

in the anterior part of the temporal fossa, whereby part of the malar bone, including the external angular process and the zygoma, were detached as a loose fragment. The lungs were somewhat hyperæmic and oedematous. There was no obvious lesion of the heart, kidneys, or other viscera. The brain and lungs were hardened in formalin, microscopical sections cut, and stained with Sudan III. They showed the capillaries to be blocked with fat; in the lung the smaller arterioles were also filled with fat.

This case differed from the first one in that the injuries were much more extensive, and that clinically the course of the illness was much more rapid, while the post-mortem examination did not show either multiple hæmorrhages in the brain or broncho-pneumonia in the lungs, both of which changes must be regarded as secondary.

It will be thus seen that both these cases presented very similar symptoms and signs, they both had fractures of the thigh, in one case a comparatively simple fracture and in the other two simple fractures, together with some crushing of the bones about the knee-joints and a lacerated and contused wound of the foot. They were both given chloroform for the setting of bones. Within about 12 hours they both developed a comatose state more or less suddenly, without any previous noticeable pulmonary distress, the temperature rose rapidly, the pulse was of low tension and rapid, and there were no localising signs in the nervous system pointing to a lesion in any particular part of the brain. Both might have been thought to be cases of cerebral compression if it had not been for the character of the pulse. One of them was trephined over the right side because he had evidence of injury to the skull on that side. The operation probably did not hasten his death, the only cause of which that could be demonstrated was fat-embolism of the brain. The other patient recovered to a slight extent from what appeared to be an almost hopeless condition, and his death was no doubt partly due to a secondary broncho-pneumonia; in this case also some of the typical signs of cerebral compression were absent.

There are three points of especial interest to be noted.

1. The difference in the extent of the original injury in the two cases. Whereas one might have expected fat-embolism to occur in the case where there were several fractures, some of which were of a crushing nature, there was nothing about the simple fracture in the other case to suggest the likelihood of its occurrence, or at all events of its causing any grave symptoms.
2. The absence of dyspnoea or other symptom of pulmonary embolism in one case and the fact that the other patient only complained that his bandages felt tight.
3. The resemblance superficially of the condition to that of cerebral compression, but only superficially, because the characteristic pulse of compression was never observed. There were never any signs in the nervous system pointing to a localised injury to the brain.

It will be interesting to note how far the two cases described above compare with other published examples of fatal cerebral fat-embolism.

Hämig¹ collected the published cases in 1900, taking only those in which no other cause of death could be found. He gives 12 and adds 5 of his own. The older clinical reports are valueless on account of the complications resulting from sepsis. All the cases had suffered fracture of one or more of the large long bones, and except for shock their general condition was good when first seen. After a latent period varying from six hours to nine days coma supervened, sometimes, but not usually, preceded by delirium. The temperature showed a rise coincident with the onset of the coma, and the rise continued until death. In two cases there were signs suggesting a local lesion of the brain, so that one of them was actually trephined over the middle meningeal; this was done in spite of the fact that the pulse had not a high tension. As in our case nothing was found. Fat was found in the urine in nearly all the cases. There was slight cyanosis in some of the cases, but in none was there any evidence of extensive involvement of the pulmonary circulation. The pulse was in all the cases rapid and of low tension. The post-mortem appearances were much the same, the brain showing numerous punctate hæmorrhages, the lungs an early broncho-pneumonia. The same extensive microscopical evidence of fat-embolism was also a feature.

Oehler² published two cases, only one of which was fatal, so that this one alone can be taken as a definite case. It had fractures of the right femur and tibia; there was no rise of temperature and no fat was discovered in the urine, but the post-mortem findings were the same as in our cases. The other case certainly corresponded in its course to the general clinical picture, but no fat was found in the urine.

Other cases are reported as a result of forcible manipulation of ankylosed joints. The importance of a recognition of the condition cannot be over-estimated, more especially in view of the difficulty of diagnosis from intracranial hæmorrhage. It is well known that hæmorrhage may occur some time after the original injury, a latent period similar to that seen in these cases being no uncommon occurrence.

As points in the differential diagnosis we would lay stress on (1) the absence of the high blood pressure seen in cerebral compression; (2) the absence of isolated paralyses; and (3) the absence of fulness of the retinal veins.

