Correspondence

R.M.B.F. Christmas Appeal

Sir,—Christmas comes but once a year, and when it comes should bring good cheer. I therefore appeal to your readers to help those who have to look to others for whatever extra comforts may come their way. The beneficiaries of the Royal Medical Benevolent Fund are either aged or infirm practitioners, their wives, widows, or children; and, but for the grace of God, any one of us or our families might be in like need.

I hope subscribers to the Fund will send an extra donation to make this Christmas a little less bleak and grim. I earnestly ask those who are not subscribers to show their sympathy for their less fortunate colleagues, not only by sending Christmas gifts, but by becoming regular supporters of the Fund.

Contributions and subscriptions should be sent to the Secretary of the Royal Medical Benevolent Fund, 1, Balliol House, Manor Fields, Putney, London, S.W.15, and marked "Christmas Gifts."—I am, etc.,

Alfred Webb-Johnson,
President,
Royal Medical Benevolent Fund.

Poliomyelitis

Sir,—I was interested in the letter from Dr. J. M. Alston (Sept. 13, p. 432) calling attention to the evidence suggesting that infection by poliomyelitis virus is common, with paralysis occurring in only a small proportion of those infected. Investigation of another neurotropic virus, that causing louping-ill in sheep, has emphasized to me the importance of factors which in the individual animal allow a potentially neurotropic virus to invade the nervous system from the blood.

Injected directly into the brains of non-immune mice and sheep, louping-ill virus always causes fatal encephalitis. Injected peripherally (intraperitoneally or subcutaneously), it multiplies and reaches the blood, but in only a proportion of animals does it subsequently invade the brain. There is evidence that environmental conditions affecting nutrition determine the proportion of sheep which develop encephalitis. In two experiments carried out in June and July of successive years a group of four and a group of three sheep were inoculated subcutaneously with virus, and none developed encephalitis. Of 27 sheep similarly inoculated during winter and early spring 55% died.

It has been shown that the virulence of louping-ill virus and some other encephalitic viruses can be modified by culture in the developing chick embryo or in tissue culture. Although virus thus modified retains unaltered its ability to produce encephalitis after intracerebral inoculation, it progressively becomes less capable of invading the brain after peripheral inoculation. A strain of louping-ill virus attenuated in this way, so that normally not more than 10% of sheep inoculated subcutaneously developed encephalitis, proved capable of killing nearly 50% of sheep when the inoculations were made during the severe weather of last winter.

These observations call attention to the importance of the mechanism ("the blood–brain barrier") which tends to prevent spread of virus from the blood into the brain. They suggest that in these neurotropic infections one must study not only the virus but the factors which influence this spread in the individual animal under different environmental and nutritional conditions.—I am, etc.,

Frant, Kent. D. G. ff. Edward.

Sir,—I was most interested to read the letter on this subject from Dr. E. C. H. Huddy (Sept. 20, p. 468). While in India in 1944 I was medical officer to an officers' training school where we had 500 British and Indian cadets continuously in training. These cadets messed in six separate messes, which I supervised as regards cleanliness and administration, assisted by a most precise non-medical lieutenant.

We had a sudden outbreak among newly arrived British cadets of poliomyelitis. We had six cases in all, of which two died in iron lungs and four recovered with varying degrees of disability. On the assumption that intestinal infection spread the disease I ordered all eating and drinking utensils to be soaked in chlorine baths for some hours before re-use, and following the implementing of this order the spread of infection ceased. It was significant that the ordinary British Tommy (of whom there were several hundreds in the station), using his own individual eating and drinking utensils, was not affected at all by the outbreak, which was confined to the British cadet messes, where eating and drinking utensils were used communally. The fact that the last two cases occurred 17 days after the commencement of the outbreak suggests infection before the institution of the precautions described.—I am, etc.,

Burton-on-Trent. J. R. Salmond.

Disseminated Sclerosis and Poliomyelitis

Sir,—Your annotation entitled "On the Track of Disseminated Sclerosis" (Sept. 20, p. 460) was of very great interest to me, and I write because I think I can make a useful contribution to the discussion. I am familiar with the experimental work done on swayback in the Peak district under the auspices of the Derbyshire Agricultural Committee, with the help of Dr. Innes. It was thought that the problem had been solved by giving the ewes long before the birth of their lambs salt blocks containing copper to lick. Undoubtedly this treatment considerably reduced the number of cases, but anyone visiting these sheep farms and talking to the shepherds and farmers would soon be convinced that we have still a long way to go.

I think it may be admitted that the element copper is essential to the conduction of nerve energy, and when it is absent in those nerves supplying muscles we get paralysis and changes in the nerve leading to degeneration and demyelination. A copper deficiency in the soil would no doubt predispose to the paralytic condition, but the point that has been overlooked is that copper may be abundantly present, but supplanted in the nerve tissue by some other mineral.

