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COMMUNICATIONS

THE OCULAR MANIFESTATIONS OF SPONTANEOUS SUBARACHNOID HAEMORRHAGE*

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SUBARACHNOID haemorrhage may occur spontaneously or as a result of head injury. The traumatic and non-traumatic cases present many points of similarity; but important differences result from the physical factors attending their onset.

The present communication is concerned with the non-traumatic group, and, especially, with the pathological findings in five cases which came to post-mortem examination. It is believed that these throw some light on the nature and mode of origin of the ocular complications.

Since the haemorrhage, in the majority of cases, comes from the vessels of the circle of Willis, being due to the rupture either of an aneurysm or of a weakened portion of the vessel wall, cases occur in which the effusion of blood is relatively slight, but prone to recurrence. The leading symptoms here, are severe deep-seated pain often accompanied by paresis of one or other of the ocular muscles. Cerebral arteriography may reveal the presence

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of an aneurysm, and lumbar puncture discover blood staining of the cerebro-spinal fluid.

In the severe cases, such as those dealt with in this paper, the clinical picture is entirely different. The patient is seen in a more or less comatose state, with a history of sudden onset, and with signs suggestive of cerebral compression and meningeal irritation: pupil anomalies, nystagmus, conjugate deviation of the eyes, and paralysis of cranial nerves, including the oculomotors, are often observed. These are not in themselves sufficient to justify a diagnosis of subarachnoid haemorrhage. The presence of retinal haemorrhages and papilloedema, if these are found on ophthalmoscopic examination, strongly supports such a diagnosis, and a history of minor attacks adds to its probability, but the matter is settled by the discovery of blood in the cerebro-spinal fluid.

Such cases usually terminate fatally, and it is not surprising that in the majority no record is obtained of the state of the vision.

At post-mortem examination blood is found in large amount in the subarachnoid spaces at the base of the brain, sometimes breaking through into the substance of the brain or into the lateral ventricles. The source of the blood can usually be found in a ruptured aneurysm or a ruptured branch of the circle of Willis.

Immediately behind the optic canal, the optic nerves may show a bluish coloration owing to the presence of blood between the sheaths, and blood in this situation may be seen on the cut surface of the nerve. Even in cases where no blood is found in this region, exposure of the orbital part of the optic nerve often shows this to be expanded and discoloured by the presence of blood between its sheaths. In some cases (one of the five under discussion) this appearance affects the whole nerve from the optic foramen to the eyeball; but in the majority the ecchymosis is patchy and intermittent. It is usual to find some degree of bulbous distension of the optic nerve sheaths immediately behind the globe. Distension of the orbital veins, and haemorrhages amongst the orbital contents and on the surface of the sclera, may also be observed.

There has been an increasing interest in this subject in recent years, especially since the introduction of lumbar puncture.

Symonds (1923, 1924), in reporting his personal cases, reviewed the bibliography, which then contained records of some 124 cases. His earliest reference is to Wilks (1859), but Wm. Mackenzie (1830) gives a clear description of the clinical and post-mortem appearance in a case described as one of " Amaurosis from dilatation of the cerebral arteries."

" The sheath of the right optic nerve at its entrance to the eye was thickened and distended with blood, the veins on the

back of the sclerotic were much enlarged, and the ramifications of the retinal veins were readily seen all over the retina as far forward as the lens."

Throughout the literature attention has been concentrated on the question of the source of the blood which is found within the sheaths of the optic nerve and in the retina. It was natural to assume that the blood effused under pressure in the basal subarachnoid space passed through the optic canal by continuity into the subarachnoid space of the orbital part of the optic nerve, and thence into the retina or even into the vitreous (Tureen, 1941; Lyngar 1939).

That the blood in the sheaths of the optic nerve is of intracranial origin is still what may be called the "text-book" view; (Duke-Elder, 1940), but the investigations of Liebrecht (1906-1912) concerned chiefly with subarachnoid haemorrhage in injuries of the head, showed that the matter was by no means so simple. Several authors (Key and Retzius, 1870, Bartelli, 1938) cast doubts on the freedom of communication between the subarachnoid spaces of the brain and those of the optic nerve; but more recently Wolff (1940) has stated that, while in the upper part of the optic foramen the optic nerve, its sheaths and the periosteum are firmly bound together, there is, on the lower side, a clear channel between the intracranial and the orbital subarachnoid spaces.

Schiek (Discussion on Bücklers, 1935) held that if papilloedema and distension of the optic nerve sheaths, in cases of cerebral tumour, was due to the passage of cerebro-spinal fluid, the blood stained C.S.F. in subarachnoid haemorrhage could pass by the same route to the optic nerve sheaths and to the retina. This view, however, has no histological support. Liebrecht pointed out that sometimes blood was found in the sheaths of the optic nerve even when none was present in the subarachnoid space of the brain, that it was exceptional to find blood within the optic canal, and that at the apex of the orbit the perineural haemorrhage was not in the subarachnoid space of the nerve sheath, but in the dural sheath (for the most part surrounding the vessels in the sheath), passed forward by the lymph channels surrounding the vessels into the subdural space, and might remain entirely subdural or penetrate into the subarachnoid space.

He suggested three possible sources for the blood in the perineural subarachnoid space: (1) That in a few exceptional cases it might pass along the sheath through the optic canal. (2) That it might pass by the lymphatic spaces around the ophthalmic artery into the dural sheath and thence into the subarachnoid space. (3) That, in the majority of cases, blood from the torn vessels of the dural sheath passed into the dural tissue and thence

by pre-existent lymph channels into the subdural and subarachnoid spaces. As Liebrecht was dealing with traumatic cases, his third suggestion is not applicable to the cases of spontaneous haemorrhage.

Grimminger, at a later date (1924), made the suggestion that blood from the middle fossa may pass along the sheath of the first division of the 5th nerve, through the superior orbital fissure into the orbit, forming a haematoma of the ciliary nerves. It will be seen below that in one of my cases effusions of blood were found around the ciliary nerves and ciliary ganglion, but serial sections lend no support to the idea that this blood had tracked along the course of the 5th nerve.

