Epidemic Collapse

SIR,—Referring to the mysterious illness among pupils at a girls' school reported in the *B.M.J.* for 16 October (p. 950) the fact that laboratory investigations so far have proved negative prompts me to relate an incident with which I was concerned at a mixed secondary modern school some years ago.

I was called to this school one morning by the head teacher as a number of pupils had been taken ill. When I arrived I found that some of the children had already been sent home not feeling well. Others were lying in classrooms and corridors exhibiting various stages of "collapse." Among those I examined some had tremors, some were overbreathing, and many complained of headache, or pain in the chest and abdomen. Otherwise the results of clinical examination for any gross abnormality were negative. All recovered spontaneously sooner or later when left alone.

The curious fact was that none of the boys seemed to be affected, which lent support to the opinion I formed at the time that this incident was in the nature of a mass hysterical manifestation following the sight of the first casualty that morning (probably a normal fainting attack) during assembly.—I am, etc.,

Dover, Kent.

C. F. LYNCH.

Hysteria and "Royal Free Disease"

SIR,—In reporting that psychiatrists consider the "mystery illness" among school-girls in Blackburn to be attributable to mass hysteria, Dr. Alfred Byrne, medical correspondent of the *Sunday Times*, remarks "Some of the nurses involved in the 'encephalitis' outbreak at the Royal Free Hospital 10 years ago ended up with this." The Medical Correspondent of the *Daily Telegraph* (18 October) makes a similar observation. These tend to convey the impression that hysteria was the predominant feature of that outbreak. While it is true that a "functional overlay" was a not uncommon feature in many cases, the problem of "Royal Free disease" can be brought into perspective only if the following facts are noted:

(1) The outbreak at the Royal Free Hospital was not an isolated incident. A large epidemic of the same disease occurred in Iceland in 1950, and permanent sequelae, both neurological and psychological, were recorded. Outbreaks were also reported from Adelaide (1951), Coventry (1954), New York State (1954), Durham (1955), and Athens (1958).

(2) During the summer of 1955, and throughout 1956, 1957, and 1958, many cases of the disease were treated in the infectious diseases department of the Royal Free Hospital at Hampstead. Indeed, the condition has been endemic in North London since 1955 (although it may be significant that no cases were seen during the very cold winter of 1962-3).

(3) Since October 1964 some 90 cases have been seen in a geographically circumscribed area in North London, and some of these have been severe enough to require hospital treatment.

(4) Many patients, both in 1955 and at the present time, show clinical evidence of severe involvement of the central nervous system with no hysterical overlay.

(5) Most cases have a low-grade pyrexia, cervical lymphadenopathy, scattered myalgic foci, and abnormal electromyograms and/or electroencephalograms. Dr. Ruth Harris and Dr. Jean Kennedy, of the Department of Electroencephalography at the Royal Free Hospital, have demonstrated abnormal encephalograms in 38 patients even after a considerable period from the onset of the illness.

In presenting a paper on the disease at a recent meeting of the World Union for Social and Prophylactic Medicine at Mittendorf, Austria, I was interested to find that a German physician, Dr. Frisch, of Freiburg, was thoroughly familiar with the condition and was able to show photographs illustrating severe neurological damage. It would be singularly unfortunate if the impression was created that this still prevalent and unexplained condition were thought to be the result of mass hysteria.—I am, etc.,

A. MELVIN RAMSAY.

Infectious Diseases Department,
Royal Free Hospital,
London N.10.

"Winter Vomiting" and Collapse

SIR,—On 11 October our daughter aged 11 woke from sleep with severe vomiting and continued retching every twenty minutes for the next three hours. The following day she had a temperature of 99° F. (37.2° C.), slight anorexia, and dizziness. She returned to school and was well the remainder of the week until 17 October, when she complained of dizziness in the afternoon. She was well next morning but sent home from school with dizziness at midday. On 18 October she was slightly dizzy and remained at home. At 8.30 p.m. she complained of severe high epigastric pain going through to the back, and whilst I helped her upstairs she collapsed, was pulseless, sweating, and ashen. She remained unconscious possibly half a minute, her pulse was thready for the next two hours, and checked half-hourly remaining at a rate of 86. Her temperature was subnormal all night. She had acute high epigastric tenderness on palpation, no rebound, no guarding; rectal examination was negative, no nausea or vomiting. She slept fitfully with bouts of upper abdominal colic. By morning her temperature was 99° F. (37.2° C.) and pulse remained relatively slow at 84.

A consultant surgeon and paediatrician were called early morning and after discussion it was felt that this acute episode was possibly "winter vomiting." Her blood count was normal.

The epigastric tenderness persisted for several days and her general malaise gradually lessened over the next week. There was no nausea or vomiting during this time, or signs of meningeal irritation. The acute collapse presented as some abdominal emergency, but careful observation over a period of hours did not give clinical signs to substantiate this.

In retrospect, while conducting surgery on 7 October, I suddenly felt extremely ill, sweating, and collapse, and dizziness with nausea. Fortunately, I managed to telephone my partner and recall fumbling to replace the receiver, before transient loss of consciousness. I was taken home somewhat ashen in colour and recovered sufficiently in early evening to see my patients. I had Stemetil

(prochlorperazine) to relieve the dizziness and nausea.