In 1938 I made a determined effort to find the cause of fowl paralysis, and, profiting by the results obtained in swayback, I put great faith in copper. For several years the mortality from this disease had been very high, and the complaint was invariably fatal, although the bird might live for a few weeks. I determined to try one mineral after another in dilute solution injected daily into the crop. The bird was artificially fed as well. I began with a typical and unmistakable case of fowl paralysis. When copper solution had been injected for a fortnight without the slightest benefit, my assistant said: "Don't you think, sir, we have tried copper long enough? May I go on to the next mineral?" I explained I was loath to abandon copper because it was the substance from which I expected most benefit. I said: "Go on to No. 2, but continue with copper as well." The next on the list was cobalt, and the third was nickel, neither of which we had in the laboratory, but we had manganese, which we used. The bird had not had this combination of copper and manganese for more than two days before a remarkable effect was produced. It could stand up and peck. In a few days it had the appearance of a healthy bird. Its comb became red and it started to lay, giving me an egg nearly every day. We had cured fowl paralysis, but my poultry friends who had watched the experiment could not believe it. They reasoned: "As fowl paralysis is an incurable disease there must have been a mistake in the diagnosis." My reply was: "If my theory is correct I can bring back the fowl paralysis." Accordingly I administered a solution of zinc sulphate, and in two days the bird was again on its side, unable to stand. I did not attempt a second cure, but killed the bird and sent the corpse to Weybridge Research Station for a report. The answer soon came: "Advanced fowl paralysis." Since that time I have treated 28 cases, and nearly half of the number have been completely cured. In half of the failures the post-mortem revealed that some other disease was present, and in the remainder probably treatment had not been started early enough. Copper by itself was ineffectual and so was manganese. It required the combination. Probably the manganese acts as a catalyst.

The conclusion I reached was that fowl paralysis is a disease in which copper in some of the nerves is displaced by another metal, usually zinc, derived from the galvanized poultry utensils so commonly used, or from lead which is present in the limestone used as a powder in the mash, and as grit. Referring

once more to swayback, I have ascertained that the soil in the Peak district where swayback is prevalent is heavily charged with lead. Is it possible that the present outbreak of infantile paralysis has a copper displacement factor in its aetiology? One mineral has been used to an extraordinary extent during the war years and since. I refer to tin. Aluminium cooking vessels were difficult to get during the war years, but since the shops have been flooded with them. Might I suggest that in each case of infantile paralysis the urine should be tested for traces of tin and aluminium?—I am, etc.,

Kenwood, Chesterfield. J. A. Goodfellow.

Miliary Appearances in the Lungs in Mitral Stenosis

Sir,—I was most interested to read Dr. T. E. Gumpert's article (Sept. 27, p. 488) on "Miliary Appearances in the Lungs in Mitral Stenosis." In the past year I have noted three such cases. Two of these cases had had mitral stenosis of long standing. The miliary appearances in the lungs were seen shortly before death from congestive cardiac failure.

The third case was rather different. The patient, a woman aged 36 years, was admitted to hospital in an extremely collapsed condition with cyanosis, orthopnoea, marked tachycardia, pyrexia, venous distension in the neck, and widespread adventitious sounds in the lungs. She had a previous history of rheumatic fever. *X*-ray examination of the chest showed widespread miliary appearances very similar to those seen in miliary tuberculosis. Her pyrexia was intermittent in type and proved resistant to penicillin and sulphathiazole in full doses. She had no sputum.

After four days she was put on full doses of salicylate. Her temperature subsided rapidly and her tachycardia became less marked. As it did, a well-marked mitral stenosis was discovered. She remained apyrexial though the tachycardia tended to persist. *X*-ray examination of her chest 14 days after the first film showed the lung fields almost clear. Although she remained apyrexial her convalescence was protracted, with marked tachycardia on the least exertion. She had no peripheral manifestations of rheumatic fever.

I should very much like to hear Dr. Gumpert's views on this case.—I am, etc.,

Dublin. Haldane G. Nelson.

Sir,—I read Dr. T. E. Gumpert's interesting article (Sept. 27, p. 488) on "Miliary Appearances in the Lungs in Mitral Stenosis," but do not agree with the pathological interpretation or the radiological findings. The haemosiderin-laden histiocytes from the alveoli are motile and travel to the intralobular collections of lymphoid tissue. Here fibrous tissue reaction results in the formation of pseudo-tubercles, at first microscopic, but later increase in the amount and maturity of the fibrous tissue gives the appearances of nodulations apparent in the photograph. The rosette-like appearance he describes in the magnified photograph I should interpret as hyaline degeneration in the centre of the fibrous nodule, which is more opaque to the *x* rays than the young fibrous tissue at its periphery. The pathology is in fact extra-acinar. The diffuse haziness in the later photograph suggests interstitial fibrosis—i.e., in the interalveolar and interlobular lymphatics—seen in Whitehaven in the lungs of haematite miners from Egremont. I think if a photograph were taken now of this patient it would show the same pattern as that seen in 1946.—I am, etc.,

Belfast. Muriel O'Doherty.

