The matter is urgent. Complaints are being made from all over the country, not only of the condition of streams which are recognized as passing through industrial areas, but of some of our beautiful fishing streams in rural country, where until recently practically no pollution existed. These new trade pollutions are of immensely greater magnitude than the old. Formerly there were numberless comparatively small factories with discharges varying between twenty and thirty thousand gallons a day of dirty water. Now, from beet sugar factories, artificial silk factories, and large dairies, the volume of foul water may amount to millions of gallons from one factory in a day. Thus one such large factory may entirely change the character of a formerly clear and unpolluted stream, and I am sure that many of you are aware of this having happened. If such pollutions are not promptly and properly dealt with the deterioration of our streams is certain.

This is a serious matter from many aspects, but especially serious in view of our increasing industries and population, and the difficulty of obtaining sufficient water supplies.

The question of fishing deserves consideration, for instances are known where, by the establishment of one of these modern factories, fish have been utterly destroyed and gathered up literally in cart-loads; and the value of fisheries is very great, although rapidly lessening because of pollution. The pleasure given to countless thousands by the fishing available in our clean streams and their enjoyment of the amenities of our country valleys are also to be considered. Perhaps none but those who have lived in our industrial valleys near a stream fouled by sewage and trade refuse can fully appreciate the delight afforded by a clear running stream in beautiful surroundings. To improve the condition of streams which are already polluted, and to prevent the pollution of these clean streams, is certainly well worth a great effort.

I have said that much good work could be done under the existing Acts of Parliament, but there is no doubt that in many respects these require amendment. This has been pointed out by various commissions, and will no doubt be dealt with by the advisory committee now sitting; but there is no reason to wait for an alteration in the law before putting into force the present powers. There is, however, one point where, possibly by agreement, an important change in the law would be of great value. The powers and rights of sanitary authorities and traders respectively, with regard to the admission of trade refuse into sewers, are by no means definite, and the practice in this matter varies from town to town. Some towns admit to their sewers, and deal with at their sewage works, all trade refuse without terms and without payment. Some towns absolutely refuse to admit any trade refuse into their sewers, and there are many modifications between these two extremes.

To my mind, in industrial areas, no satisfactory purification of the streams will be effected until trade refuse is dealt with along with the sewage of the towns. This, of course, ought only to be on terms of preliminary treatment of the trade refuse, or payment by the traders, or both. The matter has been dealt with by the Royal Commission of 1898 (see third report, paras. 22, 23, and 24, and the ninth report, para. 10). As the Royal Commission has pointed out, it is obviously inadvisable that the law should give the manufacturer "the absolute right" to discharge his refuse into the public sewer. This is evident on considering the case of one of the new large factories, already referred to, established in a small town or country village, where the amount of trade refuse may be a hundred times the volume of the domestic sewage of the place. In any change of the law provision would have to be made for dealing with such cases, but this is no time to discuss details.

I need only refer to one other desirable alteration in the law. Power should be given to some higher authority to decide between the authorities of adjacent districts when they propose, each of them, to set up their own sewage works. It is a scandal to see the countryside dotted with separate sewage works, where these could readily and more economically have been centralized. Without additional legal powers it is nearly impossible to overcome the jealousies between many of these local authorities, although in numerous cases, soon after they have constructed separate sewage works, they wish the policy of combining and centralizing had been followed.

In conclusion, the measures for dealing properly with river pollution are not likely to be thoroughly undertaken until special boards are set up for the purpose. Such boards could be set up, and do good work, under the existing laws. The laws need amending, especially that relating to the admission of trade refuse into public sewers. Every county council and council of a county borough should consider the condition of the rivers and streams within its district, and where pollution exists should represent to the Ministry of Health the desirability of constituting a rivers board.

# THE SIGNIFICANCE OF VITREOUS OPACITIES.*

BY

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To assess the true significance of vitreous opacities in every patient is an impossibility, but certain factors help us to come to a decision as to their importance or otherwise in most cases.

