toxic factor in the blood associated with an altered chemical condition of the vitreous may give rise to their formation. Vision appears to be little affected by these bodies *per se*.

Our knowledge is not sufficiently advanced to give a satisfactory explanation as to why some vitreous opacities are completely absorbed and why some remain easy of recognition by the ophthalmoscope and slit-lamp. So long as the toxic state of the blood or the weakened condition of the capillary walls continues the floaters are likely to persist, though the removal of the cause does not in every instance clear the vitreous.

Absorption can be hastened by medicinal and local treatment. Iodides internally and dionine externally appear to be the most efficient drugs, though many other drugs are used. Aspiration of the vitreous is helpful where the floaters are so heavy as to interfere seriously with vision, but I have no personal experience of this method of treatment. The detection and removal of the focal source of infection is, of course, indicated.

To sum up, we find that vitreous opacities must be considered in the light of the surrounding picture—the shape of the eye; the condition of the fundus, of the vessels, and of the lens; the age of the patient and his general physical condition, past and present. The occurrence of opacities in the vitreous is comparatively rare in children, but frequent in middle life and old age. Their presence is more frequent than we are led to suppose, and in the aged they are a usual occurrence when arteriosclerosis is present. In myopia of even moderate degree floaters are frequently observed in adults, and in young people showing progressive myopia their appearance must

not be disregarded. In early cataract the occurrence of vitreous opacities is common, and it is quite possible that the cause of these may be the same as that which leads to the lens changes.

Much the most usual cause of vitreous opacities is an altered condition of the blood due to general or local toxic causes; they may, indeed, be the only sign of focal infection in the eye. The glandular system, and even the ductless glands, may be a factor in their causation.

Apart from those of developmental origin, all vitreous opacities have some special significance, though their origin, as in the case of focal infection, may be difficult to discover.

The cases in which vitreous opacities interfere seriously with vision are few, and the subjective symptoms caused have little proportion to the number of vitreous opacities present.

The careful noting of the presence of vitreous opacities and their rate of mobility will have its reward in the future treatment of our patients, is often an aid to prognosis, and may aid us as to the expectation of fluid vitreous in cataract operations.

This very inadequate review of a subject which has hardly had its proper place in the literature of ophthalmology may lead to its study by others; it is a subject of which much has yet to be learnt, but which, with modern and more adequate methods of examination of both the vitreous and the general condition of the patient, is capable of much exploration.

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A CASE OF EMBOLISM AT THE BIFURCATION OF THE ABDOMINAL AORTA TREATED BY OPERATION.

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THE following case is reported because it presents several points of interest. The condition is a very rare one; our investigation of the literature shows that less than a dozen cases have been reported—mostly from Sweden—and all have ended fatally. Heart disease has been present in all. This case is, we believe, the first of its kind to be published in this country. Embolectomy has been successfully performed for emboli in other large vessels; Jefferson¹ reports some 28 successes; one of the chief factors in this respect seems to be the time of operation—any time up to twelve or fifteen hours gives a fair prospect of success. Our patient was not operated on until thirty-six hours after the onset. Circulation was, however, successfully re-established—this is the second reported case only in which the circulation has been re-established after so long an interval. As the patient died twenty-four hours after the operation it is impossible to be sure that the recovery in the circulation would have been permanent, but the post-mortem appearances were such as to suggest that at even so long an interval after the onset it is worth while attempting to free the vessel of clot. The multiplicity of emboli in this case, and in many others reported, has been the determining factor in producing an unsuccessful result. The clinical record is as follows.

A married woman, aged 62, was seen by one of us (J. le F. B.) on December 29th, 1927, and admitted to the Leeds General Infirmary the same morning. The history was as follows. The patient had not been well for a few weeks; she was easily tired and out of sorts. There was a history of rheumatism in her youth, but she had enjoyed fairly good health on the whole up to a few weeks prior to admission to hospital. The day before she was seen she

was seized with a most sudden and severe pain in the left iliac region, which was promptly followed by loss of power in both legs. The pain continued to be most severe to the right and left sides below the umbilicus, but settled down chiefly in the left iliac region a few hours before she was seen. She complained of complete loss of power and sensation in both legs. She was unable to pass her water.

