It was possible later to withdraw a microfilaria from the anterior chamber; this was found on staining to be *Onchocerca volvulus*. A skin snip showed many microfilariae (*O. volvulus*), although no nodules could be felt. The right eye showed one fresh yellow speckled opacity in the cornea, which I have seen only in cases of onchocerciasis.

The patient was seen again in 5 and 7 months. He was well and had no trouble with vision or with pain in the eyes despite the presence of microfilariae in each anterior chamber. The fundi were normal, except the faintest veiling of the margins of the right disk.

Case II

The second case was admitted on July 3, 1943, with a painful swelling in the right axilla, mild oedema of the right upper eyelid, and ciliary flush. The axilla was incised and cleared in a normal fashion. The right eye, however, soon became closed with oedema of the upper lid, and proptosis developed. Both fundi remained normal, but vision dropped line by line over a period of 10 days from 6/9 on admission to 1/60. The right eye was painful with lacrimation. Radiographs of the orbits were normal. The Kahn reaction was negative. Central nervous and other systems were normal. The patient was unable to co-operate in perimetry. Day and night films showed scanty microfilariae (*A. perstans*), but a skin snip had many *O. volvulus*, and typical nodules were felt over the right and left iliac crests. No microfilariae were seen in either anterior chamber, but fine dots of pigment were observed on the posterior corneal surfaces.

On the 17th day vision was 6/12 R., 6/5 L.; oedema of the lid was lessening and proptosis and ciliary flush had cleared. On the 27th day vision was 6/5 R., 6/5 L. Slight fullness of the right upper lid remained. The right fundus was normal throughout, except for a transitory haziness on the 7th day, so the neuritis in this case was retrobulbar.

Discussion

The syndrome presented by these two soldiers is not a familiar one in general ophthalmic practice. In the cases described the diagnosis must rest on probabilities, since no single finding was sufficient to establish the diagnosis beyond doubt. The presence of oedema of the upper lid, proptosis, ciliary flush, and optic neuritis indicates a lesion behind the eyeball. The probable causes are (a) haemorrhage, (b) tumour, (c) infection, (d) congestion.

(a) Haemorrhage can be excluded by the absence of subconjunctival echymosis.

(b) Tumour is improbable in view of the complete recovery and lack of other signs pointing to neoplasm or cyst.

(c) Infection (i.e., an orbital cellulitis or a cavernous sinus thrombosis) is not supported by the findings. The absence of a history of previous sinus trouble, the healthy appearance of the orbit on x-ray examination, and the normal white cell count are against orbital cellulitis. The absence of a primary lesion on the face or in the mouth is against cavernous sinus thrombosis. A syphilitic optic neuritis is not commonly unilateral, and in the absence of choroidal changes and unequal pupils this diagnosis is improbable, especially in view of the negative Kahn in the second case. The group of demyelinating diseases has to be considered. Disseminated sclerosis is the commonest cause of a unilateral retrobulbar neuritis, but does not produce proptosis. Furthermore, this disease is very uncommon on the West Coast. Dr. H. Thomas has neither seen nor heard of a case in 16 years' medical service on the coast, and Dr. Carmichael Wilson has seen only one case in Nigeria in 18 years' service. Neuromyelitis optica and Schilder's disease are ruled out by the subsequent history of the cases.

(d) Congestion can be venous or lymphatic. Venous congestion is ruled out by the absence of early fullness of the retinal veins. The association of lymphatic swelling and filariasis is well recognized in elephantiasis, although the mechanism is not quite clear. The filariae in most cases are *Wuchereria bancrofti*, but Strong reports cases due to onchocerciasis. Calabar swelling, an anaphylactic oedema, is associated with *Loa loa*, so there is a precedent for suggesting that onchocerciasis can produce lymphatic swelling. A direct block of the retrobulbar lymphatics by a host of microfilariae is possible, but since microfilariae are well tolerated as a rule by the tissues this is not considered likely. More probable is the theory that an anaphylactic oedema can be produced in the orbit as a result of *O. volvulus* toxins, and that such an oedema is the explanation of the syndrome of oedema of the optic nerve and of the upper lid, with proptosis and ciliary flush.

Summary

Two cases with proptosis, oedema of the lid, ciliary flush, and oedema of the optic nerve associated with onchocerciasis are described. One is believed to be the first case of onchocerciasis reported in a Gambian. It is suggested that the cause of the syndrome is an anaphylactic oedema caused by *Onchocerca volvulus*.