In the first place it is essential to recall the embryology of the vitreous. Those of you who were present at the 1927 meeting of the Ophthalmological Society of the United Kingdom will have heard Miss Ida Mann's admirable paper on the development of the vitreous; if not, you have probably read the paper. Miss Mann[1] there states that from the union of the mass of fibrils derived from lens and retina with those from the walls of the hyaloid artery the primary vitreous is formed in the foetus. She goes on to describe the formation of vessels in this vitreous and their subsequent atrophy, forming near the retina an avascular area named the secondary vitreous. After the third month of foetal life fibrils from the growing margin of the cup help to form the tertiary vitreous with a condensation of fibrils from the hyaloid membrane separating the secondary from the tertiary fibres.

As development continues condensation takes place, so that the adult vitreous consists of the primary portion filling the retrolental space and passing along the canal of Cloquet to the optic disc; the secondary vitreous extending from the boundaries of the primary to the internal limiting membrane, and the tertiary forming the suspensory ligament of the lens. The point is emphasized that the vitreous body is not enclosed by a structureless hyaloid membrane, being separated from the retina only by the internal limiting membrane.

The slit-lamp has added much to our knowledge of the structure of the adult vitreous. With a dilated pupil the retrolental space can be made out, and behind it the anterior portion of the vitreous, with its delicate waving fibrils and nodules. Even a portion of the canal of Cloquet can be distinguished, and separate moving bodies in certain cases, pigmented or otherwise. The vitreous is seen to differ in appearance with the individual, and may show a disorganization of what we consider the normal. The rapidity of the movements of the fibrils, their density, or their altered appearance to that of cotton-wool fibres are examples of what is meant. Bedell[2] states that pathological changes in the vitreous depend on changes in the framework and pigmentation, and that framework characteristics are marked by absorption of some fibres and the massing of others. He describes floaters as appearing like fine white lines, isolated fibres, single short threads, as a skein or tangle of cotton-wool, as cotton-like masses, white dots, heavy dust, and as snowflakes. Blood appears as roundish clumps, ovals, granules, and large masses.

With the contact glass much more of the vitreous can be seen; but of this I have no personal experience.

The investigation of the chemical and physical properties of the vitreous has recently been fully undertaken by some

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

of our own scientists, and to Mr. Duke-Elder we are indebted for his valuable work on the subject. He describes the vitreous as a colloid gel lying in the meshes of a scaffolding composed of a network of long interlacing fibres. This liquid is practically identical in every way chemically with the aqueous, though more viscid. Protein is contained in considerable amount; immune bodies, fat, cholesterin, urea, sugar, salt, calcium, and potassium are all present. It differs from the aqueous by containing, in addition, a muco-protein and a residual protein whose chemical nature has not yet been determined. In Duke-Elder's own words, "It seems that these two proteins are secreted by the surrounding ectoderm (mainly the retina) and that the common intraocular fluid (a dialysate of the capillary blood) percolates them and swells them up to form a gel."

Continuing his deductions, Duke-Elder[3] argues that any alteration in the metabolism suffices to make the gel fluid with the separation of its protein basis in the form of strands, films, or strings, and this change is due to some internal variation in the economy of the vitreous. What these variations are cannot yet be fully established—probably either a change of salt content or a failure in the efficiency of an autoxidation system. Probably the largest number of vitreous opacities we see in seemingly healthy eyes are from this cause. The haemorrhages and exudates in cases such as irido-cyclitis take place largely from the retinal vessels or the ciliary body, the endothelial cells in the media or intima of the capillary walls being affected by certain toxins circulating in the blood. The function of the cells being impaired a leakage takes place—the leakage taking the form of fibrinous exudates or haemorrhages.

With these facts before us—namely, the changes which take place in the development of the eye in foetal life and after birth, the condensation of the vitreous, and the disappearance of the embryonic elements, the structure and chemical constitution of the vitreous, and the function of the capillaries in the retina—we are in a position to suggest a classification of the most usual vitreous opacities as due to (1) changes in the condition of the capillary walls; (2) changes in the condition of the blood; (3) changes in the vitreous—congenital, traumatic, and mechanical.

