ON THE MINUTE CHANGES OCCURRING IN VESICULAR EMPHYSEMA.

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IT is the prevailing view with respect to the textural alterations in emphysema that they are of a degenerative and atrophic character throughout. These changes, according to the researches of Rainey, Waters, Rindfleisch, Eppinger and others, may be summed up as loss of elasticity and degeneration of the elastic fibres, stretching and progressive wasting of the capillaries, and granulo-fatty degeneration of the corpuscular and connective tissue elements of the air cells. Perforations of the alveolar walls ensue which lead to fusion of the air cavities. A hypertrophy of the muscular fibres around the infundibular openings has likewise been noted along with this degeneration. Some regard the wasting of the capillaries as primary and so causative of the nutritive changes. This opinion is adopted in some of our textbooks—e.g., Dr. Woodhead's. Others, again, amongst whom may be mentioned Hertz and Ziegler, consider the de-

FIG. 1.

Stage of reaction. *a*, capillaries, showing outlines of red corpuscles; *b*, epithelial cells, enlarged and dividing; *c*, connective-tissue corpuscles; *d*, elastic fibres. (Zeiss, $\frac{1}{2}$ in. oil immersion.)

generative change in the walls to be antecedent to and so far determining the destruction of the vessels.

The observations which I have been enabled to make are such as to negative the supposition that in emphysema there is wholly a degenerative and atrophic process. On the other hand, I find that there are two stages in the morbid anatomy of the disease: one reactive and productive, the other degenerative or atrophic. Further, the conception of the changes according to this scheme renders clear the priority of the textural alterations and places the vascular atrophy as secondary.

In a portion of lung about to become the seat of a compensatory emphysema the first effects of the mechanical strain are a general dilatation of the part and a reduction in the velocity and volume of its blood circulation. The dilatation extends to the smallest bronchioles, alveolar ducts, infundibula and air cells. Seeing that the air cells are mutually arranged like the cells of a honeycomb, their expansion must in the first instance be mainly uniform in all diameters and not, as some have supposed, a mere flattening out, in order to effect which an extensive destruction of the septa would be necessary. The slowing and diminution of the circulation are caused by (1) impairment of the normal recoil, (2) the expelling influence of the air at a high pressure, and (3) stretching of the vessels, more especially those disposed round the mouths of the alveoli. A great deal depends, however, on the extent and site of the involved

part and the surrounding conditions, as the size of capillaries is chiefly regulated by the amount of blood, and a heightened pressure would overcome a certain degree of stretching, so that in the early stage of emphysema it is sometimes difficult to detect much difference from the normal in the size of the capillaries. Now, as to the air cells, the first effect of the increased air tension is to produce a compensatory reaction in the respiratory elements. It is a mistake to suppose that they immediately submit to the injurious influence. Both the epithelial and connective tissue cells enlarge, and many of them divide. (Fig. 1.) They behave, in fact, like any other tissue on which an inordinate demand is made, and this hypertrophy is in so far the analogue of what occurs in the muscle nuclei in the infundibula. I have carefully guarded against the possibility of confounding this change with a merely inflammatory phenomenon by comparing the emphysematous and non-emphysematous portions of the same lung. It is true that Villemin, Klob and Wunderlich have noted an increase in the intercapillary nuclei in emphysema; this, however, they regarded as inflammatory and unconnected with the heightened air pressure. But the phenomenon I describe is compensatory and as such may not inaptly be compared to what occurs in certain cases of atheroma. In these the increased tension in the vessel leads to swelling and division of the nuclei in the intima. As in

FIG. 2.

Degenerative stage. *a*, capillaries, showing red corpuscles compressed by swollen endothelial nuclei; *b*, granular and fatty condition of intercapillary elements; *c*, perforations in wall; *d*, endothelial nuclei; *e*, collapsed capillary; *f*, elastic fibres; *g*, red corpuscles, broken up and granular; *h*, leucocytes in capillary; *i*, granular portion of capillaries devoid of corpuscles. (Zeiss, $\frac{1}{2}$ in. oil immersion.)

atheroma also, this nuclear change is accompanied by a swelling of the areolar ground tissue. The result of this alteration is a widening of the capillary meshes, with consequent narrowing of the capillaries. Many of the endothelial nuclei of the capillaries likewise enlarge and cause a permanent constriction of the vessel opposite the point. (Fig. 2.)