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THE NERVOUS COMPLICATIONS OF INFECTIVE HEPATITIS

BY

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The nervous complications which may arise during the course of infective hepatitis are by no means common. They occur occasionally in spirochaetosis icterohaemorrhagica, but they seem to be even rarer, judging from the literature, in the epidemic form of infective hepatitis.

Nervous Complications of Weil's Disease

Apart from such conditions as mild headache, restlessness, delirium, convulsions, and coma, which are due more likely to the general infection than to any actual involvement of the nervous system, the nervous complication most often met with in Weil's disease is that of meningitis. Yet, judging from reports of cases in the literature, this is of comparatively infrequent occurrence, despite the writings of some French physicians. Meningitis occurred, for instance, in only 3 of the 40 cases of this disease reported by Davidson and Smith (1936).

Meningitis may occur in Weil's disease with or following the jaundice. It may even be the prominent feature in this infection, the jaundice and haemorrhages being slight or even absent—"spirochètose méningée anictérique" of Costa and Troisier (1916, 1917). The cerebrospinal fluid is under pressure, and it may be clear or opalescent, with a fibrinous clot which forms on standing. There is a pleocytosis, with a predominance of the polymorphonuclear variety in the graver cases, lymphocytes in the less severe. Spirochaetes may be found in the fluid, and if the latter is injected into suitable animals jaundice and haemorrhages will generally occur. Eleven cases of meningitis of leptospiral origin, proved by the inoculation into guinea-pigs of the patient's blood or cerebrospinal fluid, have been reported by Costa and Troisier. Cochez and Fichet (1933) have published an account of three similar cases with, however, no obvious signs of jaundice. Inoculation of the urine, blood, and cerebrospinal fluid into guinea-pigs caused jaundice and haemorrhagic lesions in the liver and lungs, and spirochaetes were found in the livers. Agglutination reactions against *L. icterohaemorrhagiae* were positive in the sera of all three patients. These authors emphasize the following additional clinical signs as particularly characteristic of this disease: mild meningeal signs, labial or palpebral herpes, and conjunctival infection. Troisier and Boquien (1933) have examined a number of case histories of patients suffering from meningitis of leptospiral origin, accompanied with, or without, jaundice. Schüffner and Walch-Sorgdrager (1940) had, up to 1936, collected 25 case reports of meningitis from among 312 Dutch patients suffering from Weil's disease. According to their experience, meningitis is commoner in this infection without jaundice; so it seems advisable that serological tests should be carried out in all cases of non-pyogenic meningitis in which the aetiology is obscure. According to the reports of most

authors, nervous symptoms tend to appear during the acute phase of the disease, although Davidson and Smith (1936) and Murgatroyd (1937) have each reported a case of Weil's disease in which meningitis occurred at least four months after the jaundice.

Recently I have seen a patient with Weil's disease in whom the illness started with violent headache and vomiting. Two days later jaundice appeared. On admission to hospital there was slight stiffness in the muscles of the neck. The cerebrospinal fluid was under increased pressure and was pale yellow in colour. It contained 75% neutrophils and 25% lymphocytes, and 80 mg. of protein per 100 c.cm. The gold curve was normal. No spirochaetes were found in the fluid. There was marked hepatic and renal impairment. Nevertheless this patient made a good recovery, both clinically and biochemically.

Whereas in Weil's disease meningitis may not be quite so uncommon as is generally supposed, yet the involvement of the nervous parenchyma itself is decidedly rare. Mortensen (1940) was able to find only four cases in the literature in addition to one of his own. Two of these patients suffered from peripheral neuritis in the lower limbs, the others with myelitis. His own case was one of jaundice which at first was thought to be due to Weil's disease; but the serological reactions were abnormal, so that it was more likely to be due to an infection with a leptospira of a different type. Three weeks after the beginning of the illness signs of meningitis appeared, together with a transitory flaccid paralysis of the lower extremities. The patient made a good recovery. The same author has found in the literature the reports of a few cases of Weil's disease with paralysis of the third, sixth, and seventh cranial nerves.

Nervous Complications in Epidemic Hepatitis

Nervous complications seem to be uncommon in epidemic hepatitis, although perhaps if a special watch were kept their occurrence might be found to be more frequent than the literature would suggest. As in leptospiral infections, the least uncommon of these nervous complications is meningitis.