1. Changes in the capillary walls, such as thickening, are a fruitful source of vitreous opacities. In arteriosclerosis it is almost the exception not to find floaters, which assume many forms. It may be taken for granted that in most people over middle age capillary changes to a greater or lesser degree are found, and a careful examination of the vitreous reveals in many cases the results of the exudates from the capillaries in the form of floaters.

2. Changes in the condition of the blood are responsible for the largest number of vitreous opacities. Whatever the toxin may be, the nutrition of the endothelial cells of the capillaries is interfered with, and leakage may occur. The grosser opacities found in focal infections giving rise to irido-cyclitis are due to this, but many focal infections may show no other effect in the eye than that of vitreous opacities. As an example of this, a patient examined a few weeks ago showed heavy floaters in the right eye as a result of a very severe irido-cyclitis some three years previously, but the left eye, which had never been seemingly affected, also showed numerous opacities to a lesser degree. To my mind this (and several other cases investigated) would prove that the toxin in the blood may not be sufficiently potent to produce external signs and yet be powerful enough to produce this leakage into the vitreous—a point I have never seen noted. In the more serious systemic diseases one expects and finds such leakage—for example, the numerous dust-like opacities often found in syphilis. In a mild chronic cyclitis which produces little more than an irritable eye I have found floaters in many cases, but the frequency with which they are found in the seemingly healthy eye gives much more room for speculation as to their cause. In the routine examination of a young woman of 25, floaters were found in both eyes, the fundi being normal. I found that she had been feeding her baby for six or seven months, and was feeling run down. A blood change through excessive lactation is a possibility in this case, and an examination of the recently delivered women in the maternity ward of my local hospital failed to show any evidence that any change took place during early lactation. Floaters have been observed in blood diseases showing glandular enlargement, and it is more than possible that the ductless glands also give rise to the appearance of vitreous opacities. A woman whose hair was falling off, whose skin was dry, and who showed other evidence of thyroid interference, gave a beautiful appearance of asteroid bodies in her vitreous. Two years afterwards I examined her again, when the thyroid symptoms had disappeared, as had also the asteroid bodies to a large extent. Another case illustrates the same point. A patient examined two years ago then showed normal fundi and media with low hypermetropic astigmatism. When seen again recently the hypermetropia had increased 1 D, and in the vitreous of each eye were numerous fine floaters, though the fundi were normal. Uncorrected distance vision had fallen from 6/6 to 6/12 in each eye, but the corrected vision was 6/5. The patient said he was run down, his digestion was uncertain, and that his doctor said his heart muscles were weak—all since an attack of influenza a few months previously. These symptoms all point to a toxic condition of the blood with the manifestation in the vitreous of exudates. In several cases of iritis of one eye vitreous opacities were clearly evident in the other, and many similar cases could be cited. The class of vitreous opacities where the protein base of the vitreous separates out owing to deficiency in oxidation or change of salt content, being affected by the changed capillary blood, also indirectly comes under this heading, and most cases of myopic opacities, but reference will be made to these later.

3. (*a*) Congenital remnants. In the condensation of the fibrils it is likely that some residue is separated out, and it is quite possible that some of the single, almost colourless strands we often find in the vitreous are from this cause. Miss Mann is of opinion that condensation of the fibrils continues during life, and in certain cases the effects of this are seen as floaters; the possibility of this change being to some degree pathological must be considered. Also the single dark speck not infrequently seen in children is possibly of congenital origin. Floating worm-like remains of the hyaloid artery are not uncommon, and the attached persistent hyaloid is often seen. These vascular remains are found more frequently in the extreme anterior or extreme posterior part of the vitreous.

(*b*) Trauma, when not sufficient to cause haemorrhage, may still upset the delicate mechanism of the vitreous gel, and a very small degree of trauma may cause separation of the protein base and liquefy the vitreous, producing floaters.

(*c*) Mechanical obstruction, as in venous thrombosis, produces haemorrhages from the venules (not the capillaries) with the unabsorbed residue left as vitreous opacities. It is an interesting fact that retinal haemorrhages occur at birth in from 15 to 20 per cent. of all confinements. These haemorrhages are purely mechanical, and fortunately clear up in the large majority of cases, but we find occasionally in children unexplainable vitreous opacities which may be due to this source.

