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COMMUNICATIONS

TWO CASES OF VISIBLE EMBOLI IN RETINAL ARTERIES
With a Histological Study in one case

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1.—Case Reports (G.C.P. and L.H.S.)

In view of their rarity it seemed worth while recording the ophthalmoscopic appearances of two cases in which actual emboli could be seen in retinal arteries. Subsidiary matters of interest were the reactions of neighbouring vessels to the emboli and to vasodilator drugs.

Case 1.—At 8.30 a.m., on May 26, 1941, when going on duty, Sister K. H., of Horton Hospital, suddenly noticed a complete “black-out” of her left eye. She rubbed her eye with her hand, and in about two minutes the lower half of the field of vision began to clear. After about 20 minutes this half of the field was approximately normal; but the upper half was still absent. Next day about 6.50 p.m. her eyes were examined. At that time the R. vision was 6/5 and L. vision 6/9. The left pupil reacted sluggishly to light. The retina in the lower half was white and opaque. The inferior temporal branch of the central artery was contracted from the disc to just above the second fork of the vessel. (Fig. 1). The upper branch of the first fork running beneath the macula was also in a state of contraction. At the bifurcation of the second fork (Figs 1 and 2) a white mass could be seen in the lumen and apparently including the wall of the vessel, so that the width of the mass appeared to be a little wider than the calibre of the vessel. The artery just above the fork was dilated, as were also the two branches of the fork for about 1/4 disc breadth below—after that the branches
were contracted. It was concluded that the white mass was an embolus lodged in the vessel. The field was contracted generally, especially in the upper nasal quadrant, where the boundary approached the fixation point. (Fig. 3).

Although rather a long interval had elapsed a capsule of amyl nitrite was administered. A subconjunctival injection of 40 mg. acetylcholine was made followed by 20 mg. of mechloly (acetyl-beta-methyl-choline) subcutaneously. There was marked general upset within two minutes of the mechloly injection. The pulse raced, marked vaso-dilation occurred, and the systolic blood-pressure dropped from 140 mm. Hg to 120 mm., rising again to 150 mm. in 20 minutes. Diastolic pressure was unchanged.
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Total loss
Recovered

LEFT.

K.H. 5/330 mm. white

Fig. 3.

Fig. 4.
The patient was so upset that it was impossible to scrutinise the figure very closely, though the impression was gained that the spastic vessels had dilated. Next morning this could be seen to be the case. (Fig. 4). The artery had dilated from the disc to the second fork, and so had the inframacular tributary. The embolus was seen to have shifted a short distance down the upper tributary of the second fork. The lower tributary of the fork was now dilated, while the upper branch below the embolus was still in spasm. (Fig. 4). The patient thought that the visual field had improved, and in fact the 5/330 isopter for white had receded a few degrees further from the fixation point. (Fig. 3).

Rather reluctantly the patient agreed to have yet another injection of mecholyl. It made her feel extremely ill; but caused no appreciable change of vision. An area of contraction still remained in the vessel below the embolus (Fig. 3); but this and the whitening of the fundus background gradually disappeared during the next few weeks. Viewed on June 12, 1941, the white embolus could no longer...
be seen and the vessel looked normal with the ordinary light of the electric ophthalmoscope. Using red-free light a break could be seen in the vessel at the site of the embolus (Fig. 6). With the Bjerrum screen a relative scotoma could be demonstrated (Fig. 7) corresponding to the inframacular branch artery and this shaded off into an absolute scotoma above. (See Bedell, 1937).

Six months later the disc was atrophic in the infralateral quadrant; and white lines showed along the walls of the inferior temporal branch of the central artery.

No conclusive evidence was obtained as to the source of the embolus. Sister had been running a very heavy ward, and had been constantly overtired. She had had mild attacks of precordial pain. She was examined by Dr. Terence East, who reported a very faint and inconstant mitral systolic murmur. He would not commit himself to any definite opinion that the heart was the source of the embolism.

CASE 2.—On May 6, 1942, a man aged 30 was admitted to the hospital from his home complaining of dyspnoea on slight exertion; attacks of sweating and blindness in the right eye. He was aware that he had heart disease for many years, but no diagnosis had been made until he was admitted to hospital during December, 1941, where a diagnosis of congenital heart disease was made. This led to discharge from the Army in February, 1942.

