that may have a physiological basis in the normal pigment cells, just as in pigmentary degeneration of nerve cells the granules that replace the protoplasm are merely an increase in a normal element. The proliferated endothelial cells of the trabeculæ and of the outer surface may show a similar change, but their cell plates being very small it is less prominent. The proliferated cells of the trabeculæ are constantly being shed and carried away in the arachnoid fluid, coverglass preparations of which always show large numbers of them usually in a more or less shrivelled and disintegrated state. This simple breaking down into granular *débris* is probably the most common change that these proliferated endothelial cells undergo. A point of considerable importance is that osmic acid preparations, whether of sections or of coverglass specimens of the arachnoid fluid, prove that fatty changes do not occur to any great extent in this milky condition of the pia-arachnoid. The same opinion has already been expressed by Adler ([10]) though, as I have mentioned, an opposite statement is made by Ziegler. Another type of degeneration that occurs in these proliferated endothelial cells of the trabeculæ is one that may be provisionally termed hyaline. It is probably a change closely related, if not identical, with that which, in the endothelial cells of the outer surface, leads to the development of concentric bodies, of which I shall speak presently. Another, though somewhat rare retrograde change, is one that manifests itself in vacuolation and swelling up of the nucleus. It is worthy of mention here because on the opposite side of the sub-dural space, in the endothelial cells of the surface of the dura and of the dural perivascular canals, it is a common and important change. Extravasated red corpuscles and granular *débris*, resulting from their disintegration, are frequently to

be observed in the arachnoid spaces. Hæmatoidin granules and crystals often occur, especially in senile insanity, in association with miliary aneurisms of the pial vessels. These morbid elements, resulting from recent or old-standing hæmorrhage, are the chief causes of the smoky or rusty tint that the arachnoid occasionally presents in the fresh state. In many cases granular *débris* of various kinds is in great abundance in the arachnoid spaces. It is probably chiefly derived from disintegration of extravasated red and white blood corpuscles and degeneration of endothelial cells.

While all these degenerative endothelial changes already described can also be seen on the outer surface of the membrane, a very interesting further change can there be observed in the cells that have undergone the hyaline metamorphosis, of which I have spoken. It leads to the development of the concentric bodies that produce such striking appearances in many superficial horizontal sections of morbid pia-arachnoids and duras. These bodies were undoubtedly seen by Meyer in arachnoid granulations more than 30 years ago, but, as far as I have been able to ascertain, Obersteiner (8) is the only authority who has described them, though he does not do so fully. He calls them *corpora arenacea*, and believes that they are composed of carbonate and phosphate of lime. This is undoubtedly an error, as they are unaffected by dilute mineral acids. He makes no statement as to their origin.

I have studied these structures very carefully, both as they occur in the dura and in the arachnoid, and I have been able, as I have indicated, to trace their origin from endothelial cells. I shall confine myself here to a description of their development in the arachnoid, and shall not enter into the exceedingly difficult problems connected with their relationship to certain hyaline rods that are frequently found in association with them in superficial horizontal sections of the dura. They arise specially in the endothelial granulations already described. The early stages in their development are difficult to trace, owing to the fact that their marked affinity for certain stains is only assumed at a somewhat late period. As far as I have yet been able to trace the process, it is as follows:—The cell plate becomes first affected, assuming a homogeneous appearance and a slightly increased affinity for eosine in hæmatoxylin and eosine preparations. At this

stage the nucleus has an increased affinity for hæmatoxylin, but as the morbid change advances it gradually loses this affinity, becomes homogeneous and stains with eosine in the same way as the degenerated cell plate, with which it ultimately blends. A single homogeneous globule is thus developed from an endothelial cell. This may become a small concentric body, but more commonly, owing to the circumstance that several endothelial cells in a granulation are usually affected simultaneously, the hyaline globules, developed from several adjacent endothelial cells, coalesce into one large mass. This being apparently of a semi-fluid consistence, assumes a spherical form. Concentric rings appear subsequently, evidently owing to shrinkage. In many developmental forms there is an irregular central mass that stains more deeply with eosine than the peripheral portion. It may be that this central mass corresponds to the nuclei of the cells, but the point is doubtful. This deeper staining of the central portion is often maintained in the fully-developed concentric body. I have never observed any disintegrative changes in these structures. It is doubtful if they ever develop from the endothelial cells of the trabeculæ.

I shall not here fully describe the structure and development of the osteoid plates that are so common in the spinal arachnoid, though rare in that of the brain. I maintain that they are the result of a retrograde metamorphosis in arachnoid opacities. They arise by a peculiar change in the dense fibrous tissue of which these opacities are composed very similar to that which occurs in the intra-membranous development of bone. They may, therefore, probably be correctly termed osteoid. In my experience their infiltration with calcareous salts is rare. With few exceptions they are unaffected by the action of dilute mineral acids.

For the many details that I have omitted in this description of the morbid changes associated with milkiness and thickening of the pia-arachnoid, I must refer to the papers in the "Edinburgh Medical Journal" (1).