Liebrecht, having found that, as a rule, there was no continuity between the blood in the middle fossa and that in the sheaths of the optic nerve, inclined to trace the latter to rupture of the vessels of the dural sheath within the optic canal. He did, however, point out (1906) that in histological examination of three cases the veins of the optic nerve, choroid, retina and other structures were found to be extremely full of blood, and that this was due to the sudden haemorrhage within the cranial cavity, and the consequent stasis of the blood in the ophthalmic vein and its branches. In several cases haemorrhage was found in direct relation to veins in the tissues peripheral to the lamina cribrosa, and Liebrecht expressed the view that the haemorrhages in optic nerve and retina occurred at the moment of the injury and were not secondary to that in the sheath.

To naked eye examination, only one of my own cases showed the optic nerve to be surrounded by blood in the canalicular segment. In two other cases serial sections in this region showed some blood corpuscles in the dural sheath; but this could not be taken as evidence of the passage of blood from the subarachnoid space of the brain to that of the optic nerve.

My own observations therefore support the view of Liebrecht, that while it may be a physical possibility for blood to pass through the optic canal in this way, it does so only in exceptional cases.

The occurrence of haemorrhage in the retina or in the vitreous has led to much speculation regarding the cause of this haemorrhage, and the source of the blood. There is no reason to believe that isolated haemorrhages in the retina could be derived from blood in the subarachnoid sheath of the optic nerve; but cases occur in which haemorrhage, sometimes massive, is found in front of the disc or crossing its borders. Such a haemorrhage is particularly liable to become subhyaloid, or to break through into the vitreous, and its clinical characters lend some colour to the view that the blood has tracked forward by the side of the optic nerve,

or along with the central vessels from the sheath spaces of the nerve. Uhthoff did not think that this was possible, and in this was supported by Dupuy Dutemps (1914), Riddoch and Goulden (1925) and others, but Leslie Paton (1924), as the result of personal observations, expressed his belief that such a leakage of blood could occur in a small percentage of cases. In this connection he reports two cases in which there was a large subhyaloid haemorrhage overlapping the optic disc and, in one case, subsequently breaking into the vitreous. Only one of these two cases came to necropsy. There was severe haemorrhage from a branch of the right middle cerebral artery extending into the right lateral ventricle, the basal subarachnoid space and the sheaths of both optic nerves. Paton's paper does not state that any histological examination was made to establish the connection, if any, between the perineural haemorrhage and that in the retina, and so far as I am aware, there has been no microscopic proof of the passage of blood from the optic nerve sheaths to the retina in the way assumed by Paton for his cases. The more generally accepted view is that distension of the subarachnoid space of the nerve by the effused blood causes obstruction of the blood flow in the central retinal vein where it crosses the subarachnoid space, in a very similar way to the condition seen in thrombosis of the central vein. Paton sees the difficulty of reconciling this explanation with the very different appearance of the retinal haemorrhages in the two conditions. But the circumstances under which the retinal haemorrhage occurs in thrombosis of the central vein and in subarachnoid haemorrhage are very different. In the former, although the final block may be somewhat sudden, there has been a long period of progressive change in the affected vein. There has been increasing narrowing of its lumen with a gradually increasing impediment to the venous circulation and consequent changes in the permeability of the walls of the retinal branches. In the case of subarachnoid haemorrhage, on the other hand, whether traumatic or spontaneous, there may be no antecedent cause for such a general weakening of the walls of the retinal veins, and the stasis is sudden and severe.

As I have already mentioned, Liebrecht found, in histological examination of three cases, that the veins of the choroid, retina, optic nerve and other structures were extremely full of blood; and attributed this to the sudden rise of intracranial pressure with consequent stasis of the blood in the ophthalmic vein and its branches.

The sudden rise of intracranial pressure here referred to, and the influence of the consequent venous stasis on the occurrence and distribution of the haemorrhages throughout the orbital and ocular structures was also alluded to by Bücklers (1935), but

seems to have received no further comment, although it would seem to be the most important single factor in producing the clinical picture of subarachnoid haemorrhage from the ophthalmic point of view. It is not necessary to deny that stasis and haemorrhage may be caused by sudden pressure or tension on the central vein where it crosses from the optic nerve to its sheaths; but if this does occur it can only explain the haemorrhages peripheral to that point.

It should be noted, moreover, that distension of the sheaths by blood is not invariably present; indeed it only occurred in one of my five cases; in the other four there was not even evidence that the central vein had been compressed by haemorrhage. In these four cases the distension of the sheaths was caused by fluid in the subarachnoid space, and the fact that the ampulliform swelling of the nerve ceased some 3 or 4 mm. in front of the crossing of the vessel does not suggest the occurrence of tension on the central vein. Again, the assumption of pressure or tension on the central vein as the cause of the retinal haemorrhage takes no account of the fact, which emerges from the cases here reported, that haemorrhages occur simultaneously at many points outside of the field of distribution of the central retinal vein; and one can only reach a satisfactory explanation of all the ocular phenomena in this condition by assuming a sudden venous obstruction affecting a much wider area.

Illustrative Cases

This communication deals with five fatal cases of subarachnoid haemorrhage which were submitted to post-mortem examination, and in which sections were obtained of the eyes and optic nerves. The cases differ to some extent in detail, but illustrate all the characteristic features of this condition. Four of the five were women, and one a man, the ages ranging from 39 to 62. All had elevation of blood pressure, severe in three and moderate in two. In only one were the fundi free from haemorrhage. In four the source of the subarachnoid haemorrhage was discovered; in one it came from a rupture of the middle cerebral artery, and in three from rupture of an aneurysm of the vessels of the Circle of Willis.

CASE 1.—A woman aged 50 years. Malignant hypertension, B.P. 230/150. W.R. positive. Gave a history of having had double vision, but no motor defect on examination. C.S.F. blood-stained. O.E.R. General pallor and opacity of fundus due to diffuse exudate. Clusters of white specks at the macula. Arteries very narrow, with marked variation of calibre. Thrombosis of a secondary venous branch with formation of network of collaterals. Flame-shaped haemorrhages. White woolly patches.