When seen by one of us (J. le F. B.) on the morning of admission to hospital there was a complete flaccid paralysis of both lower extremities, with loss of reflexes and all sensation. There was no pulsation below the bifurcation of the abdominal aorta, and both legs were cold and pale. Short mitral presystolic and systolic murmurs were audible, and a diagnosis of thrombosis of the abdominal aorta was made on the spot. It was recognized that the patient was very gravely ill, and her immediate removal to hospital was urged. Thereafter she was under our joint care, and as no improvement was noted within twenty-four hours it was decided to make an attempt to remove the clot from the aorta. The operation was decided upon in the forenoon of the day after admission. The following are the details of the operation.

The abdomen was opened by an incision about 4½ inches long to the left of the middle line and the aorta exposed. There was no pulsation beyond about one inch proximal to the bifurcation. The aorta was controlled some 2½ inches above the site of the clot by the fingers of the assistant and by a light rubber clamp, and then opened for 1½ inches longitudinally. Much dark clot extruded and was extracted from the aorta and both common iliac vessels; the clots were some six inches in length. The aorta was stitched with interrupted through-and-through sutures of fine silk in one layer. On releasing the aorta pulsation returned at once in all the iliac vessels, and could be felt easily in both popliteals. There was no leakage from the suture line or the stitch holes. The abdomen was closed in the usual way without drainage.

The appearance of the abdominal wound was very striking; in the upper half there was the usual bleeding after making the incision; the lower half had a greyish-green colour and bled not at all. After the aorta had been freed of the clot, sutured, and released, the lower half of the abdominal wound began to bleed freely and assumed the normal ruddy colour.

It is interesting to note that, though the operation was not performed for some thirty-six hours after the sudden blocking of the aorta, when both limbs had completely lost all power of movement, and were cold and had assumed the purplish mottling seen post mortem, yet the circulation was apparently completely restored and power in the limbs was returning after the clot was removed.

At the subsequent post-mortem examination all the veins of the legs were patent and showed no evidence of clotting; in view of this and the other post-mortem findings, it seems reasonable to assume that death was due to clotting in the cerebral vessels, the

clot being derived from the heart, and that death was not due in any way to the derangement of the circulation in the lower extremities, or its sudden return. The embolus was approached by the abdomen rather than retrogradely by way of the femoral vessels because the physical condition of the patient was satisfactory for the abdominal route.

Subsequent Progress.—The patient stood the operation well, and pulsation was evident in both popliteal arteries and the posterior tibials the evening after the operation. She seemed very toxicæmic, and the pulse was quick, though it slowed down to about 80 per minute the same evening. She died rather suddenly at 9.20 a.m. the following day, from a simple failure of respiration and heart, without any further incident such as might point to pulmonary emboli or embolic lesions in other organs. A post-mortem examination was performed the following morning.

Externally was seen a recent abdominal incision which appeared to be a healthy abdominal operation incision. The thyroid contained three adenomata, and was moderately enlarged. The tracheal and bronchial glands were small and pigmented. The lungs were healthy. The heart was obviously dilated. There was an increase in subpericardial fat. The right auricle was much dilated and the tricuspid valve admitted three fingers easily. The cusps showed chronic thickening. The coronary arteries were normal. The mitral opening admitted only one finger, and the cusps showed evidence of old sclerotic endocarditis with a superimposed recent acute endocarditis. Multiple small vegetations, with a small blood clot still adherent, were seen. Both cusps were affected. The aortic valve was competent, and the cusps showed very slight patchy thickening; the right posterior cusp showed a tiny vegetation. The abdominal aorta, which had been exposed and incised at operation, was found to be closed efficiently; there was no leak anywhere. On opening the aorta and its iliac prolongations there was found to be a very small adherent clot, and below in the iliac, especially the right external iliac, there was an adherent clot. The internal iliac was clear on this side. The veins appeared to be clear—this includes the popliteal

femoral, and iliac. The right popliteal artery contained a clot, probably post-mortem. The aorta was notably small.

Brain.—Pia-arachnoid: there was a diffuse hæmorrhage over the right occipital pole and the left frontal lobe. On section this was seen to be due to obvious recent infarction. Attached to the dura and tucked under the anterior pole of the right temporal lobe was a circumscribed mass which resembled endothelioma, although it might have been of circulatory origin—? infarction.

The spleen contained a recent large infarction in its upper pole. The upper pole of the left kidney contained a large infarction and evidence of an older one. The liver and other organs were healthy.

Comments.

The diagnosis was comparatively easy, owing to the lack of peripheral pulses in the legs.