Since a complete dental extraction he had apparently felt vaguely unwell, and for a few weeks prior to admission had attacks of sweating with an occasional rigor. On the day before admission he collapsed while at work, the loss of vision of the right eye having occurred suddenly five days before.

On admission he had a temperature of 101° and a pulse rate of 100. He was orthopnoeic and moderately cyanosed. The heart was considerably enlarged, mainly to the left, and there was a loud mid-sternal systolic murmur with a thrill. The blood pressure was 120/80. A tentative diagnosis of sub-aortic stenosis was made, but the embolism of the retinal artery, swinging pyrexia and R.B.C.'s in the urine indicated that subacute bacterial endo-carditis had supervened. The presence of a partially extruded sequestrum from the jaw suggested that the dental manoeuvres had precipitated this infection. A blood culture showed the presence of non-haemolytic streptococci.

During his illness he had a severe epistaxis and later on a sudden attack of thoracic pain which suggested a pulmonary infarction. The cardiac murmurs changed somewhat and an aortic diastolic one developed about one week prior to his death which took place on May 22.

Post-mortem showed recent fibrous pericarditis. There was gross cardiac enlargement affecting primarily the left ventricle. The aortic cusps were surmounted by very large greenish friable vegetations. There was an abnormal 4th cusp present. There was an aneurysm of one sinus of Valsalva, the endocardium of which was largely involved by the infection. The aorta was hypoplastic and 'a thickened fibrous band represented the ductus arteriosus. There was a typical septic spleen, some nutmeg change in the liver, and an early embolic nephritis. There was a bilateral effusion in the chest. The lungs showed hypostatic congestion with considerable oedema of the upper and middle zones.

Dr. W. Bridgen summarised the case as consistent with subacute bacterial endocarditis superimposed on an unusual congenital cardiac abnormality.

From the ophthalmic point of view the main interest lay in sudden loss of central vision in the right eye on May 1, 1942. Ophthalmoscopic examination showed a white mass lodged in the temporal branch of the first lower fork of the central artery just inside the disc margin. (Fig. 8). There was an area of retinal pallor in the region supplied by the branch. The circulation was not completely obstructed as cattle-truck movement of the blood-column could be seen below the macula. (Fig. 8). The arteries generally, distal to the embolus, were extremely constricted.

A subconjunctival injection of 40 mg. acetylcholine was made, followed by 20 mg. of mecholyl. The blood-pressure, which had been 118/78 mms. Hg, fell to 90/68 mms. Hg. After a further 20 mgs. of mecholyl it read 78/60 mms. Hg. The patient felt very hot and vomited, so he was given atropine gr. 1/100. Symptoms at once disappeared and the blood pressure rose to 110/80 mms. Hg.

Ophthalmoscopically the artery below the embolus was seen to dilate for a short part of its length immediately below the block following the acetylcholine.
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After the mecholyl the peripheral end of the vessel began to dilate, and the proximal dilatation below the block extended a little peripherally. (Fig. 9). Next day the vessel was still more dilated, the proximal and distal dilatations, being separated by a length of vessel in spasm about 1 disc diameter in length. (Fig. 10).

By May 14, 1942, the vessel was uniformly dilated (Fig. 11); the patient said his vision was improved, though his general condition precluded accurate objective testing. He had symptoms of a lung embolism on May 11, 1942. After his death on May 22, 1942, his right eye was removed for histological examination.

Discussion

Numerous points for consideration arose from these cases.

(a) Was the white patch in the vessel in Case 1 an embolus? It was difficult to see what else it could be, and its appearance was identical with the patch in Case 2—which was definitely an embolus. Werner (1913) reports a case where "just below the disc the inferior temporal artery was blocked by an oval glistening-white object, below which the vessel was thread-like, while above this part it was normal in size." The white object was 1 ½ times the normal diameter of the vessel, the apparent enlargement probably being due to opacity in the vessel wall surrounding the embolus. Paton (1933) described a similar opacity with an apparent diameter of 2 mm.