I come in conclusion to the consideration of the very important question of the nature of the morbid process at work in producing these changes. On this point I think I shall best attain the objects of conciseness and clearness by simply quoting the views already expressed by Dr. Middlemass and myself. "Excluding for the moment cases of advanced general paralysis and syphilitic insanity, we have

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seen that in the typical form of the lesion the changes consist in proliferation, degeneration, and shedding of the endothelial cells, accumulation of granular *débris* in the arachnoid spaces, and hyperplasia of the connective tissues. We would emphasize the fact that even in advanced cases small round-cell infiltration is usually entirely absent. We have seen no instance in which it had occurred to any considerable degree, excepting, of course, the rare cases of purulent infiltration which we have already alluded to. But most observers, looking upon the cells as leucocytes, have regarded their aggregation, accompanied by connective tissue overgrowth, as proof that the morbid process is of an inflammatory nature. We question, however, if the microscopic changes we have described can be correctly regarded as evidence of chronic meningitis, and we are inclined rather to adopt another theory.

"We have several times in previous papers argued for the view that the morbid changes which so commonly occur in the various envelopes of the brain in the insane are largely to be attributed to an abnormal trophic condition, in some way associated with the morbid energizing of the organ which they enclose, and it seems to us that the same influence may play a part in the production of this morbid change in the pia-arachnoid. The slight milkiness and localized opacities that occur in normal senility are especially, in all likelihood, merely trophic changes. But in insanity there is, we think, a still more important factor at work, and one the mode of operation of which can be expressed in much more definite terms. The arachnoid trabeculæ are practically non-vascular structures. Even in the deeper parts of the membrane, where the large vessels are most numerous, capillaries are few in number. The tissues must, therefore, it is evident, depend for their nourishment upon the arachnoid fluid which circulates in their spaces. This fluid, in addition to having origin from the choroid plexuses and the vessels of the pia-arachnoid, is derived from the lymph that flows through the cerebral lymphatics, which, after leaving the capillaries, supplies nourishment to the nerve-cells and fibres and connective tissue elements of the brain, and receives from them at the same time their waste products. Now in insanity these structures show profound morbid changes, and it is therefore evident that the waste products of their metabolism must be abnormal. There will thus be introduced into the arachnoid fluid substances

which it is easy to understand may seriously affect the nutrition of the arachnoid tissues. We, indeed, need nothing more to account for the histological changes we have described. These changes are frequently accompanied by what is generally regarded as a hypertrophy of the Pacchionian bodies, which it is generally admitted are excretory organs for the arachnoid fluid. Such a change in them would indicate an increased demand for the elimination of morbid products from the cerebral fluid, and therefore an abnormal condition of it. But the changes in the Pacchionian bodies in insanity have not been worked out, and it is possible that their enlarged condition is not altogether a true hypertrophy, but in part a morbid change, so that their excretory functions may really be diminished. This possibility is at least to be borne in mind as one which may constitute an additional cause of abnormality of the arachnoid fluid. We attach, however, greater importance to the introduction of morbid products from the subjacent brain. According to this view, which seems to us to be the most rational theory of the etiology of the milky and thickened pia-arachnoid of the insane, the endothelial proliferation and degeneration and the connective tissue overgrowth are due to abnormal, and perhaps in some degree irritative, qualities of the arachnoid fluid.

"Now, granting that this view is accepted, there are probably those who will stretch their definition of inflammation far enough to include within it such a process as this. It is, however, a different process from that which has been understood to occur by those who have looked upon the morbid appearances as the result of a chronic leptomeningitis, and if this name is applied to it there will be grave risk of conveying a false impression of its true nature. For our own part we think that it cannot correctly be spoken of as a chronic inflammation. It is a hyperplasia attended by marked degenerative changes. The fact that the deeper tissues are comparatively less affected by the morbid change than the rest of the membrane may perhaps be owing to the circumstance that the former are in part nourished by capillaries which supply them with a more healthy nutriment than that which is afforded by the arachnoid fluid. Whether this is the correct explanation or not, the fact is at least in direct opposition to the inflammatory theory."

There being no sufficient warrant for calling the condition a chronic leptomeningitis, I think that in the meantime it

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should be referred to merely by the naked-eye appearances that it presents. In those exceptional cases, which I have carefully defined, in which a small round-cell infiltration is added to the other morbid appearances, the existence of an inflammatory element is of course beyond question.

I shall not further detain you with a discussion of the question why the morbid changes are most marked over the convexity of the hemispheres. It has been fully gone into in the published papers.

There is just one other point to which I wish to refer. It is that on the opposite side of the subdural space—that is to say, in the tissues of the dura—I have found that there are also specially prone to occur in the insane morbid changes of the same kind as those I have been describing in the pia-arachnoid. By the use of superficial and deep horizontal sections it can be shown that similar proliferative and degenerative changes affect not only the endothelium of the inner surface of the dura, but also that of the peculiar perivascular canals. This lesion in the latter situation, with an associated weakening of the capillary walls, is, I maintain, the explanation of the proclivity of the insane to the formation of subdural membranes. The subject is a complicated and difficult one, and I shall not further pursue it here. The interest of the fact that I have mentioned, and the importance of the generalization that it involves, must be apparent to all of you.

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