As some slight guide to the frequency with which vitreous opacities occur I made a careful examination of them in a series of patients in my private practice. I took 100 consecutive patients and included all except those whose fundi could not be examined, either through extensive lens changes or corneal disease. In this series vitreous opacities occurred to a greater or lesser extent in 37 per cent. of the cases. I then took a series of the next 100, and in this the percentage was, curiously enough, exactly similar. Notes were made as to the sex, age, type of refraction, lens changes, and fundus abnormalities, and though from a statistical point of view the result may be of little value, many points of interest came to light. These two series of cases contained a comparatively small number of children, as it was not the holiday season, but the number of cases showing opacities is rather more than one would expect, and it is, if anything, underestimated. In many cases the opacities were of the light, almost colourless, cotton-wool variety, in some cases one or two small specks, in others dust-like opacities, but in the majority of older people, at any rate, a mixed variety.

In these 200 cases there were 33 examples of myopia above 1D, and in 23 of them floaters were found. Only 6 had fundus disease apart from arterio-sclerosis, while in the last-named condition floaters were almost universally present.

In the first place, as to the method of examination—the ophthalmoscope was chiefly employed, the slit-lamp being used only in a few special cases. The luminous ophthalmoscope used in these examinations for vitreous opacities contained two lenses which could be moved coaxially to the path of light and between them an achromatic lens which could be moved up and down along the axis of the instrument by means of a collar. The two lenses first mentioned could be moved out of position, thus allowing the light to fall incident on the achromatic lens. This produced a slightly divergent beam, effecting the result of a plane retinoscopy mirror, and this was found to be the most favourable position for detecting the opacities. Contrary to what was formerly taught, it was found that the greatest possible light that could be reflected on the vitreous made the opacities more obvious. A red-free filter was also available, but only in certain cases was it of any value. Quick up and down movements of the eyeball were made by the patient, and with light focused on the reflection of the optic disc and by adding *plus* lenses of from +5 to +20 little difficulty was experienced, even with an undilated pupil, in detecting the floaters if the method was carried out thoroughly. In the majority of cases where the opacities were very faint they were detected in the posterior part of the vitreous. Whether the position in the vitreous of the opacities has any special significance I have failed to determine, though it is stated that when using the slit-lamp the early appearance of the cells and deposits in the retrolental space is a feature of considerable significance in the detection of early corneal inflammation.

Should it be wished to measure the depth of the opacities with the ophthalmoscope—and I should judge that it is only necessary when we find a large single opacity more or less fixed in position—it can be done by determining the difference in dioptres between the lens by which the opacities are clearly seen and the lens by which the fundus is visible; 1 mm. corresponds to a different refraction of 3D, and so if an opacity is best seen by +8D and fundus with −4D, 12 ÷ 3 = 4 mm. in front of the retina.

Few of the patients examined in whom floaters were found complained of "black spots" until asked directly about them. In several whose chief complaint was the inconvenience caused by spots no vitreous opacities were detected. In some of these cases, where none were found, the slit-lamp showed lens changes almost unrecognizable by the ophthalmoscope. Floaters may be present in large numbers, and may give rise to practically no subjective symptoms, and yet a solitary floater may cause much annoyance. After an illness, or during the stress of heavy office work, or when glasses need to be changed, the floaters appear to cause the patient much trouble. The liver theory that is popular among our patients may have some substratum of truth.

Incidentally, among myopes the subjective symptoms are the most marked, as the uncorrected vision is indistinct and the opacities better projected on the hazy background. The reason why myopic eyes should be more subject to floaters is not fully established, just as the reason for the elongation of the eye is theoretical. Possibly v. Graefe's theory that the elongation is due to an inflammatory process in the choroid and sclera is the most sound, the sclera becoming softer and more yielding. This mild inflammation would give rise to exudates and haemorrhages and hence to opacities. Possibly the changes are so slight as not to be detected with the ophthalmoscope, and so opacities are found in seemingly healthy myopic eyes. In other cases of myopia elongation may take place from congenital deficiency of the ciliary muscle retarding the outflow from the eye and so increasing intraocular tension. This may give rise to internal changes in the vitreous and cause separation of the protein base and ultimately opacities.