O.E.L. Condition somewhat similar, but no venous thrombosis.

Death. Post-mortem examination: Extensive subarachnoid and intracerebral haemorrhage probably from right middle cerebral artery. Bulbous expansion of both optic nerves behind the globe. Bluish discolouration due to haemorrhage within the sheath.

Sections of the retina showed the characteristic changes of hypertensive retinopathy: ganglioform degeneration in the nerve fibre layer, masses of eosin-stained exudate in the internuclear layer and behind the layer of rods and cones, haemorrhages in all the retinal layers, and between the neuro-epithelium and pigmented layer.

The larger arteries in the retina showed little or no change. The walls of the capillaries and smaller arterioles were thickened and the lumen round and open. Thickening of the vein walls was also seen.

Serial sections of the right optic nerve (and sections of the left optic nerve presented no essential difference), from behind the optic foramen to the anterior extremity of the nerve, demonstrated the distribution of haemorrhage within the sheaths. Blood was found throughout the whole length of the nerve, but varied in situation and in quantity from point to point. Thus: in the canalicular segment there were two minor effusions of blood (one of them very small), within the dural sheath, and a few red blood corpuscles in the pia-arachnoid (Fig. 1). Further forward in the same segment the blood in the dura was very scanty and that in the pia-arachnoid relatively increased. In this part of the nerve there was no subdural haemorrhage linking that in the dura with that in the pia-arachnoid. Passing forward the haemorrhage increased, till, coming to the apex of the orbit, we found several massive haemorrhages in the dural sheath, and relatively slight haemorrhage in and under the arachnoid (Fig. 2). At some points these dural haemorrhages seemed to be closely related to the vessels.

Behind the point where the central vessels cross from the dural sheath to the nerve, there was an increased amount of subarachnoid haemorrhage and only a few corpuscles outside of the arachnoid (Fig. 3). Throughout the next segment of the nerve, which included the central retinal vessels in their passage across the vaginal spaces, there was no massive haemorrhage, but scattered clusters of red cells both subdural and subarachnoid in position. Where the central vein lies in the dura, just before crossing the space (Fig. 4) there was some blood lying close to it at one side, but nothing to suggest compression of the vein; and the artery and vein were not accompanied by blood at any other part of their course. Further forward the blood became more scanty; still found

Fig. 1

Fig. 2

Fig. 3

Fig. 4

Fig. 5

Fig. 6

CASE 1.—FIGS. 1 TO 6.

Transverse sections of R. optic nerve. 1. The canicular segment. Light haemorrhage in dural sheath and pia-arachnoid. 2. At apex of the orbit. Massive haemorrhages in dura, light in pia-arachnoid. 3. Behind entrance of the central retinal vessels. Haemorrhage mainly subarachnoid. 4. Where central vessels lie in dural sheath. Light haemorrhage, dural, subdural and subarachnoid. 5. Near anterior end of vaginal space (section somewhat oblique). Scanty haemorrhage in dural and pial sheaths; and in space. 6. At level of lamina cribrosa. Scanty haemorrhage in space and in border tissue.

in the subdural and subarachnoid spaces. Where the vessels reached the centre of the nerve there was again an increase in the quantity of blood, this time entirely subdural. Near the anterior end of the intervaginal space there was again a slight increase in the haemorrhage in the dura, subdural and subarachnoid, and in the pial sheath (Fig. 5). At the level of the lamina cribrosa there were merely a few clusters of erythrocytes in the dense tissue which separates the nerve from the anterior extremity of the intervaginal space (Fig. 6).

Antero-posterior sections through the optic nerve entrance and the neighbouring retina showed haemorrhage under the neuro-epithelium apparently tracking forward, via the "border tissue"

CASE 1.—FIG. 7.

Vein on outside of dural sheath. Dissecting haemorrhage between endothelium and media and between media and adventitia.

into the retina, in a situation corresponding with the marginal haemorrhage seen ophthalmoscopically.

The segment of the nerve behind the optic canal included the internal carotid artery and the ophthalmic artery. The carotid at this point was grossly atheromatous, with calcification and fatty changes in the atheromatous patches, and breaking down and calcification of the internal elastic lamina. The ophthalmic artery at its point of origin from the carotid also showed some thickening of the intima and calcification of the elastic lamina; but throughout the rest of the sections the ophthalmic artery, the central vessels and the vessels of the sheaths were practically normal.

Comment

The changes in the retina have probably no bearing on the subject of subarachnoid haemorrhage, being those found characteristically in hypertensive retinopathy: but the sheath haemorrhages are definitely part of the picture of subarachnoid haemorrhage.

The points of special interest are: (1) the wide distribution of the haemorrhage throughout the whole course of the optic nerve, intracranial, canalicular and orbital, and (2) the fact that the haemorrhage is patchy in its occurrence, and varies in its intensity and in its situation. There is no continuity between the effusions of blood in the dural sheath and those in the subdural and subarachnoid spaces, and we are obviously dealing with a number of discrete haemorrhages, not with blood tracking along the sheaths from the middle fossa to the anterior extremity of the nerve.

It is of interest too to note that there is no evidence of compression of the central retinal vein where it crosses the space between the optic nerve and its sheaths. In this particular case haemorrhage between the sheaths is nowhere abundant, and at the point in question it might be called scanty, and it is practically unrelated to the central vessels.

CASE 2.—A woman aged 56 years. Moderate hypertension. W.R. positive. O.E.: Optic atrophy. Death twelve hours after onset of severe headache with loss of consciousness. C.S.F. blood-stained. Clinical diagnosis subarachnoid haemorrhage, confirmed at post-mortem examination. Effusion of blood basal and intracerebral, probably originating from the anterior part of the circle of Willis. Bluish discoloration with distension of the optic nerve sheaths immediately behind the eyeballs, and patchy discoloration of the nerves throughout their orbital course. A small ecchymotic patch in both nerves just behind the optic canal. A haemorrhage of pin-head size on the anterior aspect of the chiasma.