This case also shows how it is possible safely to open the abdominal aorta and restore its integrity; and if the patient had lived long enough opportunity might have offered to study whether the wound of the artery would have led to any subsequent aneurysm or blocking of the vessel by later clotting.

The cause of death, in the light of the post-mortem findings, was evidently a sudden embolism which travelled towards the brain. There was no convulsion, but the patient died quite suddenly after appearing to rally from the operation. The loss of sensation very closely corresponded anatomically in its upper limit to the first lumbar nerve root on the surface; there was actual tenderness on pressure over the bifurcation of the abdominal aorta, and the striking feature of the case was the intense pain complained of, chiefly below and to the left of the umbilicus and in the left iliac region.

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GLUCOSE: WHEN AND HOW TO USE IT.*

BY

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WHEN I was a child I read a book entitled *Madam How and Lady Why*. Its author, in ennobling "Why" and making "How" a commoner, symbolized a profound truth; it is one thing to know the workings of natural phenomena, but quite another to discover the reason which underlies those workings. My old professor of chemistry used to say: "Don't bother too much about *why* things happen. If you know how they happen, can make them happen, and can control them while they are happening, it does not much matter what theory you use to explain your results."

My subject to-day is the use of glucose—chiefly as an antidote to the shock of operation and anaesthesia—and the mechanism of its working according to McDonagh's theory of electrical and other changes in the body protein, which he expounds in his book *The Nature of Disease*.

McDonagh's theory seems to me not only to explain much that was obscure in pathology, but also to show many directions in which physicians, surgeons, and anaesthetists can advance in their use of the means they already possess. Its exact truth does not matter so long as it helps us to get better results, and this it undoubtedly does. Its substance is as follows:

If the blood serum of a healthy person be examined under dark ground illumination it shows a large number of little globular refractile particles in colloidal suspension, exhibiting Brownian movement. These are known as protein particles; they exist throughout all the tissues of the body, but those of the blood are the most convenient for examination. Each protein particle can be compared to a solar system; its sun is the protein nucleus, and its planets, in the order of their distance from the nucleus, are fats, amino-acids, urea and uric acid, sugar, inorganic salts, and electrons. The whole system is kept together by water, as is the solar system by gravity. It is always being attacked by hostile substances, whether chemical

products or micro-organisms, which tend to break it up. The most distant "planets," since they are the least closely attached, are the first to be split off. An invader, therefore, will strip off the electrons first; their electrical energy is dissipated in the form of heat and the body temperature rises. This appears clinically as fever. Then the salts are broken off; those which the plasma cannot hold are excreted in the urine. As the attack develops, the sugar, urea and uric acid, amino-acids, and, lastly, the fats, are reft from the protein nucleus and dissolved in the plasma, leaving the nucleus bare. In the end this itself fades away into true solution. Now as these planets are stripped away one by one, the water in which they swim is set free; the process is therefore called "dehydration." Some of the particles, however, undergo dispersion—that is to say, they subdivide to a certain point and there stop; there is a numerical increase and, in places where their volume exceeds that of the liquid present, the liquid and solid colloidal phases of the blood may be reversed. Instead of being a suspension of solid particles in a watery medium, it becomes a suspension of liquid globules in a solid medium; in other words, it "sets" like a jelly. This is called "gelation," and when it happens the patient suffers from intravascular clotting and thrombosis, which cause the clinical signs of cerebral sinusitis, hemiplegia, pulmonary embolism, and thrombosis, hyperemesis gravidarum, phlegmasia alba dolens, and a host of other conditions. Though gelation may occur primarily, it occurs more often secondarily, as a cyclical change of hydration. This explains why venous thrombosis is met with more frequently after operations and in the puerperium.

Now when the protein particles are being subjected to dehydration they do not follow one another like sheep. Only some of them are dehydrated—that is, split up into their constituent planets and driven into true solution, or dispersed; others resist the process, and they have only two means by which they can do this: one is to increase in size, the other is to agglutinate, or cling together. Since in both cases they take up water, they are said to become "hydrated," the opposite of dehydrated. When protein particles hydrate, their planets are at once drawn into a more intimate connexion with the nucleus and resist the more stoutly any attempt to split them off. Protein particles which have been hydrated tend to draw in upon themselves and work out their own salvation. Hydration is for the most part a defence mechanism

* Read in the Section of Anaesthetics at the Annual Meeting of the British Medical Association, Manchester, 1929.