In thinking over these cases one cannot but be struck with the fact that out of a large number of severe accidents only three had symptoms and signs that could be attributed to fat-embolism, and however striking may be the coincidence of the symptoms and the post-mortem appearances it is impossible to forget that Cohnheim and others have thrown doubt upon the whole matter, because it has been shown that large quantities of fat can be injected into animals causing extensive embolism without in some cases producing even serious symptoms.

SOME OCULAR AND VISUAL CONDITIONS IN MEDICAL CASES.¹

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IN order to make what I have to say of as much practical use as possible, I am basing my remarks upon observations which I have made, for the most part, on cases referred to me at Moorfields by the surgeons to that hospital during the last five years. I propose to divide my remarks into five sections, first of all considering conditions of the pupil; secondly, conditions of ophthalmoplegia externa; thirdly, changes in the optic nerve; fourthly, retinal changes; and fifthly, hemianopia.

First, with regard to pupil conditions. Inequality of the pupil is by no means uncommon, and is not necessarily, in my opinion, associated with serious morbid conditions. The late Marcus Gunn, however, was strongly of opinion that a constant inequality of pupils in an adult usually meant some neurotic predisposition. When it is met with in children some conditions affecting the cervical sympathetic should be looked for. And it is not infrequent to find such inequality of pupil, not only in children, but also in older patients who have suffered from cervical glandular enlargement or suppuration in early life. Another condition of pupil which is of great importance, and undoubtedly of great significance, is that condition in which it is inactive to light, while still reacting during accommodation—the Argyll Robertson pupil. This was described, as you all know, in the first place in connexion with tabes dorsalis. But further observation has shown that it exists in many other conditions, more especially in patients who have been the subjects of syphilis, and not infrequently in association with general paralysis of the insane. Irregularity in the shape of the pupil is frequently associated with this inaction to light. Dr. Farquhar Buzzard has recently alluded to its occurrence in connexion with lesions in the neighbourhood of the third ventricle, but of this I have myself no personal experience. The question has been asked, Do we ever find the Argyll Robertson pupil unassociated with any other condition? I have notes of several cases in which it persisted for several years under observation without any associated symptom or sign of nervous disease developing. One such patient I saw 16 years ago on account of neurasthenic symptoms. At that time I examined him carefully, but could find no sign of any organic disease except the

¹ Beiträge zur Klinischen Chirurgie, 1900, Band xxvii., p. 333.

² Ibid., 1909, Band lxx., p. 11.

¹ A paper read before the Medical Society of London on Jan. 9th, 1911.

existence of slightly unequal pupils, which were inactive to light. I suspected it might be early general paralysis. That man I have recently seen, and he has still unequal Argyll Robertson pupils, as well marked as they were 16 years ago. And yet there has developed no other sign of disease except that he has become deaf in both ears, apparently the result of early syphilis also. In some cases the Argyll Robertson pupil is present on one side only. Of this I have seen a good many examples; and, curiously enough, in two of the cases on the side on which the Argyll Robertson pupil was present the knee-jerk was absent; while on the other side, the side on which the pupil acted to light, the knee-jerk was present. This, of course, was probably only a coincidence.

In some cases in which the Argyll Robertson pupil is present, optic atrophy may also coexist, and in some such cases the knee-jerks are absent; in others the knee-jerks are active. The former, of course, naturally fall into the class of tabetic optic atrophy. In the latter, I think, there is always a presumption and a fear lest general paralysis of the insane may develop. In some other cases with Argyll Robertson pupil, paralysis—hemiplegia or paraplegia—has been present; and I have also seen it in a case with wasting about the shoulders—the result of syphilitic pachymeningitis. So that, to recapitulate, we may find the Argyll Robertson pupil present without any other morbid sign; we may find it present on one side without any sign of other disease of the nervous system; we may find it present on one side with signs indicating involvement of other parts of the nervous system, such, for example, as the absence of one or both knee-jerks. We may find it also present with very definite signs of tabes, or with very definite signs of general paralysis of the insane. It may also be present associated with marked atrophy about the shoulder girdle. It may be present with a condition of paraplegia, also syphilitic in origin; and I have known it present in a case in which there was a history of repeated hemiplegic attacks, those, no doubt, being the result of syphilitic thrombosis.