Anteroposterior sections of the retina, choroid, sclera and optic nerve of the right eye showed no haemorrhages in these structures, but small discrete haemorrhages in and beneath the dural sheath, under the arachnoid and in the pial sheath. The subarachnoid haemorrhage was scanty, and the notable distension of the sheath must have been caused by fluid. It was observed also that some of the vessels of the retina were surrounded by a clear perivascular space, more pronounced in the veins than in the arteries. Since the veins as a whole were well filled with blood and the arteries empty, the perivascular space could not be looked upon as an artefact. It apparently indicated a stasis of fluid analogous to that within the nerve sheaths. The optic nerve itself was atrophic, with thickening of the connective tissue septa.

Similar sections in the left eye also showed a pronounced distension of the subarachnoid space apparently due to fluid.

Only a few small clusters of red blood corpuscles were found in the anterior part of the space. Anterior to the point of emergence of the central vessels there was some subdural haemorrhage, with scanty subarachnoid haemorrhage here and in the anterior extremity of this space. In the neighbourhood of the vessels themselves, crossing the space, haemorrhage was very scanty, and still more so behind this point. No haemorrhages were found in the retina or choroid or in the optic nerve itself.

The central retinal artery and vein or their branches in the retina, presented little abnormality other than the enlargement of the perivascular lymph space referred to, but among the vessels

CASE 2.—FIG. 8.

Transverse section of optic nerves in front of chiasma. Large haemorrhage in front of the junction of the nerves, diffuse haemorrhage in the soft membranes.

on the outer aspect of the dural sheath there were arteries with thickened hyaline-looking walls and veins with lymphocytic cuffing.

The presence of a small haemorrhage on the anterior aspect of the chiasma had attracted notice at the *post-mortem* examination (Fig. 8), and the chiasma with stumps of the optic nerves and optic tracts was fixed and cut serially in paraffin. In addition to the compact haemorrhage between the optic nerves in front of the chiasma, the sections showed a good deal of diffuse haemorrhage in the meningeal covering of the chiasma and nerves. The walls of the vessels of the arachnoid were feebly stained with haemalum, possibly owing to a *post-mortem* change; but, buried in the principal haemorrhage, in this situation, there was an incomplete ring of endothelial cells representing the wall of such a vessel, and probably the site of the rupture which was the source of this haemorrhage. Among the vessels of the pial and arachnoid

sheaths several were found to have red blood corpuscles between the layers of their walls, and in the perivascular space.

Within the chiasma and the attached portions of the optic nerves, the smallest vessels presented thick hyaline-looking walls and a small lumen (about 14 to 40 μ) and appeared as rigid as pipe stems. At more than one point vessels of the pia and of the arachnoid showed a breach in their wall through which blood had apparently escaped; but more interesting was the discovery, in the substance of the chiasma and optic tracts, of many vessels showing haemorrhage within and outside of their walls (Figs. 9-14). In some cases this amounted merely to one or two red blood corpuscles between the layers of the wall, in others many corpuscles could be seen at varying depths throughout the wall, while

FIG. 9.

FIG. 10.

FIG. 11.

FIG. 12.

FIG. 13.

CASE 2.—FIGS. 9 to 14.

Dissecting haemorrhages in the walls of blood vessels in the optic nerves, chiasma and optic tracts. In 14 the ruptured vessel is visible.

in others the vessel was completely surrounded by blood which either filled a perivascular lymph space or had broken through into the surrounding nerve tissue. These lesions might be described as intramural haematomata or dissecting aneurysms. A series of interesting transition forms were found up to the stage at which the vessel was only recognisable as a more or less complete tube of endothelium buried in a massive haemorrhage. Here again, in one or two instances, an apparent point of rupture of the vessel wall could be discovered (Fig. 14).

Comment

The first point of interest in this case is the fact, already recorded in the first case, that the extravasated blood within the subdural and subarachnoid spaces, was nowhere very abundant. The largest haemorrhage was found in the subdural space at one side of the series of sections anterior to the point where the central vessels cross the vaginal spaces, and there was nothing to support the idea that compression of the vessels could be produced by the haemorrhage. The haemorrhage, such as it was, was mostly in the subdural space: while the notable expansion of the nerve behind the eyeball was mainly due to fluid distension of the subarachnoid space. Further, there was no evidence of tracking of blood from the subarachnoid space behind the optic canal, along the nerve to

its anterior extremity. The haemorrhages in different situations were discrete, and there was at least some evidence of rupture of vessels at a number of points within the dura and elsewhere. Finally, the unexpected finding of isolated haemorrhages from vessels of the membranes covering the chiasma, and still more unequivocal haemorrhages from vessels in the chiasma and optic tracts, shows that the familiar hypothesis of compression of the central vessels, offered to explain the occurrence of the retinal haemorrhages, is quite inadequate when we come to consider the wider pathological picture.

This was the only one of our cases in which investigation extended beyond the optic nerve, the eyeball and the orbital structures. The significance of the haemorrhages in the chiasma and optic tracts is therefore uncertain, but if, on further enquiry, it can be shown that such haemorrhages occur, for example, within the brain stem, they would provide a reasonable explanation of the oculomotor and other palsies which are such a frequent feature of the condition (Case 4). Such a sign as conjugate deviation of the head and eyes (see Case 5) cannot be satisfactorily explained by direct action of the intracranial haemorrhage on nerve trunks.

CASE 3.—A woman, aged 62 years. Unconsciousness of sudden onset $2\frac{1}{2}$ hours before admission to hospital. Twitching of hands, arms and face. Vomiting. B.P. 226/138. Blood in C.S.F. O.E. R.E. large recent haemorrhage in lower temporal quadrant of fundus, and other smaller ones elsewhere. L.E. A few small haemorrhages.

Death occurred within 24 hours. *Post-mortem* examination showed gross subarachnoid haemorrhage over the base of the brain, as well as haemorrhage in the lateral ventricles, and in the left frontal lobe. The source of the blood was the rupture of an aneurysm at the junction of the left anterior cerebral and anterior communicating arteries.

On exposing the contents of the orbit the orbital veins were found to be markedly distended. There were haemorrhages among the orbital fat, in Tenon's capsule, on the posterior surface of the eyeball, on the outer surface of the dural sheath, and within the sheaths of both optic nerves from the optic canal to the sclera. Naked eye examination of the retina confirmed the ophthalmoscopic appearances. Papilloedema was discovered both on slit-lamp examination and in sections.