(b) In case 1 we thought the sequence of events to be as follows:—

In the first instance the embolus blocked the main trunk of the central artery causing the complete black-out initially noted. Sister rubbed her eye with her hand, causing the embolus to move on
to the second fork of the inferior temporal branch. The lower visual field cleared while the onward passage of the embolus set up spasm of the branch and its tributaries, presumably by the mechanical stimulation. We think the thread-like vessels must have been in spasm, rather than masked by oedema, or it is difficult to see why they appeared of wider calibre after the administration of vasodilator drugs. The spasm of the inframacular branch in this case must have been intermittent or incomplete, or the whole of the scotoma would have been absolute instead of relative near the fixation point.

There is evidence that spasm of the retinal vessels is a comparatively common phenomenon, (Davenport (1931), Griffith (1931), Traquair (1933), Minton (1936)). Here one would suppose the passage of the embolus excited the spasm mechanically, causing the vessel to grip the embolus during its passage, in the same way that a renal calculus may set up spasm and be gripped during its passage down the ureter. Similarly an application of the Larsen diathermy electrode may set up waves of spasm in a retinal vein. Coats (1905) mentions the interesting possibility of "incomplete embolism." The plug stops at a part of the artery which it does not completely fill and the obstruction is made complete by spasm of the arterial wall.

(c) Was the white fundus background oedema? The rest of the retina was of tigroid type in Case 1. We cannot help thinking mere oedema less likely than the ischaemic necrosis of the ganglion cell and nerve fibre layers suggested by Coats (1913). Otherwise one would have expected to see more of the tigroid background than was shown after the vessels dilated. The same reasoning applies to Case 2. Oedema is obviously present in such cases as that described by Paton (1933). He saw Mr. H. E. Juler apply massage to an eye, when "the whole condition cleared up, just as if one had taken a sponge and had wiped the white oedema off the retina."

(d) Were any of the therapeutic measures taken of the slightest value to the patients in these two cases? We think the patient in Case 1 herself shifted the embolus by massage in the first instance. Mules (1888) reported a very similar case of an embolus shifted by massage of the globe. The amyl nitrite, acetylcholine, and mecholyl undoubtedly dilated contracted vessels, but the time-lag was too great for much benefit, except possibly in the case of the inframacular branch mentioned. The fact that the embolus was shifted along the lumen of the vessel made no difference—an absolute scotoma still corresponded with that particular area of retina. In Case 2 the patient claimed subjective improvement in vision after the vasodilator drugs, but no accurate estimate was
FIG. 5.
The section on the slide, shown in Figs. 11, 12 and 13, passes through the peripheral part of the disc, but not actually through the optic nerve itself. This small peripheral section of the disc shows some subacute swelling, with considerable patchy small-round-celled infiltration, some slight proliferation of fibro-blasts around the small and congested vessels supplying the disc itself, a slight degree of oedema, and—what is the matter of greatest importance—a section of the blocked artery itself, this being one of the larger branches of the central artery of the retina and this photograph is a very-low-power one, to show the general "geography" of the section. Below and to the left are nutrient vessels passing to the sclerotic and to a less extent through it to the choroid. These vessels, both small arteries and veins, as well as others in the neighbourhood not shown in the photograph, are normal in structural appearance and their walls do not show chronic thickening or any appreciable degree of inflammatory change, either acute, subacute or chronic. (The darkly stained structures are transverse and oblique sections of nerves, not vessels). (×30).
Fig. 12.

An ordinary low-power view of the peripheral portion of the disc, showing the blocked vessel, with its distinctly thickened walls and a small empty and contracted branch of the same vessel (its continuity can be traced in a neighbouring section) to the left, and a small empty vein immediately below these. Note also the moderate but distinct small-round-celled infiltration in the more central part of the disc and subjacent area of the cribriform part of the sclera, etc. (×100).
FIG. 13.