The reaction of the respiratory cells is not of long duration; they enter sooner or later on the degenerative stage. The protoplasm becomes granular and fatty and the nuclei vacuolate and fall to pieces. (Fig. 2.) The part, has, however, become permanently enlarged through atony of the elastic fibres, and many of the finest of these show a granular change—a finely beaded state of the fibrils. Small holes appear in the granular protoplasm, which speedily enlarge, and in this way many of the septa become completely destroyed. The capillaries undergo the following further changes: their walls become increasingly attenuated and the pressure of the endothelial nuclei blocks the circulation at many points; the red corpuscles become elongated and some of them undergo a granular degeneration (*g* in Fig. 2); many of the capillaries soon come to appear as whitish lines stretching across the granular substance. It was asserted by Isaaksohn¹

¹ Virchow's Archiv, Bd. liii., p. 466.

that the vessels were blocked by what he terms "white thrombi"—i.e., colourless blood-corpuscles. He affirmed that the alteration began by a granular change in the endothelial walls of the capillaries, so that the characteristic white lines did not appear on treatment with nitrate of silver. On these granular places white blood-corpuscles were deposited and became so firmly fixed that even a strong injection would not drive them away. New homogeneous deposits were gradually formed over these cells, the whole obstructing mass soon undergoing a fatty degeneration. Now I am not disputing these conclusions in the main, for it is evident that a granular state of the capillaries ensues, and it is likewise true that leucocytes are frequently found in the vessels at points where they are breaking down; but it is too much to assert that this is the chief or only mode of obstruction of the capillaries, for they are often seen interrupted at points where only red corpuscles are to be found, and in particular I would draw attention to their being intercepted by endothelial nuclei in the manner described.

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THE RELIEF OF INTRA-CRANIAL PRESSURE BY THE WITHDRAWAL OF CEREBRO- SPINAL FLUID FROM THE BASAL SUBARACHNOID CAVITY.

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It is a well-recognised fact that in many affections of the brain the cerebro-spinal fluid is increased in quantity and that this is sometimes associated with increased intra-cranial pressure. The best known example is that of chronic hydrocephalus, in which disease the increased pressure leads to separation of the cranial bones and other symptoms. There is no doubt that in this instance the increased pressure is actually due to an excess of cerebro-spinal fluid, but it by no means follows that increase in the quantity of cerebro-spinal fluid is always associated with increased intra-cranial pressure, any more than that increased pressure is always associated with excess of the fluid in question. Briefly stated, the clinical conditions in which the cerebro-spinal fluid is increased in quantity are (a) meningitis, simple, epidemic or tuberculous, especially the last-named; (b) chronic hydrocephalus; (c) general paralysis of the insane and some cases of dementia; and (d) as a direct result of injury to the skull. Pathologically it is met with as a result of (a) meningeal inflammation, basal or cortical; (b) ventricular inflammation or ependymitis; (c) growths pressing on the veins of Galen; and (d) shrinking of the cortex from degenerative changes. Anatomically considered the fluid occupies the spaces between the pia mater and arachnoid, these being especially large at the base of the skull and beneath the cerebellum; a smaller amount is contained between the arachnoid and dura mater. Physiologically there is every reason to believe that the fluid is secreted by the ependyma of the ventricles and by the cells of the pia mater and arachnoid. The fluid appears to be constantly secreted and as constantly absorbed along the perivascular lymphatic vessels. In a case on which I operated some months ago, after removing a portion of dura mater from off the cortex, drops of cerebro-spinal fluid could be seen to collect on the dural aspect of the arachnoid and, running together, trickle down the surface of the brain to disappear beneath the cut edge of the dura mater, exactly like water collecting on the window of a railway carriage. That an increase in the amount of cerebro-spinal fluid is detrimental to life has been much discussed from time to time. When the fluid simply takes the place of shrunken convolutions and there is no coincident increase of pressure, as in many cases of dementia, it may fairly be argued that the increased amount can do no harm; but when the intra-cranial pressure is increased it is apparent that the excess of fluid can in no case be compensatory, for it would be equally rational to say that a pleuritic effusion compensates for a collapsed lung lying in an expanded chest, thus confusing cause and effect. As it is unlikely that the brain itself can swell so as to increase the pressure within the cranium (the question of