Microscopic examination showed the severe and widespread character of the bleeding in this case. On the right side there was abundant effusion of blood filling the vaginal spaces along the course of the optic nerve, from its point of contact with the internal carotid artery to the back of the eyeball, at all depths: dural, subdural and subarachnoid; and in the anterior part so

Fig. 15

Fig. 16

Fig. 17

Fig. 18

Fig. 19

CASE 3.—FIGS. 15 TO 19.

Transverse section of right optic nerve. 15. Near anterior end of nerve. Blood almost completely filling the space. 16. Near apex of orbit. Massive haemorrhage in dural sheath breaking through into subdural and subarachnoid spaces. 17. Slightly anterior to 16. Haemorrhage among the orbital tissues. 18. Haemorrhage surrounding ophthalmic artery, in the dural sheath, and in the sheath spaces, embracing the central retinal vessels. 19. Behind the entrance of the central vessels. Haemorrhage in the pia-arachnoid, and overfilling of vessels of the pial septa.

massive that the arachnoid membrane could not be traced. There was also massive haemorrhage among the fibres of the dural sheath, and on its outer surface. In the posterior third of the nerve there was massive haemorrhage, breaking through into the subdural and subarachnoid spaces. But, in addition to these effusions in the usual situations, blood was found within the optic canal, outside the dural sheath at the apex of the orbit (Fig. 17), and, further forward, in the pial sheath (Fig. 18). There was

CASE 3.—FIG. 20.

Antero-posterior section of right optic nerve. Massive haemorrhage fills the spaces and distends the sheaths. There are haemorrhages at all depths in the retina, before and behind the retina, in the sclera and round the vessels of the Circle of Zinn.

haemorrhage surrounding part of the ophthalmic artery, in the dural sheath where penetrated by the central artery and vein, and in the pia surrounding the central vessels (Fig. 18).

About 15 mm. behind the entrance of the central vessels there was at one side of the nerve a notable distension of the vessels of the pia; and of the septa of the nerve, and diffuse haemorrhage in the adjacent part of the pia and arachnoid (Fig. 19). In the anteroposterior sections at the optic nerve entrance (Fig. 20) haemorrhage was found surrounding the vessels of the circle of Zinn, in the sclera, among the nerve fibre bundles in front of the lamina cribrosa, in all the layers of the retina, in front of the disc, in the vitreous, and in front of, and beneath the retina.

In the intracranial part of the nerve there was patchy haemorrhage dural, subdural, subarachnoid, and in the pial sheath.

CASE 3—FIG. 21.

Antero-posterior section of left optic nerve. Massive haemorrhage fills the spaces, and there is diffuse haemorrhage in the epibulbar tissues near the optic nerve entrance.

The internal carotid artery showed atheroma with calcification, and in the adjacent portion of the optic nerve there was hyperaemia in the septal vessels and a good deal of haemorrhage in the substance of the nerve itself.

In the left eye no retinal haemorrhages were seen in the sections passing through the papilla; but the sheath haemorrhage was almost as abundant as in the right eye. At the anterior end of the nerve the sheath spaces were completely filled with blood as far as the cul-de-sac, and haemorrhages were found among the fatty and connective tissues in the angle between the dural sheath and the sclera. It was interesting to observe that the central retinal vein, in its course through the nerve, was not well filled with blood, and in this situation there was no undue filling of the vessels of the septa of the nerve.

In both eyes there was unequal filling of the choroidal vessels; in some parts this seemed to be excessive. Throughout both the retinal and the ciliary vessel systems the arteries showed medial sclerosis and some thickening of the intima, and some of the veins had thickened walls. The arteries of the choroid were markedly sclerosed, and the veins here also had thickened walls.

Comment

This case, like the two preceding cases, shows in the first place that we are not dealing with the simple question of intracranial subarachnoid haemorrhage passing along the subarachnoid

space of the optic nerve sheaths, through the optic canal and along the orbital portion of the nerve. We found a striking venous congestion in the orbital venous system and haemorrhages in the orbit, in all the sheaths of the nerves, and in the optic nerves themselves. In the more markedly affected right eye there was haemorrhage in all the layers of the retina, as well as behind and in front of the retina, and in the vitreous.

It is noteworthy that of the five cases here reported, this was the only one in which we could say that the sheaths of the optic nerves were *distended* with blood, and even here we had no evidence that compression of the central retinal vein was the cause of the retinal haemorrhage.

CASE 4.—A woman aged 39 years. Essential hypertension of at least 8 years duration. No nephritis. Father died 2 weeks before her admission. Injury to face and side five days before admission. Headache, vomiting, confusion and drowsiness for 24 hours before admission. B.P. 238/116. Albuminuria. R. hemiparesis. Paresis of L. 3rd and 4th cranial nerves. Blood in C.S.F. W.R. negative. O.E. Many white woolly patches and haemorrhages. The latter of varying size but mostly large, dark and of rounded form. *Post-mortem* examination: Subdural haemorrhage in small amount in both anterior fossae. Marked subarachnoid haemorrhage in neighbourhood of circle of Willis, some of it of apparently old standing. Ruptured aneurysm at junction of basilar and posterior cerebral arteries. Haemorrhage into the superficial layers of the pons, especially to the left of the middle line.

Marked distension, and blue discoloration of both optic nerves behind the globe. Ecchymosis of the nerve sheaths at one or two discrete points between the globe and the optic canal, but no naked eye evidence of blood within the sheaths behind the optic canal.

Sections of the right retina showed gross haemorrhages in the nerve fibre, and outer molecular layers. For the most part these seemed to be quite independent of each other, the inner nuclear and inner molecular layers being unaffected; but at several points the haemorrhage had broken into the intervening layers. In the same region the outer molecular layer, over a wide area, was occupied by an amorphous (albuminous?) coagulum.

