In one of these cases the ataxia was thought to be partly vestibular in origin. It would indeed be surprising if the vestibular branch of the eighth nerve escaped entirely when the auditory branch was involved in the degenerative process. This syndrome was regarded by neurologists as a deficiency disease. There was some difference of opinion as to its nature. Lack of vitamin A was considered at one time, but the consensus of opinion was that it was due to shortage of the B complex.

The cases of spastic paraplegia formed a small but sharply defined group. They all came from two camps, and had developed about the same time within a period of six months. They showed gross spasticity, walking on the tips of their toes and unable to put their heels to the ground. Deep reflexes were exaggerated and plantar responses extensor. Cases occurred in men who had suffered less privation than average. One N.C.O. had been in charge of a cookhouse and fed comparatively well. It seemed probable that the cause here was some toxic factor, and neurological opinion favoured the diagnosis of lathyrism.

The cases of peripheral neuritis were not infrequently associated with signs of Group A syndrome, giving a mixed picture. Others, as already stated, were due to diphtheritic infection. A number were left, however, in which a diagnosis of neuritic beriberi seemed justifiable.

Diseases Present

Pulmonary tuberculosis was the most serious disease occurring commonly among them. This had already taken a considerable toll of lives before release. More than 50 cases were received here, showing all stages of the disease. The high incidence is not surprising considering the lowering of resistance by malnutrition, overwork, and intercurrent infection, particularly malaria. The overcrowded living conditions predisposed to the spread of the disease. Most of the cases are now doing well on sanatorium treatment, and it would seem reasonable to assume that the majority of these patients would never have developed the disease had they been leading normal lives. Thus it may be argued that as a group they have a higher basic resistance than a comparable group who have developed the disease in civil life.

The military patients had nearly all had repeated attacks of malaria. Some reported as many as 50 attacks. Though accurate counting becomes difficult after five or six relapses, it is certain that the majority of them had had many attacks. In sharp contrast were the civilians, most of whom had been living in Malaya for years before the war. Many of them had not had a single attack, and few had had more than one or two. This suggests an acquired resistance to the local strains. All Service cases were given a full course of mepacrine, if they had not already had it, and were instructed to take a suppressive dose for a further six weeks. It was felt that this would make freedom from relapse fairly certain during the journey home.

A remarkable feature of our series was the low incidence of dysentery. Our dysentery ward never had more than a dozen cases in it at any time. A considerable number of patients gave a history of proved amoebic dysentery. This had cleared up completely without any specific treatment, and full examination, including sigmoidoscopy, showed no abnormality. This confirmed an impression already gained—that in this disease the soil may be more important than the seed.

A few cases of sprue were seen, all in civilians, and they were our only patients who had suffered from being released. The sudden change-over to a full diet was too much for them and had a disastrous effect. One arrived *in extremis* and died within 24 hours of admission. Two others gave us a great deal of anxiety before they turned the corner. With strict dieting and transfusion they eventually made a good recovery.

Conclusion

To summarize, it may be said that the condition of these patients was, on the whole, better than might have been expected. The greater resilience of youth was seen in the more rapid recovery in Service cases. This was particularly marked in those showing nutritional oedema.

The medical attention these men and women had received from British doctors in the camps calls for the highest praise.

Working usually without x rays or other equipment, they had made accurate diagnoses, which were confirmed when full investigation was possible. Where screening could be done pneumothoraces had been induced and maintained. The full and careful notes which the patients brought with them were of inestimable value to us in carrying on with their treatment. Finally, it may be said that our patients presented a true example of survival of the fittest.

My thanks are due to Col. W. J. Officer, O.B.E., commanding 114 British General Hospital, for his help and criticism.

TREATMENT OF ACUTE OTITIS MEDIA BY DECONGESTION

BY

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The role of ephedrine as a decongestive agent in the nose is well recognized. Its value in the treatment of middle-ear inflammation does not seem to be known.