I now pass on to the second condition—namely, ophthalmoplegia externa. The conditions determining this are varied. One occasionally meets with paralysis of one external rectus muscle, and the question arises whether such paralysis may be the result of neuritis from cold affecting one sixth nerve. In such a case the paralysis would be analogous to the ordinary form of facial or Bell's paralysis, but I am bound to say that in most of the cases in which cold is supposed to be the exciting cause, a careful investigation fails to reveal sufficient justification for this explanation. It is also curious to think that if paralysis of one external rectus may be the result of cold, it does not happen to occur in association with a similar paralysis of the seventh nerve. And I may mention that in all the cases of facial paralysis of peripheral origin that I have seen—and they must now run into hundreds—I have never observed an associated weakness in the sixth nerve. The sixth nerve undoubtedly not infrequently suffers as the result of an accident. If one remembers the long course of the nerve and the delicacy of its filament, it is not surprising that in cases of concussion or fracture such an accident may cause paralysis of one sixth nerve. Third nerve paralysis is probably one of the commonest that we meet with. And in the great majority of cases it is, I believe, syphilitic in origin. The lesion, I think, is gumma occurring in the sheath of the nerve and pressing upon the filaments; and frequently complete recovery results from the effect of antisyphilitic treatment. In some cases, however, recovery does not take place. I have known two cases of third nerve paralysis occurring in patients whom I knew before as being the subjects of Argyll Robertson pupil. In both instances the third nerve paralysis cleared up completely, but the patient in each case died within a very few years from general paralysis of the insane. I need only refer to many of the conditions with which ophthalmoplegia externa is associated, such as tubercular meningitis, post-diphtheritic paralysis, and intracranial disease. In intracranial tumour the nerves supplying the eye muscles may suffer from direct involvement in the tumour. They may, however, suffer indirectly even in cases in which the growth is far removed from them—e.g., in the cerebellum—either from pressure against the bone, or, as Harvey Cushing has recently suggested, from pressure on them by branches of the basilar artery. In tabes, also,

besides the paralysis of the third nerve, which may clear off under antisyphilitic treatment, there may also be permanent paralysis of ocular muscles, probably the result of degeneration of the nuclear cells. There is a form of hemiplegia with which third nerve paralysis is associated, the so-called *migraine ophthalmoplégique*. This probably depends upon a lesion occurring in the course of the third nerve itself; and the condition is characterised by recurrences of paralysis of the third nerve with recurrent headaches, the paralysis clearing up less and less completely after each attack, and finally settling down into complete paralysis of all the branches of the nerve.

One disease with which ophthalmoplegia externa is associated is of great importance because of the danger there is of mistaking it for a hysterical condition. I mean the condition of myasthenia; and I might mention a case of this class which was referred to me not very long ago at Moorfields by Mr. Holmes Spicer as one which is very instructive, in the suddenness of the onset, in the rapidity of the course, and in the conditions which were found post mortem. The patient was a young married woman who suddenly found that after she had gone a little way from home one day she became so tired that she was scarcely able to drag one leg after the other. She was able to return home only with considerable difficulty. But the weakness persisted, although at times in the morning when she got out of bed she was able to move about for a time quite actively. This weakness of the legs was associated with a gradual heaviness and drooping of the eyelids, and when she came into hospital she had double ptosis, almost complete paralysis of all the ocular movements, variable, however, in its degree, and attacks of very great weakness, with difficulty in breathing. She finally died, after only about six months of illness, and at the post-mortem examination there was found present a very large thymus, which was completely covering the heart. The nervous system was quite normal. I am publishing details of this case in another place, but I mention it merely as being so characteristic of the sudden manner in which the disease may arise, the rapid course which it runs, the absence of changes in the nervous system, and the morbid condition of the thymus which is found in, at least, some cases.