Serial sections of the right optic nerve showed, at the level of the lamina cribrosa, some scanty haemorrhage in the border tissue and in the inner layers of the dura at the anterior extremity of the subdural space. Absence of other haemorrhage was conspicuous in the first two or three millimetres of the nerve.

Between 3 and 6 mm. behind the globe the maximum distension of the sheath was situated. This was for the most part due to effusion of fluid in the subarachnoid space, which contained very

few blood corpuscles, while the subdural space over the same segment of the nerve contained several gross patches of haemorrhage which were evident to the naked eye (Fig. 22). Passing backwards to the point of entrance of the central retinal artery, the sheath haemorrhage became progressively less, until, at the level where the vessels first appeared at the centre of the nerve, there was a mere trace of blood in the subdural space (Fig. 23). No significant haemorrhage was found in association with the central vessels at the point of crossing or elsewhere.

The central retinal vessels and those of the pial sheath showed slight medial sclerosis.

Sections of the optic nerve and surrounding tissues in the posterior part of the left orbit, showed only traces of haemorrhage in the subdural space and still less in the subarachnoid, but there were a number of haemorrhages in the dural sheath, and several of these surrounded or were closely associated with thin-walled vessels of the sheath. Some diffuse haemorrhage was found among the bundles of the orbital muscles, and in the connective tissue framework of the orbital fat. Still more interesting was the discovery, in a region where there was practically no blood in the

FIG. 22.

FIG. 23.

CASE 4.—FIGS. 22 and 23.

Transverse sections of the right optic nerve. 22. A few mm. behind the globe. Massive haemorrhages in subdural space, and distension of subarachnoid space by clear fluid. 23. At the level of the entrance of the central vessels. A mere trace of haemorrhage in the subdural space.

CASE 4.—FIG. 24.

Haemorrhage in connective tissue sheath of ciliary ganglion.

sheaths or spaces, of rather massive haemorrhages in the fibrous sheaths surrounding the ciliary ganglion (Fig. 24) and the posterior ciliary nerves (Fig. 25). This seemed to lend some support to Grimmer's view that in such cases blood might find its way along the ophthalmic division of the 5th nerve and its branches and form a haematoma of the ciliary nerves; but it was found that the haemorrhage in the sheaths of the ciliary

CASE 4.—FIG. 25.

Haemorrhage in connective tissue sheath of posterior ciliary nerves.

nerves was at its minimum in the posterior part of the orbit and increased in amount further forward. The reverse would be expected if the blood were tracking forward. Moreover, the blood surrounding both the ciliary nerves and the ciliary ganglion was confined to the fibrous sheaths of these structures, and was absent from the space beneath the sheaths.

Comment

In this case, as in all but one of the group, there was no massive haemorrhage between the sheaths of the nerve, and such as it was the haemorrhage was mainly in and beneath the dural sheath. There was nothing to suggest the view that the central retinal veins were compressed by haemorrhage within the spaces. The subarachnoid space, on the other hand, was, anteriorly, greatly distended by fluid. If this were sudden in its onset it could conceivably cause both pressure and tension on the vein, as it traversed the sheaths after leaving the nerve, that might cause the sheath haemorrhage anterior to this point as well as the

haemorrhages in the retina. But it seems more reasonable to seek for a mechanism which will embrace and explain at the same time the haemorrhages discovered in the orbital tissues and in the sheaths of the ciliary nerves and ciliary ganglion.

CASE 5.—A man aged 50 years. Admitted to hospital in a state of coma, having collapsed at his work without warning. Blood pressure was 188/98. W.R. negative. The C.S.F. was blood-stained. The right pupil was larger than the left, and unresponsive to light stimuli, direct and consensual, the left pupil contracted slightly to light directly but not consensually, there was slight drooping of the right upper lid and there was paralysis

CASE 5.—FIG. 26.

Subhyaloid haemorrhage in the left fundus. Post-mortem photograph.

of conjugate movement of the head and eyes to the left. O.E. Moderate papilloedema and fulness of the retinal veins in both eyes, and in the left a large subhyaloid haemorrhage occupying the centre of the fundus (Fig. 26). No other haemorrhages discovered. Death 2 weeks after admission. *Post-mortem* examination confirmed the diagnosis of subarachnoid haemorrhage, the source of the blood being a ruptured aneurysm at the junction of the anterior cerebral and anterior communicating arteries. The optic nerves presented a bluish-coloured bulbous expansion behind each globe, and irregular discoloration as far back as the optic canal. Naked eye and slit-lamp examination of the fundi revealed no lesions other than the subhyaloid haemorrhage, and in sections of the left eye only one other small retinal haemorrhage was found. The retinal vessels, especially on and near the disc,

were well filled and many capillaries could be traced by their blood content. No pathological changes were found in the retinal vessels. In many of the transverse sections of the left optic nerve, especially behind the point of entry of the central vessels, there was only scanty haemorrhage; this was entirely subdural in position and showed discontinuity both in the length and in the circumference of the nerve (Fig. 27).

CASE 5.—FIG. 27.

Transverse section of left optic nerve. Scanty subdural haemorrhage and fluid distension of sub-arachnoid space.

In front of this point there was an increase in the amount of haemorrhage, perhaps mainly in the neighbourhood of the emerging vessels; but the blood had no close relation to these vessels, nor could one trace any haemorrhage accompanying the vessels in their course into or through the dural sheath.

At this level, but a little distance from the central vessels, there was a small independent haemorrhage between the lamellae of the dural sheath. Some distance behind this point, where the central artery and vein were emerging from the dural sheath to join the ophthalmic artery and vein, there was haemorrhage in the adventitia of both vessels, especially of the vein. At the

anterior extremity of the optic nerve there was a compact layer of haemorrhage on the inner surface of the dural sheath, but none in the widely dilated subarachnoid space.

The subhyaloid haemorrhage was definitely in front of the internal limiting membrane of the retina, its anterior aspect covered by a thin membrane which at some points showed continuity with delicate membranes or filaments in the vitreous (Fig. 28). The apparent source of this haemorrhage was a well filled vein coming up to the retinal surface, whose blood content seemed to be in continuity with the subhyaloid extravasation (Fig. 29).

CASE 5.—FIG. 28.