In the periodic examination of young myopes, where we find the myopia increasing in a rapid manner we are often in difficulty as to whether to advise withdrawal from school for a period as a means of checking the myopia. Should we find a normal myopic fundus but the appearance of floaters never before noted, or an increase where they have previously been present, it is a decided point in influencing us to advise rest for the eyes.

As previously stated, in cases of marked arterio-sclerosis floaters are generally present as a result of changes in the capillary walls, and their absence is a more favourable sign for prognosis; also, opacities are frequent in eyes with early lens changes, and possibly the cause of the two conditions is similar whatever it may turn out to be. A record of the motility of the opacities before the advancing lens changes obscure the view may be helpful with a view to subsequent operation, or the rate of movement of the floaters may give us some idea of the fluidity of the vitreous.

The size, shape, number, and rate of motility of vitreous opacities vary enormously with the eye and the conditions causing them, but in very few cases do they interfere seriously with vision. I have seen a fixed opacity causing a central scotoma owing to its position, but fortunately this is rare.

I have purposely avoided much mention of large haemorrhages and exudates, new blood vessels in the vitreous, vitreous detachments, congenital abnormalities such as persistent hyaloid artery, and suchlike, as their significance is obvious. In retinal detachments the presence of vitreous opacities would tend to point to a serous origin of the condition rather than to a neoplasm.

One type of vitreous opacities, however, must be described in some detail as their detection is at any rate of academic interest, and probably more than this. I refer to the asteroid bodies first described by Benson—my first teacher in ophthalmology—in 1893. These bodies, when seen by the ophthalmoscope, vary in appearance from white star-like globules, lazily and gently floating in their firmament, changing their position with the movement of the eye, but returning to somewhat the same area, to the snowball variety, more massive and fewer in number, or the more quickly moving and less luminous type. They are easily detected, even with the non-luminous ophthalmoscope, by the indirect method, and it has been noticed that when focused against the whitish background of the disc they often lose their luminosity.

The occurrence of these bodies has been recorded in the literature very infrequently from the time of Benson's case till the last few years, though it is more than probable that they were confounded with cases of synchysis scintillans, from which they differ both in appearance and in origin. The glittering shower of coloured crystals of cholesterin falling rapidly to the lower part of the vitreous is entirely different from the appearances above described. On seeing my first case some six or seven years ago I discovered a description of the condition,[4] and the statement that only about twenty such cases had been recorded. Since then I have seen nine or ten cases, and, curiously enough, have not seen one case of synchysis scintillans in my own practice since that time. One must conclude that the asteroid type is much more common than is generally supposed, and much more frequently met with than the cholesterin type.

Various theories have been put forward as to the origin of the asteroid bodies. Among such theories are: that they are an exudate secondary to tuberculous cyclitis; that they depend on arterio-sclerosis in association with an altered blood condition; that they are formed through deficiency of oxygen in the vitreous; that they are due to disease of the ductless glands through an altered condition of the blood; together with others less likely. The constitution of the bodies has been investigated, and it has been proved that they consist chiefly of calcium soap, calcium carbonate, and a certain amount of fatty or lipoid substance in combination of various grades of stability.

Among my own cases were some old people with marked evidence of arterio-sclerosis; a woman of middle age, in whom the asteroids had cleared up almost completely when seen again after three years, as had the symptoms of thyroidism previously present; a woman aged 35, whose doctor gave her a clean bill of health; and a man of 60, who said that for thirty years he had had chronic indigestion. These cases cannot all have a similar origin, but a

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.

References.

[1] Mann: *Transactions of the Ophthalmological Society of the United Kingdom*, vol. xlvii, p. 172. [2] Bedell: Ibid., vol. xlv, p. 650. [3] Duke-Elder: Various publications. [4] Holloway: *Amer. Journ. Ophthalmol.*, vol. v, p. 100.

## 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; Jefferson[1] 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 1¼ 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