Thirdly, I should now like to refer to conditions of the optic nerve that are found in cases which have other medical signs and symptoms. Atrophy of the optic nerve is found especially in tabetic cases. It is said to occur in the pre-ataxic stage of tabes. But I should venture to demur to this statement, because I am quite sure that many of the cases of tabes with optic atrophy never become ataxic at all. Sometimes the optic atrophy of tabes is associated with Argyll Robertson pupil; sometimes that condition of pupil is not present. Perhaps it is hardly correct to say that in some of these cases of tabetic optic atrophy the knee-jerk is present, because then it may be said, "Well, after all, perhaps these cases are not tabes." And that is quite true, but many of those cases, I believe, are tabes, for I have frequently known the knee jerk disappear under observation. Many of them are general paralysis of the insane, and perhaps it is the more correct way to regard those cases as really cases of one and the same disease—a parasyphilitic disease affecting in one case chiefly the spinal cord and the optic nerve, and in the other case affecting the higher centres and giving rise to mental changes and also involving the optic nerve. In disseminated sclerosis there is a form of atrophy met with, but this, in my experience at all events, contrary to the tabetic atrophy, never leads to blindness. I have never known a patient with disseminated sclerosis become permanently blind, whereas in tabes when optic atrophy once sets in it is the rule, almost without exception, for the patients rapidly to lose sight altogether. In disseminated sclerosis also it is not infrequent to have a central scotoma for colours present, and in that sense the atrophy resembles very closely that met with in the family form of optic atrophy, and also the pallor of the optic nerve which is present in so many cases of toxic amblyopia. I refer later to optic neuritis occurring in disseminated sclerosis.

One condition of optic atrophy deserves to be mentioned—that which occurs with tumours pressing on the optic chiasma. Of course, there is associated with it bitemporal hemianopia, but the interesting point is that the condition developed as a result of pressure there is not one of optic neuritis so-called, but one of a simple atrophy. I have only seen one case in

which definite symptoms of tumour at the chiasma were present, in which there was optic neuritis.

With reference to optic neuritis there is no doubt that tumour of the brain is by far the most efficient cause of this. I was rather surprised to see in a recent paper by Sir Victor Horsley the following statement: "I am, of course, aware of instances in which the occurrence of optic neuritis was said to have been the first symptom calling for notice, but I have not myself seen such chronology in a case of intracranial tumour, and would rather suggest that the neurological symptoms had not been observed." Perhaps the statement is natural from the fact that patients would hardly be likely to seek the advice of a surgeon or a physician on account of visual defect from optic neuritis, but that it is a statement requiring considerable qualification will, I think, be sufficiently evident if I mention that I have looked through the notes of the cases referred to me at Moorfields during four years, and in those four years I have come across no less than 20 cases of patients who had gone to Moorfields as the first hospital they had been to on account of eye symptoms, and in all those cases optic neuritis of some intensity was present. Some of them then had, or developed subsequently, other signs of intracranial growth. In a few cases of disseminated sclerosis I have known optic neuritis to be present. The lesion of the optic nerve in this disease is, I believe, as a rule, retro-bulbar neuritis, as Dr. Thomas Buzzard long ago pointed out, but one can easily imagine such a patch of inflammation occurring quite near to the disc and actually encroaching upon it, and so giving rise to this appearance. I think that certain toxic conditions also may give rise to optic neuritis. It has been described, of course, in association with anæmia, and it has also been described as a sequel to influenza. Personally, I have only seen one case of post-influenzal neuritis, or at all events of neuritis occurring after an acute illness of the influenza type, and in that case the neuritis certainly cleared up without any further symptom developing.

The fourth condition to which I should like to refer is that of neuro-retinitis. That may occur, of course, in association with albuminuria, also with glycosuria; and the appearances are sufficiently distinct and characteristic to require no description. One condition which I do not think is quite so common or quite so well recognised, except by ophthalmic surgeons, is the condition of retinal thrombosis. A patient comes complaining of sudden loss of vision or sudden impairment of vision in one eye. It is not often that the condition affects both eyes, at all events simultaneously. On examination of the eye the condition which is found is one of, apparently, blocking of a vein, sometimes of an artery, many hæmorrhages, and a good deal of effusion round, and a general blurring of, the retina. Sometimes the condition clears up very rapidly, so that the patient's vision in a few weeks' time is almost as good as it was before. In other cases the clearing up is extremely slow and never becomes anything like perfect. I have been interested in observing the associated conditions of the circulation, and I have been very much struck with the fact that in many such cases there is no sign of any other disease, no albuminuria, and no cardiac hypertrophy. In some cases, however, cardiac hypertrophy is present, but it is certainly exceptional. And I suppose that one must assume that in such cases in which there is no associated visceral disease the condition really depends upon local arterio-sclerosis, for some of the patients in whom I have known the condition occur are now alive after as many as six or seven years.