A high-power view of the blocked vessel and its immediate neighbourhood, the latter showing small-round-celled infiltration and an occasional polymorph below and to the right. The block in the lumen of the artery suggests an *embolus*, say a bit of impacted vegetation from heart-valve, rather than a portion of a thrombus formed *in situ*. Its looks "firm" and has definite edges. One can at a glance exclude the possibility of its being merely an agony-thrombus and still more so a post-mortem clot, as there is proliferation of the endothelial cells of the inner coat of the vessel, with actual buds of the proliferated cells "eating" into and absorbing little "bays" in its periphery (see also Figs. 14 and 15, to which much of this description also applies, and note the channel being "canalised" through it in Fig. 15). The proliferation at the right can be better studied in Fig. 14. (X400).
This is from the same section as Fig. 13, but focused farther down into the thickness of the celloidin section, and so as to bring out the little buds of proliferating endothelial cells of the intima digesting their way into the "embolus," the edge of which looks firm and more "discrete" than in most of the ordinary thrombi formed in situ. Note the large bud of endothelial cells, the "shadows" of which can just be seen at the upper left-hand edge of the "embolus." \((\times400)\).
A neighbouring section through the body blocking the vessel. This shows the "firm" appearance of the mass with its well-defined edges, and the buds of proliferated endothelial cells of the intima digesting their way into its periphery. There are some non-haemolysed red corpuscles amongst the endothelial cells to the left, suggesting some degree of patency of the lumen, or perhaps some communication with the "canal" shown in Fig. 15. Note the contracted or semi-contracted left, which one would have expected to be thrombosed and blocked if the parent vessel had been merely "thrombosed." Note the small empty vein below. (x 400).
A further section through the embolus on its "far surface" (in relation to the appearances seen in Figs. 13 and 14). The celloidin sections are fairly thick, and, on focusing down into the deeper level, there is a suggestion of proliferated cells on its far surface, and from this far surface the "canalisation" well seen in this section has occurred. In this "canal" are closely-packed non-haemolysed erythrocytes and some leucocytes, as well as the canalising endothelial cells which show only as shadows round its periphery. To the right there is considerable proliferation of the intimal cells, suggesting that the process has been going on for a week or two. Note the "bud" attacking the periphery of the embolus immediately above the "canal." A portion (or tributary) of the small empty vein is seen below the artery. (X400).
FIG. 16.

A composite photograph of an area in which the pigmentary layer has remained attached to the choroid (which itself shows well-marked congestion and inflammatory cell-infiltration), and there is therefore no mechanically intermingled pigment-cell remains mixed with the debris on the deeper aspect of the retina. But, in this area of retina, there is definite and well-marked patchy subacute to slightly chronic granulation tissue thickening on the inner aspect of the retina, as well as irregularity of the ganglion-cell and thickening of the nerve fibre layer and internal limiting membrane. (X100)
Section passing longitudinally through optic nerve and disc central to the position of the blocked artery seen in the previous sections illustrated in Figs. 11 to 15. There is a certain amount of chronic irritation in the region of the lamina cribrosa—but this is not a specially pronounced feature at this particular part of it: nor is there any great degree of this in the pial sheath of the nerve, nor of the arachnoidal sheath (though there is some little irregularity of its outlines), or of the dural sheath which forms the margin of the photograph on each side. The row of sections of small vessels on the right side are comparatively normal in appearance, only the third from below being filled with closely-packed red corpuscles, amongst which there is an excess of leucocytes. The white space in the centre of the nerve just touches the extreme periphery of the coats of a vessel, presumably the central artery, but in adjacent section no part of its lumen is available for examination. (×30).
FIG. 18.

High-power view of a longitudinal section of the optic nerve near the nerve-head to show the subacute to chronic small-round-celled infiltration in the sheaths of the nerve-bundles. In various presumably normal optic nerves the amounts of fibrous tissue and of cells in the various parts of the sheath vary considerably in individual specimens, but the above photograph illustrates a distinct and undoubted excess of cells, though not of the fibrous tissues of the sheath itself.  (×400)
A high-power view of a section of the choroid just to the left of the nerve-head (see Fig 11). It shows a small artery with thickened walls and almost empty lumen. There is intense congestion and dilatation of the venules, one of these extending across the entire field, and another being seen below this to the right. Between the latter and the artery there is some subacute inflammatory cell-infiltration, also seen above the long venule, together with some extravasation of red corpuscles. The erythrocytes in the congested venules are discrete, with no obvious evidence of any thrombosis, and amongst the red corpuscles there were numerous collections of leucocytes, mostly polymorphs—not distinguishable in the photograph. There is, therefore, in this section, evidence of fairly chronic, subacute, and recent acute irritative phenomena in the eye. (×400).
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possible. Orr and Young (1935) secured a therapeutic success with acetyl-choline. Emboli have been shifted with benefit in the cases of Mules (1888), Coverdale (1929), Butler (1927), Orr and Young (1935) and others so that in cases of retinal embolism vaso-dilator therapy seems worth trying in spite of its discomforts and even dangers (see Cushny, 1941).