Fliessinger (1924) has reported the case of a young man who developed meningitis. Less than a week after the onset jaundice appeared. He was thought to be suffering from Weil's disease, but spirochaetes were never found in the blood, nor in the urine or cerebrospinal fluid; and although inoculation of some of the patient's blood into a guinea-pig caused the death of the animal, jaundice did not occur, nor were haemorrhages or spirochaetes found in the internal organs. The patient's serum did not agglutinate leptospirae. Among the 300 cases of epidemic hepatitis of which an account was published by Ford (1943) there was only one that showed signs of meningitis. Waring (1943) has reported a case of this type of hepatitis in which meningitis started five days after the onset of jaundice. The cerebrospinal fluid was opalescent and under increased pressure. There were 544 cells per c.mm., chiefly lymphocytes, together with an increased amount of globulin. This patient made a good recovery.

Lemierre, Lhermitte, and Bernard (1929) have related an interesting case of a young man who complained of acute abdominal pain and vomiting for which no cause was found on laparotomy. Shortly afterwards acute painful flaccid paralysis developed in the lower limbs, to be followed in a short time by a similar condition in the upper limbs. The right trigeminal and facial nerves then became affected, together with loosening and falling out of the teeth. Meanwhile, jaundice developed, which progressed until death occurred from asphyxiation due to paralysis of the muscles of respiration. At necropsy areas of degeneration were found in the trigeminal, vagal, and hypoglossal nuclei; also in the anterior horn cells of the spinal cord. But although the liver was enlarged, no appreciable changes were found. Lemierre has reported a case of a patient who developed spastic paraplegia during an attack of epidemic jaundice.

Lelong and Bernard (1935) have had under their care during a local epidemic a girl of 14 suffering from infective jaundice. Four weeks after the beginning of her illness polyneuritis developed in the upper and lower limbs, starting distally and spreading upwards. Sero-agglutination tests to leptospirae were negative; nor was the organism found in any of the body fluids. Complete recovery eventually took place. Newman (1942), describing an outbreak of infective hepatitis, noted the occurrence of mild meningitis and neuritic signs in some patients; while Brain (1943) has reported the occurrence of convulsions, hemiplegia, and mild polyneuritic signs which preceded the onset of epidemic jaundice. Out of 170 patients suffering from epidemic infective hepatitis studied by Cameron (1943) in Palestine, only five showed nervous complications. Two developed mental disturbance, which was attributed to hepatic insufficiency. In the other three patients temporary ocular difficulty with accommodation occurred. All made a good recovery. No case of meningitis was seen by that author.

Judging from the various accounts of cases published in the literature and from one's own experience, the prognosis of nervous complications in infective hepatitis seems to be good.

Two Cases of Epidemic Hepatitis with Parenchymatous Disease of the Nervous System

Case 1.—A housewife aged 28 was admitted to the Derbyshire Royal Infirmary on Jan. 28, 1943, complaining of weakness of the arms and legs, together with jaundice. At the time there was an epidemic of jaundice in the town. She had not come into contact with chemicals nor did she use hair-dye or cosmetics. She was accustomed to an ordinary mixed diet and showed no signs of dietetic insufficiency. On the night of Jan. 7 she went to bed with a cold and headache. The next day she complained of numbness and weakness in the fingers, then in the hands and forearms, and shortly afterwards in the feet and legs. A week later jaundice was noticed. The urine was dark in colour; there was no diarrhoea, and no alteration of colour in the stools.

On examination she was a well-nourished woman; cerebraation was good, there was no anaemia, and jaundice was pronounced, being more so on the face, chest, and abdomen than on the lower limbs. The lower edge of the liver was felt three fingerbreadths below the costal margin. The spleen and lymphatic glands were not enlarged. There was a marked symmetrical weakness and wasting of the muscles of the hands, forearms, and to a lesser extent of the arms and shoulder-girdles, together with loss of reflexes. A similar condition, though less pronounced, was present in the lower limbs. A slight degree of objective sensory loss was found over the hands and feet. The functions of the bladder and rectum remained unaffected. Nothing abnormal was found in the other systems. On most evenings for the first three weeks after admission her temperature was raised to between 99 and 101° F. The urine contained bile pigment but no bile salts. About the seventh week a small amount of albumin was present for a short time. The cerebrospinal fluid was examined two days after admission. It was yellow in colour, but showed nothing else abnormal. The blood contained 4 mg. of bilirubin per 100 c.cm. The serum gave negative agglutination tests to *L. icterohaemorrhagiae*. The leucocyte count was never over 9,000 per c.mm. The jaundice cleared up within about six weeks of admission, and a month later the polyneuritis had improved sufficiently for her to leave hospital, and to attend for treatment as an out-patient. Subsequently she made a complete recovery.