Some three years ago I first used it to decongest the mucosa "pouting" through perforations in the acutely inflamed tympanic membrane in acute otitis media. The anticipated result was obtained. The mucosa became less turgid, the opening through the membrane was correspondingly enlarged, and drainage was facilitated. Resolution was thereby accelerated. The effect was the same, of course, whether the opening followed myringotomy or rupture of the drum. It may well be that some of the ephedrine solution also found its way into the middle ear itself and exerted its helpful decongestive action on the mucosa lining the cavity and the tympanic end of the Eustachian tube, with consequent improved drainage through the latter. Having enlarged the opening through the drum as described, the process of drainage was further enhanced by the instillation of glycerin drops after the ephedrine. These, of course, are helpful by virtue of their osmotic action. To this combination of ephedrine followed by glycerin the term "decongestion-osmosis" is applied.

The experiment was later tried of instilling the ephedrine solution into the ear in early cases of acute otitis media with an intact, congested, but not yet distended tympanic membrane. The effect was surprising. After a few applications, at four-hourly intervals, the drum became decongested, inflammatory appearances were dispelled, the landmarks became more clearly apparent again, and all evidences of a process towards resolution were demonstrated. Pain disappeared and the patient felt better. In the majority of cases the condition cleared up, being apparently aborted by the treatment. The term "decongestion" is applied to this procedure.

The rationale of the latter cannot readily be fully explained. It is empirical. My assumption is that the reduction in hyperaemia which takes place (obviously) on the outer surface of the drum results in a reflex mucosal decongestion inside the tympanic cavity and adjacent Eustachian tube, with consequent re-establishment of drainage through the latter. The irreversible process of an inflamed middle ear with exudate shut off beyond a blocked Eustachian tube is thus not allowed to develop, and so resolution by the usual sequence of events which takes place in favourable cases of inflammation can proceed unhampered.

Details of Treatment

The solutions used are 2% aqueous solution of ephedrine alkaloid or of ephedrine sulphate for the decongestion and pure glycerin when osmosis is also required.

(a) *Decongestion-osmosis*.—This is indicated in established cases of acute otitis media when there is a perforation or a myringotomy opening which is inadequate owing to inflammatory congestion. Clean discharge from the meatus. Then, with the patient lying on his side with the affected ear uppermost, instil a few drops of the ephedrine solution into the meatus. After ten minutes' interval instil a few drops of glycerin. The sequence should be repeated at least thrice daily until a freely flowing mucopurulent discharge is obtained. This should be achieved within the maximum of a few days. The usual lines of treatment may then be followed, I myself being partial

to the use of guttae argyrol 5% at this stage—either simply instilled or, when all signs of acute inflammation have gone, by the displacement method (Reid, 1939).

(b) *Decongestion*.—This is indicated in early cases of acute otitis media (i.e., within the first 48 hours usually) with an intact but inflamed non-bulging drum. The ephedrine solution only is instilled into the affected ear, in the same way as above described—a few drops being left to act for about five minutes. Repeat at least thrice daily. The case should be reviewed in three days, the patient being told to report earlier if the pain has not gone after the first three treatments.

Both the above procedures can be simply carried out by the patients with the assistance of someone at home, but they must, of course, report within the times specified—or earlier if pain persists.

Results

These are excellent. The logical decongestion-osmosis always proves beneficial when indicated. As regards the decongestion, it can be claimed that the majority of cases of acute otitis media abort with this treatment if it is instituted early enough. Also, no risk would appear to be involved. In no case has mastoiditis developed in consequence of delay in applying other lines of treatment because of decongestion having been employed. With this method of treatment myringotomy is much less often necessary, rapid resolution with an intact tympanic membrane being the rule.

The following figures of myringotomies done in the outpatient department theatre at the Children's Hospital, Birmingham, in 1944 and 1945 show the striking drop in the number of those operations since I introduced decongestion therapy in April, 1945.

	1945	1944
January	98	184
February	94	116
March	110	230
Decongestion therapy introduced		
April	63	147
May	36	137
June	17	155
July	14	78
August	14	73
September	8	48
October	17	92
November	12	89
December	20	66

The figures for January and February of 1946 are 16 and 8 respectively, compared with 98 and 94 for the corresponding months in 1945, when decongestion was not used.

Finally, it should be mentioned that this line of treatment is not advocated in conjunction with chemotherapy, in view of the masking effects of the latter on the middle-ear appearances, the changes in which should be carefully observed. Anyway, the use of chemotherapy should be reserved for only the more virulent forms of acute otitis media.