Retina and subhyaloid haemorrhage, left eye.

CASE 5.—FIG. 29.

Showing probable source of subhyaloid haemorrhage.

The picture of the fundus of this eye shows that the lower temporal vein, lies deep to the subhyaloid haemorrhage near its lower border, and the microscopic sections show that the vessel above referred to as the possible source of the haemorrhage is a venous branch which joins the inferior temporal vein under cover of the subhyaloid haemorrhage about 3 mm. temporal to the fovea.

Comment

A notable point in this case is the limited extent and distribution of the bleeding. Haemorrhage along the course of the nerve was nowhere abundant, and it was confined to the inner aspect of the dural sheath. As in the other cases, there was no continuous track of blood but a number of discrete effusions. In the retina of the left eye, in addition to the subhyaloid haemorrhage, blood was discovered only at one small point in the nerve fibre layer. Naked eye and slit-lamp examination of the right eye showed a similar condition of the optic nerve and no retinal haemorrhage. The patient was a comparatively young man with a negative Wassermann reaction, and a blood pressure which was not unduly high. Moreover, the microscopic sections showed healthy blood vessels. The veins and capillaries were well filled, perhaps over filled, and yet we did not have the multiple and more massive haemorrhages seen in some of the other cases. We must believe that the retinal vessels as a whole were healthy, but that there were at least two points of diminished resistance in their walls. One wonders whether these were the ocular counterparts of the weakened walls which allow the formation and rupture of aneurysms on the cerebral vessels.

In this case there was no blood in the subarachnoid space of the optic nerve sheath and therefore there is no question of tracking of blood from the cerebral subarachnoid space. We can assert, too, that neither the subdural haemorrhages, nor those in the dura and accompanying the central retinal vessels where they are leaving the dural sheath, can be explained by pressure of blood on the central vein in the perineural space. It is equally certain that the preretinal haemorrhage did not arise by blood passing along the course of the central vessels or by the side of the optic nerve into the retina.

General Comment

The five cases here reported present a certain similarity in their general character; the history of sudden onset with severe pain, the symptoms of cerebral irritation and compression, and coma preceding death. Blood-staining of the cerebro-spinal fluid was, of course, present in all.

They differ more widely in the eye conditions clinically observed: the presence or absence of oculomotor paralysis, and disturbances of conjugate movements, of papilloedema, and of haemorrhage on the optic disc, at different depths in the retina, in front of the retina (subhyaloid) and in the vitreous. There were also certain fundus changes which did not result from the intracranial haemorrhage but were referable rather to a constitutional state which might be the cause of the haemorrhage, as, for example, the picture of hypertensive retinopathy in the first case, and of optic atrophy, probably syphilitic, in the second.

Coming to the pathology of the cases we find a striking and quite unexpected variety in the distribution of the haemorrhage and, incidentally in the state of the ocular and other blood vessels.

Two points of general resemblance were the presence of ecchymotic discoloration of the optic nerve sheaths, at varying intervals from behind the optic canal to the anterior extremity of the optic nerve, and of an ampulliform distension of the sheaths of the nerves immediately behind the eyeball. Subsequent microscopic examination showed that this distension was usually due to the presence of fluid in the subarachnoid space.

Priestley Smith (1884) was apparently the first to notice the phenomenon of blood in the subdural space associated with fluid distension of the subarachnoid space. That this is not very exceptional is shown by its occurrence in four of my five cases, the fifth case showing complete filling of all the spaces by blood (Compare and contrast Figs. 16 and 22).

The cause and significance of this distension of the sheath are not clearly understood. If Wolff is correct in his finding of a patent communication between the intracranial subarachnoid space and that of the optic nerve, the sudden rise of intracranial pressure could cause cerebro-spinal fluid to pass into and distend the latter. Another consequence would be the early occurrence of papilloedema which has been reported in some cases. The characteristic form and limited extent of the distension cannot be fully explained by its relation to the central retinal vessels as they cross from the sheaths to the nerve. This crossing point is some 12 to 15 mm. behind the globe, while the ampulliform swelling is not more than 7 mm. in length. Some anatomical explanation is required for the failure of the oedema to spread backward along the nerve sheaths.

It would appear that it is possible to have a good deal of blood in the subarachnoid space of the optic nerve without any discoloration of the sheaths, and that on the other hand the familiar blue ecchymotic appearance is due, not so much to the effusion of blood in the subarachnoid space, as to the haemorrhage in or beneath the dural sheath. Even in the intracranial part of the

optic nerve the haemorrhage may be only dural and subdural, and with haemorrhage present in the sheaths both before and behind the optic canal there may be no haemorrhage within the canal itself. This fact of course conflicts with the often repeated statement that the blood in the middle fossa passes, by continuity of the subarachnoid space, through the optic canal and along the course of the orbital portion of the nerve. This view seems to be the result of a too restricted investigation of the pathological material, and Liebrecht seems to be the only observer who has tried to trace the source and distribution of the blood by serial sections. On a superficial view the foregoing statement might seem to be consistent with the appearances in our third case; but examination of serial sections in all the other cases shows that separate effusions of blood occur at varying intervals along the course of the optic nerves, and may be found on the outer surface of the dural sheath, in the dura itself or in the subdural space, in the subarachnoid space and even in the pial sheath. They may occur around the vessels of the circle of Zinn, among the nerve fibres of the optic papilla, between the neuroepithelium and pigmented epithelium of the retina, and in the vitreous; on the episcleral surface of the eyeball, among the orbital fat and muscles, and in the fibrous sheaths surrounding the ciliary ganglion and posterior ciliary nerves. In many of these situations a ruptured vessel has been found in the midst of the haemorrhage, or a vessel with blood effused among the coats of its wall (Figs. 7, 30, 31). The discovery of many such haemorrhages within the

CASE 3.—FIG. 30.

Dissecting haemorrhage in vessel on outside of dural sheath of optic nerve.

chiasma and optic tracts in our second case (Figs. 9 to 14) suggests that if our investigation were to be carried further in similar cases we might find other examples of this distribution.