growths being neglected), it must be conceded that any increase of cerebro-spinal fluid coincident with an increase of intra-cranial pressure must be in the relation of cause and effect. As to the way in which the increase of fluid is brought about, it seems very probable that in irritative lesions the secretive cells are stimulated, over-secretion takes place and absorption by the lymphatics is interfered with. It is easy to imagine how dilated vessels can compress the lymphatic trunks which surround them and so further aid lymphatic obstruction. Perhaps the most frequent cause of increased intra-cranial pressure from over-secretion of cerebro-spinal fluid is tuberculous meningitis. The common condition post mortem is dilatation of all the ventricles, a rough or granular condition of the ependyma, increase of the cerebro-spinal fluid especially at the base of the brain, and more or less softening around the ventricles; in some cases the cortex is adherent to the surrounding pia mater. These cases were originally designated "acute hydrocephalus." It is a debatable point whether the softening is due to inflammation, transudation of fluid, or to the actual pressure exerted by the fluid; but for the purpose of my argument this is immaterial. My object is to show that removal of the excess of fluid by gradual drainage is of the highest importance in aiding the recovery, not only of patients suffering from meningitis of any kind, but also of those suffering from any of the diseases in which there is increased pressure from over-secretion of cerebro-spinal fluid. Taking the case of tuberculous meningitis again, it must be noted that there are usually three stages, known as "premonitory," "irritative," and "comatose," and from a study of the symptoms during these stages it is a fair conclusion that during the first two there is a gradual accumulation of fluid which leads to the comatose stage by a gradual increase of pressure on the various parts of the brain. There is a great similarity between the early diffuse symptoms of a cerebral tumour and those of tuberculous meningitis, and it seems only reasonable to infer that the gradual increase of pressure is the cause in each case, whilst the final stage of coma can only be explained on the supposition that the increased pressure leads to an annihilation of function by general compression. A sudden increase of intra-cranial pressure, as in cerebral hæmorrhage, leads to similar results. It is a matter for physiological experiment how much the intra-cranial pressure has to be increased in order to interfere with the circulation through the small cerebral veins and arteries, for this seems to be the way in which the symptoms are produced and is probably the cause of the softening around tumours and in cases of tuberculous meningitis. Arguing in this way, it seems only reasonable to conclude that removal of the excess of cerebro-spinal fluid in cases of tuberculous meningitis will relieve the symptoms and give the patients a greater chance of recovery by enabling them to battle with the tuberculous infection. The so-called natural drainage by the vertebral canal seems to be altogether inefficient. Reasoning from analogy, one would say that recovery from peritoneal or pleuritic tuberculosis, after removal of ascitic or pleuritic fluid, fully justifies the suggestion that a similar interference in cerebral tuberculosis may have a good result, with, however, the proviso that, inasmuch as the brain is a very much more delicate organ and much more likely to be damaged by pressure, the interference must be early—i.e., in the irritative stage. A similar argument holds good in meningitis from other causes and in cases of compression from over-secretion of cerebro-spinal fluid following injury. Concerning general paralysis of the insane, I can only refer to an excellent paper by Dr. T. Clay Shaw¹ and to one by Dr. J. Batty Tuke.² I have had no opportunity of carrying out their suggestions and can only say that their treatment seems perfectly justified by the results obtained.

Puncture of the brain for the relief of chronic hydrocephalus dates back to 1744, and since that time both aspiration and, later, continual drainage have been frequently performed with encouraging results whenever done antiseptically and as early in each case as possible. Various sites have been chosen for the operation of draining of the lateral ventricles, but in order to reach them it is necessary to go through a certain thickness of the cortex, a procedure which, though not dangerous, is not altogether desirable. It has been more recently suggested that the cerebro-spinal fluid should be drained by removing a couple of lumbar laminæ and opening the subdural space; this would certainly be a favourable place for drainage, but at the cost of weakening the

¹ Brit. Med. Jour., Sept. 12th, 1891.

² Ibid., Jan. 16th 1892