2.—Pathological Considerations (W.E.C.D. and A.S.)

Review of Literature

Few of the forty-odd cases of occlusion of the central artery of the retina examined histologically, are free from grave objections as to their validity for the elucidation of the nature of the occlusion or of the consequent changes in the retina. The classical case of von Graefe, who in 1859 introduced the concept of embolism of the central artery of the retina, was held by Leber—on histological evidence—to substantiate the view that occlusion of the central artery was embolic in origin but the broadened conceptions on vascular pathology that came towards the end of the last century led to the postulates that occlusion might also be due to changes in the arterial wall and to thrombosis; and Harms in 1905 could reasonably hold that the embolic nature of occlusion had not been proved anatomically in a single case.

Most of the eyes that have come to histological study are old in a double sense; the condition had been long-standing and the patient had been past middle age. In such cases, the changes observed in the artery are difficult to interpret. Is the organised obstructive mass in the vessel the primary lesion, with secondary changes in the vessel-wall; or are the changes in the vessel-wall primary, with secondary proliferative changes in the lumen? Furthermore old-standing retinal changes do not throw any clear light on the character of the immediate lesion in the retina in cases of occluded central artery.

The older literature has been fully reviewed by Coats (1906 and 1914), Harms (1905) and Leber (1915). The eclectic views of Harms who denied embolism altogether—or at any rate regarded it as unproven—represent an extreme but by no means untenable position. The studies of Coats and of Leber whilst upholding the significance of embolism, allowed for endarteritis and thrombosis as significant factors. No incontrovertible conclusions could be reached on the older material and it is only of late years that material less open to objection and examined histologically soon after the onset of the clinical symptoms has become available. This restricted material is also not always equivocal as can be seen from the following analysis:
W. E. Carnegie Dickson, and Others


The histological findings in this case have been criticised by subsequent observers, especially Coats (1914). The obstruction is reported by Velhagen at the origin of the opthalmic artery from the internal carotid artery. The site is unusual; an embolus at this site would probably be followed by the establishment of a collateral circulation; and the absence of any organisation in the clot is against expectation in a case of three months' standing. The presence of arteriosclerosis clinically—there was no general post-mortem examination—is a disturbing feature, even though it is reported that the retinal arteries showed "hardly any disease."

2. Früchte (1908). Man, aged 55 years. Established heart disease with a history of rheumatism. Loss of vision in the right eye with a characteristic picture of occlusion of central artery (cattle-truck appearance in the early stages). Infective endocarditis, with streptococci in the blood, followed by gangrene of right foot, infarcts in nose, spleen, kidney and liver as complications. Patient died 22 days after onset of eye trouble. Post-mortem examination showed old and recent endocarditis, aortic and mitral stenosis and insufficiency, and infarcts in various organs. Moderate arteriosclerosis was present.

Histologically, the central artery of the retina showed an almost complete obstruction immediately behind the lamina cribrosa. The arterial wall was normal, the endothelium being absent only where the embolus adhered to the wall. The obstruction was a homogeneous mass darkest at the centre and was surrounded by a layer of elongated cells. Staining for fibrin was negative. The ganglion-cell layer of the retina showed vacuoles and there was disorganisation of the rods and cones at the macula. The picture was complicated by congestion in the choroid with inflammatory cell reaction.

3. Rubert (1911). This is a case of great significance in the study of the early changes in the retina following occlusion, but has no validity in establishing the existence of embolism, for there is no evidence of any heart lesion and much evidence in favour of thrombosis as seen in marasmus.

A man, aged 41 years, underwent gastrostomy for stenosis of the oesophagus. Ten days later he developed blindness in the left eye with a classical picture of occluded central artery. Death from exhaustion ("weakness of heart, dyspnoea, etc.") four days later.

The changes in the central artery were somewhat similar to those described by Früchte. The retina showed marked oedema of the nerve-fibre layer with vacuolation and chromatolysis of the ganglion cells.

4. Meinhause (1920). Man, aged 37 years, suffering from tabes and infective endocarditis. Three days before death he developed sudden blindness of the left eye with a typical picture of occlusion of the central artery, the obstruction being situated at the nerve head at the origin of the macular branches. Post-mortem there was found a patent foramen ovale, parietal thrombi in the right heart, and infarcts in the lungs and kidney.