Early Diagnosis of Tuberculous Meningitis

Sir,—Dr. M. C. Wilkinson's call to the profession (Sept. 27, p. 507) for early diagnosis of hitherto incurable tuberculous conditions will be welcomed by all tuberculosis workers. While there is little one could add to Dr. Wilkinson's observations, I would like to call atttention to a recent work which had purely academic significance at the time of its publication, namely, that by biopsy typical miliary tubercles may be demonstrated in the bone marrow in some cases, without any changes in the C.S.F. and before radiological evidence of pulmonary invasion.—I am, etc.,

Colchester. Francis Kellerman.

Fungus Poisoning

Sir,—The article by Mr. J. Ramsbottom (Aug. 23, p. 304), the annotation (p. 302), and the letters by Dr. C. Allan Birch (Aug. 30, p. 348) and Dr. A. T. Todd (Sept. 6, p. 395) deal with mushroom poisoning, making reference to *Amanita phalloides*. I have had occasion to observe two families poisoned by this mushroom, the full details of which I published in the *Revista Clínica Española* (1945, **17**, 211). Pathologically the most striking things to be found were: intense fatty degeneration in the liver with bands of total necrosis and haemorrhagic infiltration; in the suprarenal capsules turbid degeneration in the cellules of the second and third zones of the cortex.

In diagnosis the late onset of the symptoms, almost without exception ten to twelve hours after ingestion, is of great value. The treatment of Limousin (fresh rabbit's stomach and brains) broke down in my cases, as did the intravenous glucose in large doses of Binet, and the draughts of a saturated salt solution of Le Calve, and likewise all the cardiac tonics. I was not able to give the "antiphalloid" serum treatment of Dujarric de la Rivière. My impression is that many of the cases reported as successful through various treatments are caused by other mushrooms, different from the *Amanita phalloides*, the intoxication of which I consider to be almost always fatal. I think transfusions of plasma ought to be tried.

Of the nine individuals under my observation eight died; the only survivor was a woman who had been pregnant for five months, and whose pregnancy went on successfully to its conclusion. This I believe was due to the fact that vomiting began before the others (in shortly under eight hours). In any case it would be interesting to investigate whether pregnancy embraces a state refractory to the amanita toxin, and why.—I am, etc.,

Soria, Spain. J. Calvo Melendro.

Treatment of Injuries following Insertion of Gastroscope

Sir,—Drs. C. M. Fletcher and F. Avery Jones (Sept. 29, 1945, p. 421) discussed the risks involved in using the flexible gastroscope. This article brings to my mind the treatment of oesphageal injuries from the gastroscope practised by me as well as the treatment of inflammatory lesions in the neck region. From 1931 until the beginning of the war I have done many gastroscopies. My results have been very good in the diagnosis of diseases of the stomach. Naturally it is necessary to make an *x*-ray examination beforehand to be certain there are no pathological changes in the oesophagus, stomach, or neighbouring organs. Marked kypho-scoliosis, a displaced vertebra, or marked aortic sclerosis may make an examination through the oesophagus very dangerous or impossible. One must also bear in mind that the oesophagus of older people is less elastic and more vulnerable to injury. Areas in which generally are found diverticula have a thinner pile of muscles and are easily injured. Introduction of the gastroscope must be done without pressure. Even so, injuries to the oesophagus and hypopharynx have occurred by experienced physicians, as borne out in the report by Fletcher and Jones.

In the treatment of such accidents and as well as dangerous anginal septic states—e.g., post-tonsillectomy and so on—the following method used by me has given excellent results. According to the old surgical principle of putting at rest inflamed areas—as, for example, suppuration in the extremity—I apply a padded wire splint to the patient extending over the head, neck, and back, securing it with bandages at the thorax and head and the latter about the forehead, thus preventing any side-to-side or forward-and-backward motion. In this manner all the muscles of the neck and upper parts of the oesophagus will be put at rest. Forbidding the patient from speaking and giving him only liquid food, one has produced the best conditions for localizing the inflammation and its healing. In all my cases within a few days the temperature fell, the inflammation disappeared, and the patients recovered.

This treatment is not only suitable for injuries following the gastroscope introduction but excellent results by me have also been obtained for all other inflammations in the neck region. The following case was very characteristic: A tonsillectomy performed by a specialist was followed by a septic state lasting five weeks, with fever between 38° and 39.5° C. (100.4° and 103.2° F.), greatly swollen glands of the neck, and a bad general state of