Embolism of the central artery of the retina is also a condition to recognise. Of course, it is usually associated with cardiac disease, and the characteristic appearances are too well known to need description. I have seen it, however, in one case in which other observers as well as myself could find no morbid heart condition. But it occurred immediately after diphtheria, and is, I presume, the result of detachment of some little patch of fibrin from a heart valve, although the heart valve showed no incompetence. It is interesting to note that I have seen four cases of hemiplegia occurring in children after diphtheria without cardiac disease, and in all of those the mechanism probably was similar.

I would now refer briefly to the fifth section—the condition of hemianopia, and it ought to be recognised that homonymous hemianopia may occur quite independently of any other paralysis. I have known it now in several

instances, and the cases in which it has occurred have been mostly syphilitic. I have known it, however, as a result of embolism, although in most of these cases there is a history of a transient weakness of the corresponding side of the body. But when the patients come under observation the condition is one of simple uncomplicated homonymous hemianopia. We must assume in such cases that there has been a lesion, probably embolic or thrombotic, in the neighbourhood of the posterior part of the internal capsule, in such a position as to interfere permanently with the function of the optic tracts, while allowing the other sensory fibres and the motor fibres to escape completely. Bitemporal hemianopia, of course, is found only in cases in which a lesion is present at the chiasma, and it may be associated with signs indicating the presence of acromegaly.

I am aware of the extremely discursive character of these remarks, yet I trust that they may arouse some interest, or at least impress some of those who have done me the honour to listen to them, with the importance and significance of eye signs and symptoms and the suggestions which they imply of associated morbid conditions.

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TWO CASES INDICATING THE VALUE OF THIS METHOD FOR THE DETECTION AND REMOVAL OF FOREIGN BODIES IMPACTED IN THE LOWER AIR-PASSAGES.

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THAT the entry of a foreign body, be it large or small, soft or hard, into the lower air-passages is an accident of the gravest nature is a statement the truth of which our profession has always recognised. For not only has the surgeon been cognisant of the evil, and probably fatal, results which sooner or later would result from the impaction of a foreign body in one of the deeper main airways, but until quite recent times he has been only too well aware that any attempts made to remove such obstructions were often of the nature of blind gropings in the dark, and that his intervention might hasten, rather than retard, the evil consequences of the accident.

How very different is the outlook to-day, when, thanks to the genius of Killian and the inventive mind of his assistant Brüning, practical instruments have been constructed whereby we are enabled to pass a straight, illuminated tube by way of the mouth, larynx, and trachea to the very roots of the lungs, and by direct vision inspect every inch of the way. Surely in the bronchoscope we have reached one of the highest ideals of "bloodless surgery," for by means of its cunningly devised arrangements we can not only see and remove any intruding foreign bodies which may have found their way to these hitherto hidden depths, but in other instances we may perchance ascertain the nature and situation of pathological conditions endangering the life or well-being of the patient. The following two cases will illustrate my contention and should go a long way in proving that the bronchoscope is no longer a surgical toy, but that it is an instrument of the greatest practical value and one with which surgeons should make themselves familiar.

CASE 1. *Removal of a portion of rabbit bone which had been impacted in the right bronchus for more than three years and had produced symptoms of bronchiectasis.*—The patient, a male, aged 46 years, was admitted into University College Hospital, under the care of Dr. J. Rose Bradford, on Jan. 9th, 1911, complaining of "feeling ill and coughing up blood." Three years previously the patient had "swallowed a rabbit bone, which seemed to stop at the bottom of the throat and to cause a tickling feeling between the shoulder blades." He then began to suffer from a cough which had lasted ever since. Fourteen days after swallowing the bone he was removed to an infirmary, where he was treated for "pain in the back, chest, and stomach." Eighteen months ago he had coughed up some streaks of blood for the first time. There was shortness of breath on exertion, he complained of a dry throat, and he had wasted very considerably during the past six months.

On admission to hospital the patient was found to be a