Case 2.—An R.A.S.C. driver aged 24 was admitted to hospital in Persia during Feb., 1943, suffering from epidemic hepatitis. He told me that this condition was common in that country. He was discharged to duty eight weeks later. On April 28 he suddenly felt giddy and sick and complained of headache. On readmission to hospital the next day he was feverish and drowsy. A few days later, on waking up, he was unable to move his left upper limb, and his left leg was weak. On examination he was found to have a left hemiplegia of the upper neurone type, together with a left homonymous hemianopia. Nothing abnormal was observed on examination of the rest of his nervous system, nor in his cardiovascular or any other system, with the exception that his blood pressure was only 100/55. Two weeks later the cerebrospinal fluid was normal, although the pressure was not given. The Wassermann reaction in the blood and cerebrospinal fluid was negative. The white cell count in the former was 10,500 per c.mm. Nothing abnormal was ever discovered in the urine. He was invalided home and admitted to the Derbyshire Royal Infirmary last September. He still showed signs of a left hemiplegia with left homonymous hemianopia, although both are now improving.

Discussion

From the paucity of the sporadic reports of nervous complications occurring during infective hepatitis, it can be argued that there may be a possibility of coincidence and that two separate diseases may coexist. On the whole, however, the probability is that the hepatitis and the complications in the nervous system are but two phases of the same disease. It is, of course, well known that certain pathogenic viruses, such as that of poliomyelitis, have a selective action on the nervous tissue. Other viruses, such as those causing measles, chickenpox, mumps, atypical pneumonia, etc., may under certain circumstances infect the nervous system. Now, although definite proof that epidemic hepatitis is caused by a virus infection is still lacking, yet during the last few years evidence that it is so caused has been accumulating. Thus, if this assumption be correct, it is not unreasonable to suppose, or even to expect, that nervous complications could occur in some of those who suffer from epidemic jaundice. Another possibility is that lowered resistance of the liver in infective hepatitis may predispose to infection by the same or a different virus in other parts of the body. During the past

year, for instance, I have seen herpes develop in several patients who were suffering from infective hepatitis.

Judging by the analogy of the affinity of the *Spirochaeta pallida* for nervous tissue even in the early stage of syphilis, it is not unreasonable to assume that the nervous system may also be affected by other spirochaetes, such as *L. ictero-haemorrhagiae*, during the course of its invasion.

If the association of nervous complications with infective hepatitis is kept in mind, it may be found that this combination, especially in cases in which the jaundice is not obvious, is not so uncommon as is at present supposed. Therefore, all cases of so-called "aseptic meningitis" of doubtful aetiology, or polyneuritis without obvious cause, should be carefully scrutinized for the presence of latent jaundice. The association of jaundice with nervous complications may possibly be further exemplified by such a condition as progressive lenticular degeneration (Wilson's disease), in which cirrhosis of the liver and a varying degree of jaundice occur together with involvement of certain parts of the brain, especially the corpus striatum.

Summary and Conclusions

The occurrence of nervous complications in Weil's disease and in epidemic hepatitis has been discussed.

Meningitis is the nervous complication most commonly met with in both diseases.

Disease of the nervous parenchyma, though it does occur, is rare in both infections.

Meningitis occurring in a patient suffering from Weil's disease has been described; also two cases of epidemic hepatitis, in one of which polyneuritis developed. In the other, hemiplegia, presumably due to an encephalitis, occurred within a few weeks of the attack of epidemic hepatitis.

While the possibility of coincidence has been kept in mind, the probability is that the hepatitis and the nervous complications are but two phases of the same disease.

This combination may be found to be not quite so uncommon as was formerly supposed, especially if the presence of latent jaundice is looked for in all cases of infective nervous disease of doubtful aetiology.