Histologically the obstructing mass in the central artery was hyaline and fibrillated in structure, the fibres representing fibrin. The mass took on a brownish tint with v. Gieson's stain; nuclei and fragments of cells were present between the fibres.

The main retinal changes were oedema of the outer part of the internuclear layer, with large net-like vacuoles. The inner part of the internuclear layer was much less affected. The ganglion-cell layer was swollen particularly towards the macula, and its nuclei stained unevenly, some nuclei being darker than others; the cytoplasm itself was unchanged, but there was some vacuolation between the cells. The fovea itself was normal. The choroidal vessels were well-filled and contained an abundance of leucocytes. The walls of the retinal and choroidal vessels were normal.

5. Mohr and Böhn (1921). This is a case of bilateral occlusion of the central artery. A man aged 40 in 1918 had a war injury (grenade splinter) to his forehead
18 months previously, blinding him in both eyes for eight days. He returned to full service but was in indifferent health and in March, 1919, was diagnosed as suffering from heart and kidney disease. The left eye became suddenly blind six months later, followed by loss of vision of the right eye 14 days subsequently. There were the typical appearances of occluded central artery of the retina. Aortic insufficiency from endocarditis of the aortic valves was found. The patient became acutely ill with joint pains. A blood culture was negative and the Wassermann reaction doubtful. Within five weeks of the onset of blindness in the first eye the patient's condition warranted the diagnosis of embolus in the brain without focal symptoms. There was increasing cardiac failure; five weeks later the patient died suddenly after having developed erysipelas three days earlier. Post-mortem, aortic and mitral disease with vegetations were found, and there were infarcts in the spleen and localised thickenings of the walls of some of the cerebral vessels.

Histologically, both eyes showed obstructive masses behind the lamina cribrosa, interpreted as emboli. The retinal vessels here showed no endarteritis except in one peripheral branch, but it could not be asserted that they were completely normal. Especially massive changes in the retina were observed in the inner layers (nerve fibre, ganglion and inner nuclear layers). The maculae were affected and there was migration of pigment epithelium into the cone layer. The choroid showed hyaline degeneration of some vessels, with increase of leucocytes and the presence of streptococi.

6. Karbe (1924). A boy aged 12 years died of acute pulmonary tuberculosis, purulent bronchitis and broncho-pneumonia. About 36 hours before death the child had complained of loss of vision of the right eye, diagnosed as embolism of the central artery some 12 hours later.

Histologically the central artery in a horizontal section of the nerve showed the picture of a softening or pus-forming disintegration of a loosened thrombus in the stage of incipient shrinkage. There were no organisms. The artery itself was normal. There was mild oedema at the papilla in the ganglion cell layer. The nerve fibre layer was rather wavy but the nuclei of the ganglion cells stained well. There was no cellular infiltration in the oedematous area, and the rest of the retina was normal. The author believes the embolus to be derived from parietal-thrombus formations in the heart (which, however, was found normal and devoid of parietal thrombi at the post-mortem examination).

7. Engelbrecht (1924). A man aged 57, the subject of chronic suppurative ethmoiditis and sphenoiditis, developed occlusion of the central artery of the left eye. Four days later he underwent a radical operation on the sinuses under pantopon-scopolamine anaesthesia, but died three hours later without recovering consciousness. Post-mortem revealed nothing abnormal apart from the local sinus condition; the carotid arteries in particular were examined for thrombus-formation and the heart for any abnormality.

Histologically, the central artery was found normal in structure; there was no increase in elastic fibres. An obstruction incompletely filling the lumen was present in the region of the lamina cribrosa. It was an unorganised; partly a hyaline, partly a granular mass clearly separated from the intima of the arterial wall and was regarded as an embolus.

A sequel to this report is the note contained in an article by Scheerer (1925), who gives a personal communication from Engelbrecht changing the histological diagnosis of embolus to one of post-mortem clotting.

The cases of Schwitzer (1906), Shoemaker (1908), and Coats (1914), which are recorded as examples of embolus have no conclusive value, the first two because of the insufficiency of the data supplied, and Coats' case because of the complication by glaucoma and the advanced age of the patient (71 years).