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A CASE OF INDIGENOUS MALARIA IN NORTHERN IRELAND

BY

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The "ague" has been present in England throughout historic times, but in recent generations it was little heard of until 1917. From September that year onwards until 1919 almost 600 locally contracted cases of malaria occurred in England. The cause of that outbreak was the return of large numbers of infected military personnel from malarious areas abroad, chiefly Greece, Mesopotamia, and Africa, to areas in England where anopheline mosquitoes were prevalent. Cases were scattered over South-East England, especially in coastal areas, and a small epidemic occurred in the Isle of Sheppey, in Kent. This outbreak was described in a report to the Local Government Board (1919).

Cases of endemic malaria were rare between the wars, but Shute (1944) drew attention to the possibility of an increase

of indigenous malaria after the war then in progress was ended and when large numbers of infected Service personnel would be returning from abroad. One such case is here described, and it is considered to be of especial interest as it occurred in Northern Ireland—an area in which malaria does not appear to have been previously recorded.

Case History

The patient was a member of the W.R.N.S., aged 24, who was admitted to a naval auxiliary hospital in Londonderry on July 16, 1945. She gave a history of headache and attacks of shivering and sweating for a week previous to admission. Her temperature, which was 101° F. (38.3° C.) on admission, was normal next day. It rose to 105° F. (40.6° C.) on July 18. The tongue was heavily furred, but no physical signs were found to account for her pyrexia. She was well again on the 19th, but on the 20th her temperature rose to 104° F. (40° C.). The temperature chart was by now typical of tertian malaria, and a blood slide taken that day showed a heavy infection with benign tertian parasites. She responded well to treatment and there were no complications. The spleen was not palpable at any time. Blood counts showed 4,500,000 red cells per c.mm. and a haemoglobin of 90%. The patient had never had any similar illness before, and she had never been outside England and Northern Ireland.

Comment

The chief point of interest in this case lies in trying to trace the place where infection occurred. For the transmission of malaria the necessary factors are: (1) the existence of human carriers of malaria with gametocytes in the peripheral blood; (2) the existence of suitable mosquito vectors; (3) certain further conditions which are not yet fully understood, but which include a suitable temperature for the development of the parasite in the mosquito. Studies of malaria in England and in other temperate climates have shown that, under the conditions there, cases of malaria tend to occur in two main seasonal groups. Primary infections may develop symptoms either in the late summer and early autumn—that is, when conditions for transmission are most suitable—or in the spring months of March, April, May, or even June. It is presumed that this second group of cases have been infected during the previous summer but that the infection has remained latent for a period of from 6 to 9 months. This phenomenon has been discussed by Shute (1939).

The subject of this present case had spent 1944 at Southampton and Newhaven, with occasional brief visits to her home at Braintree, in Essex—an area where malaria is known to occur. In March, 1945, she was drafted to Londonderry and remained there until she took ill, except for a visit to Braintree from April 20 till May 1, on leave. That visit may be left out of consideration as a possible time of infection as the early summer mosquitoes would not yet be hatched out. It remains to decide whether this patient was a latent case, infected in England during the previous summer, or was, in fact, infected in Northern Ireland.

Her work kept her in the naval dockyard by day, while her living quarters were in a Nissen hut, one of eight in the grounds of a large house about a mile north of the centre of the town. Search of the area around these quarters showed that anopheline mosquitoes were breeding in a small pond about 200 yards away. Larvae from this pond were kindly identified for me by the Ministry of Health Malaria Laboratory, Horton Hospital, Epsom, as *A. maculipennis*. This is the most usual British malaria-carrying mosquito, and it was no doubt breeding in other suitable places in the district. Carriers of malaria were numerous in the Londonderry area during the spring and summer of 1945, as during the previous autumn the local Territorial regiment, numbering several hundred strong, had returned home after five years in the Middle East and Italy. Of these men some had been demobilized, while others were home on leave from time to time. Moreover, primary and relapse cases of malaria were almost always under treatment in the hospital from among the many hundreds of naval and Air Force personnel stationed in the area, of whom a large proportion had served in malarious areas abroad during the war.

It must now be considered whether it is likely that the processes of infection of a local anopheline, development of the parasite within it, infection of the patient, and incubation of the disease could have taken place before July 9, which was the date on which the first symptom appeared. The time taken for the parasite to develop in the mosquito is largely governed