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speaking, and the line of approach determined by the wounding agent. The position of the wounded man at the time of injury and the type of missile may, however, modify this appreciably. Buttock wounds, in particular, demonstrate this fact, no less than 10 to 15% involving the peritoneal cavity. With regard to the type of missile, coincident blast is a complicating factor too easily forgotten, as that of the lower lobes of the lung in wounds of the upper abdomen. Gross external with less severe internal damage by mortar wounds is less likely to mislead than the converse with shell or rifle wounds. In all types, however, the through-and-through wound may well be the problem. This, *par excellence*, is the wound to be left alone, especially when of the "gutter" type in subcutaneous tissues. Wounds of the right upper quadrant of the abdomen, involving the liver only, may also fall into the same category.

The Abdomen

Abdominal wounds, in general, are still prognostically the worst, an all-in mortality of 50% being my experience and that of numerous surgical colleagues in the North African campaign. This is high, perhaps too high; but many of the factors determining it are beyond the surgeon's control. The interval between receipt of injury and operation is still the most important, and it is reasonable to suggest that every hour after six is more serious than two before it. Civilian figures for mortality of operations for perforated peptic ulcer provide a close parallel. (In the abdominal injuries dealt with by me in forward areas 10 to 12 hours was the average time between wounding and operation.)

Once the patient reaches the operating centre, however, many problems arise—perhaps more in the five or six days after operation than at the operation itself. Given blood instead of plasma, which is no substitute in cases with mesenteric bleeding, the decision and moment to operate are not, as a rule, difficult to determine. The approach then is in the line of the entry wound, and a buttock wound, for example, should be explored first before proceeding to deal with the intraperitoneal damage complicating it. At times, as a result, laparotomy will be found unnecessary; for abdominal rigidity with these, as with thoracic wounds, may easily be found misleading.

The importance of searching the alimentary canal from stomach to rectum has been stressed by numerous observers, and a patient too ill to stand this is too ill for operation. The exceptions are few enough to ignore. With this proviso, the plan should then be clear, and the task usually involved is exteriorization of the large gut and suture or resection of the small. The higher the lesion, of course, the better the prognosis. Even then, however, coincident injuries to other parts of the body may turn the scale, as it has been the exception rather than the rule to have to deal with a single wound. Too often, in fact, the wounded man provides an operation list in himself.

This raises the interesting subject of abdomino-thoracic wounds, which account for 20% of all wounds of the abdomen. These are more serious on the left than on the right side, but experience has shown that they are not necessarily graver than simple wounds of the abdomen. The abdominal injury in these cases is likely to be high and the bowel may be intact, the liver or spleen being the only viscus damaged. Simultaneous renal injury may occur—one notable case in the series under discussion necessitating nephrectomy, splenectomy, and closure of a large pleural defect, without untoward result. A successful cholecystectomy and duodenal repair has also been done for a right-sided abdomino-thoracic injury.

Post-operative continuous gastric suction and measures designed to prevent ileus repay all the care they demand; but a proportion of disappointing results remain which, at times, seem hard to explain. Why, for example, should pulmonary oedema have proved fatal in gunshot wounds of the spleen where no more than three pints of blood or plasma had been given in the 24 hours after operation? Why too, unless hypovitaminosis is the explanation in an army in the field, should a plastic peritonitis be the cause of death three weeks or more after operation? A typical example occurred in a man requiring resection of four feet of ileum and a colostomy for visceral injuries from a machine-gun, seen within five hours.

SURGICAL PROBLEMS IN FORWARD AREAS

BY

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The scope of surgery, in forward areas has advanced in recent times to more than life-saving measures and heroic ablations, often in vain. In their place there is, it is true, major surgery with a high proportion of amputations; but resuscitation, chemotherapy, and new views on after-treatment have altered the principles and results out of all recognition. Difficulties, of course, remain that experience alone can solve, and when not to operate and when not to evacuate are among the most striking. Evacuation, indeed, controls the whole problem; and familiarity with the mode of transport and distances involved should be an essential part of a forward surgeon's information. Many a successful technical procedure has been ruined by premature evacuation, many a wounded man set back further by a long ambulance ride than improved by the resuscitation not long preceding it. Cases of burns and thoracic and abdominal injuries illustrate this point well.

The surgeon in forward areas has admittedly the easiest task in one respect, for the field is new ground, surgically