That the picture of occlusion of the central artery can clearly be produced by lesions other than emboli is well illustrated by the case of Jaensch (1928) (and possibly also, that of Siegrist (1900)) in which it was due to extension of a thrombus from the internal carotid artery to the ophthalmic artery; and by the case of Velhagen (1915) in which the central artery was compressed by a haemorrhage into the optic nerve. It is of interest to note that both Jaensch's and Velhagen's patients had endocarditis, to which the lesion might uncritically have been ascribed.
The complexity of the problem is brought into still greater relief by the work of Scheerer (1925), who has shown that unorganised clots in both the central artery and vein at the lamina cribrosa are a common post-mortem finding. As already stated, Engelbrecht’s final diagnosis of his histological findings in his case was that they were post-mortem artifacts. Scheerer, who re-examined the sections of Mohr and Böhms case and those of Früchte’s case, holds that these too show no organisation and represent post-mortem appearances. He is indeed critical of all the other recent cases since the eyes were removed post-mortem, and stresses that in Früchte’s case clots of the same unorganised type as those seen in the central artery were present in the choroidal vessels.

One more factor in the causation of occlusion of the central artery (and vein) stressed by Scheerer (1923) deserves consideration. His studies of sclerosing processes in the lamina cribrosa have shown that this structure has pathological changes of its own, with secondary effects on the vessels; moreover the lamina may well have nutritive functions.

Of the seven cases in the newer literature, that of Engelbrecht can be dismissed as a clear case of post-mortem clotting. On the evidence of Scheerer, the cases of Früchte and of Mohr and Böhms must likewise be excluded. Rubert’s case is distinctly less convincing than Früchte’s and this applies still more to Karbe’s case. Velhagen’s case has been criticised by earlier writers so that the one case that can be regarded as valid is Meinhausen’s. Unfortunately his description of the changes in the artery is scanty and does not contain any evidence indicating that the block of the artery showed signs of organisation.

The newer literature therefore does not add any undisputed cases of embolus to the older literature which brought forward much presumptive but no finally conclusive evidence.

3.—Histological Examination of the Eye in Case 2

The specimen (No. 42/22, Royal Eye Hospital Pathological Laboratory) consisted of the posterior segment of the right eye removed through the roof of the orbit 22 hours after death. It was hardened in formalin and fixed in celloidin. Serial sections were made and stained with haematoxylin and eosin. The conclusions of one of us (W.E.C.D.) were reached on histological evidence only.

The findings are illustrated in the photo-micrographs 11-19, and described in the legends. They may be summarised as follows:—

1. The choroid, retina, and optic nerve show considerable small round celled infiltration of the subacute to chronic type. This is
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consistent with the septic process throughout the body induced by the subacute bacterial endocarditis. (Figs: 11, 12, 16-19).

2. The block in the artery (Figs. 11-15) is an embolus of some duration (probably several weeks) lodged in a vessel with a slight to moderate degree of previous thickening of its tunica media and adventitia, but not previously of its intimal coat. There are localised proliferative thickenings of the intima in the immediate neighbourhood of the embolus, which is just beginning to be canalized by endothelial buds which are invading its periphery. (Figs. 13-15).

3. The changes in the retina, proliferative and irritative, are not extensive and are most marked in the inner layers, though the rods and cones are also affected. These changes are largely subacute and probably date from the same time as the impaction of the embolus. There are, however, more recent acute changes, especially in the choroid, which shows an acute congestion of its venules which are distended with densely packed red blood corpuscles and a large number of polymorphs, indicating the presence of considerable leucocytosis just before death. (Figs. 16-19).

The histological evidence by itself is conclusive enough but taken together with the clinical and pathological evidence as a whole,—the heart lesion with vegetations, multiple embolic processes, the absence of any arteriosclerotic changes as also of thrombus formation—there can be no valid doubt as to the nature of the block.

Summary

1. Two cases of visible emboli in branches of the central artery of the retina with superadded spasm are recorded.

2. One of the patients died from subacute infective endocarditis 21 days after the onset of the ocular symptoms which themselves occurred six days before the full development of the symptoms of his fatal illness.

3. The eye examined histologically showed an embolus in the process of canalization.

4. On clinical grounds the occurrence of embolism as distinct from spasm, thrombosis and endarteritis of the retinal arteries can hardly be doubted. Clear histological evidence of this is, however, not available. The present study supplies this evidence.

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