One would draw attention especially to the sections in Case 5, in which the walls of the central retinal artery and vein were free from haemorrhage in the pial sheath, the perineural spaces and

CASE 5.—FIG. 31.

Dissecting haemorrhage from large vessel in the dural sheath of optic nerve.

the dural sheath; whereas, some distance behind this point, where the vessels emerge from the dural sheath to join the ophthalmic artery and vein, haemorrhage was found in the adventitia of both vessels. Clearly, this could not be explained by pressure on the central vein of the retina where it crosses the perineural spaces, even if there had been evidence of such pressure.

These considerations indicate that we cannot be satisfied with the view that the intracranial haemorrhage passes along the sheaths of the optic nerve, and that it compresses the central retinal vein at its crossing of the perineural spaces and thus leads to haemorrhage into the retina. We must look for a mechanism which will

cover the wide field illustrated by these cases. It is not impossible that distension of the subarachnoid space, either by blood or by serous fluid, could cause a sudden pressure and tension on the central vein as it crosses the space, and thus bring about haemorrhage from any of the tributaries of that vein, but that could only apply to the limited area distal to the crossing of the central vessels.

It is natural that the discovery of haemorrhage surrounding the ophthalmic artery and the central retinal vessels outside and in the dural sheath, and of similar haemorrhage accompanying the posterior ciliary nerves and the ciliary ganglion, should suggest the tracking of blood from the middle fossa. It would be natural, too, for subarachnoid haemorrhage to extend some distance within the space, or for blood to spread between the lamellae of the dural sheath, but the study of serial sections shows that these effusions of blood cannot be followed over a long course. They are of quite limited extent, and cannot be shown to have any connection with the intracranial sheath spaces; moreover, as we saw in regard to the haemorrhage surrounding the ciliary nerves and ganglion (Case 4) the blood is not found in the perineural lymph spaces, but infiltrates the connective tissue sheaths of these structures; and the haemorrhages accompanying the vessels outside the dural sheath, and elsewhere, were found to be splitting up the layers of the vessel wall.

There is little doubt that the multiple haemorrhages are the result of sudden venous stasis, and that they are simultaneous and independent of each other. It would seem justifiable to assume that, when the massive intracranial bleeding occurs, there is a sudden rise of intracranial pressure which, acting upon the veins and sinuses, which receive the venous return from the eyeball and other orbital contents, produces the stasis necessary for the occurrence of the haemorrhages.

The reasons for differences in the distribution of the haemorrhages in different cases must be largely a matter for speculation. Sudden rise of pressure throughout the veins of the eye, the optic nerve and the orbital contents may be supposed to be a common factor, and no doubt the vulnerability of the vessels may be influenced by such constitutional disorders as syphilis, renal disease, or tuberculosis; but for the rest, we must assume the existence of *loci minoris resistentiae* in the walls of the veins. Such *loci* are believed to be prone to occur at the bifurcations and junctions of vessels; and points where the vessels suffer a sudden change of direction, or an alteration of the support afforded by the surrounding tissues, as for example, where the central vessels pass from the optic nerve to its sheaths or from the lamina cribrosa to the retina, may be relatively weak. The condition shown in

Fig. 18 suggests that another such point may lie at the entrance or exit of the central vessels from the dural sheath.

Summary

Five fatal cases of spontaneous subarachnoid haemorrhage are described with special reference to the pathological condition found in the eye and its related structures.

The discovery of haemorrhage in the subarachnoid space at the base of the brain, within the sheaths of the optic nerve, and in many cases in the retina and even in the vitreous, originally led to the belief that the blood passed in a continuous manner in the subarachnoid space through the optic canal into the orbit, and thence either along the course of the central retinal vessels, or by lymph channels accompanying the optic nerve at the periphery of the lamina cribrosa into the retina.

It is now generally agreed that the blood in the cerebral subarachnoid space does not, as a rule, travel through the optic foramen into the subarachnoid space of the optic nerve. It is more generally assumed that haemorrhage occurs in the dural sheath at the apex of the orbit, and tracks forward into the subdural and subarachnoid spaces, and that blood in the latter situation, by putting pressure and tension on the central retinal vein where it crosses the space, causes stasis in the central retinal vein and thus accounts for the retinal and vitreous haemorrhages.

On the basis of the five cases here described it is shown that haemorrhages occur over a much wider field than the optic nerve sheaths, the retina and the vitreous, and that these cannot be accounted for by the assumed pressure on the central retinal vein.

The haemorrhages varied in distribution from case to case, but were found in and between the sheaths of the optic nerve, among the orbital fat and orbital muscles, surrounding the posterior ciliary nerves, ciliary ganglion, ophthalmic and posterior ciliary arteries, and the vessels of the chiasma and optic tracts; as well as beneath the retina, in all its layers, in front of the retina and in the vitreous. Serial sections showed that these were not in continuity, one with another; but were discrete and independent haemorrhages, simultaneous in their occurrence; and it is submitted that the occurrence of such multiple haemorrhages can only be explained by a sudden rise of intracranial pressure causing a stasis in all the venous channels which drain the tissues of the eye and the contents of the orbit.

It is suggested that some of the clinical signs of subarachnoid haemorrhage, such as oculo-motor pareses and disturbances of conjugate movements of the eyes, may be explained by the occurrence, in the midbrain, of haemorrhages similar to those found, in one of our cases, in the chiasma and optic tracts.

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A CASE SHOWING PARTIAL DEFICIENT FUSION OF A MAXILLARY PROCESS WITH LATERAL NASAL PROCESS ON ONE SIDE

BY

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WEST AFRICAN MEDICAL SERVICE

By an odd coincidence I saw the case here described just after I received the January number of the Journal containing an account of cases with malar bone deficiency. As I have access to a very limited literature here I am unable to say much about it.

The patient in this instance is an African baby girl, aged 3 months. The first child of healthy parents who state that there is no history of hereditary defects in the family.

The outstanding features on first viewing the patient were :

1. The distortion of the right nostril with a hiatus showing the interior of the nose—the cleft extending upwards to within about 1 cm. of the inner canthus.

2. The abnormality of the inner canthus—wide obtuse—angled.

With more detailed examination the following additional abnormalities were noted :