I trust that this record of personal observation may indicate the acute and dramatic onset which this syndrome may present.—I am, etc.,

Leamington Spa,
Warwickshire.

M. DOREEN BULL.

Treatment of Lung Cancer

SIR,—Drs. H. A. Hartley and B. S. Tate (2 October, p. 822) have pointed out that a patient may seek medical advice and the correct diagnosis may be made, but the treatment of an early cancer lesion may be delayed because of the delay at hospital level. This letter repeats what has already been described in another scientific journal, for cancer at a different site.¹

It is possible that no efforts can be made to improve the treatment of the early lesion because of economic factors. It may be that the cure for cancer, which would add another 100,000 elderly citizens to a population already competing for housing, etc., could be a national economic disaster. If this is not the reason, then there is no excuse for treating patients with cancer inefficiently, and, as Drs. Hartley and Tate reported, to wait three months after diagnosis of cancer can at best be described as inefficient. No one could describe it as economic. Steps should be taken to investigate the causes of delay. How often is it due to inadequately trained personnel, how often to shortage of beds, how often to incomplete investigation?

The general public, who are not fools to be easily hoodwinked, are beginning to ask why is there delay in getting treatment when cancer propaganda preaches that early treatment may mean a cure?

The waiting in out-patients has been investigated by organization and methods teams, but at best this delay was a social inconvenience. The delay and misdiagnosis in the treatment of cancer may cost life. Both the patients and their doctors are well aware of hospital delay before treatment. Are those responsible for hospital policy equally aware of this?—I am, etc.,

London S.W.1.

DAVID WALLACE.

REFERENCE

- ¹ Wallace, D. M., and Harris, D. L., *Lancet*, 1965, 2, 332.

Sequelae of Thorotrast

SIR,—Your leading article (2 October, p. 771) and the discussion of the follow up of 2,377 patients by da Silva's group, in whom Thorotrast was administered between 1930 and 1952,¹ reminds me forcibly of the importance of a controlling body for the marketing of drugs.

In 1930-2 I carried out investigations²⁻⁴ on a large number of rabbits and rats with the preparation Heyden 1073a (which was marketed later as Thorotrast). These investigations showed that Thorotrast was stored in the reticulo-endothelial cells mainly of the liver, spleen, bone-marrow, and brain-covering membranes; the organs, especially the liver, displayed fibrosis similar to atrophic cirrhosis; phagocytosis decreased 6 to 12 months after administration from the normal 38% to 4.2%; capillaries and small vessels

were blocked by the Thorotrast particles (size about 20 $\mu\mu$) in the reticulo-endothelial cells, leading often to thrombosis. Although no changes could be found in the peripheral blood during the observation period of 18 to 24 months, the probability of radiation damage to the bone-marrow and to other storage organs was emphasized as the γ -irradiation for the usual 100 ml. Thorotrast amounted to 1.24 $\mu\text{g.}$ of radium (near the toxic dose), persisting for an unlimited period. At that time a warning was sounded against the rise of Thorotrast in man because of the limited diagnostic value achieved at a very high risk.

Indeed, experimental carcinogenesis was reported a few years later,^{5,6} and when Northfield and Russell⁷ published five cases of cerebral thrombosis after arteriography the *Lancet*⁸ and you, Sir,⁹ brought critical annotations about the use of Thorotrast. Subsequently I expressed my amazement that Thorotrast was still used for diagnostic purposes in man and repeated my previous warning.¹⁰

I am now bewildered to see that it had such a widespread use in Portugal until recently, and that the conclusion of the Portuguese workers in 1965 is that Thorotrast should never be administered to patients with a life expectation of more than two years. Surely even in therapy our first principle should be *nil nocere*?—I am, etc.,

London W.1.

Z. A. LEITNER.

REFERENCES

- da Silva Horta, J., Abbott, J. D., da Motta, L. C., and Roriz, M. L., *Lancet*, 1965, 2, 201.
- Anders, H. E., and Leitner, Z. A., *Klin. Wschr.*, 1932, 11, 1097.
- Bucky, G., and Leitner, Z. A., *Fortschr. Röntgenstr.*, 1932, 45, 561.
- Leitner, Z. A., *ibid.*, 1932, 46, 47.
- Roussy, G., Oberling, C., and Guérin, M., *Bull. Acad. Méd. (Paris)*, 1934, 112, 809.
- Selbie, F. R., *Lancet*, 1936, 2, 847.
- Northfield, D. W. C., and Russell, D. S., *ibid.*, 1937, 1, 377.
- Lancet*, 1938, 1, 36.
- Brit. med. J.*, 1938, 1, 903.
- Leitner, Z. A., *Lancet*, 1938, 1, 228.

Congenital Postural Scoliosis

SIR,—Sir Denis Browne's letter (16 October, p. 945) refuting mine (9 October, p. 877) demands an answer. He complains that Mr. Walker extracted a photograph from one of Sir Denis's articles, but this, in fact, was taken from the general teaching collection at the Hospital for Sick Children. Moreover, Sir Denis was present when the investigation was proposed and readily agreed to it. When the work was completed the resulting paper was sent to him with the suggestion that his name should appear upon it as senior author. This was intended as an act of courtesy to Sir Denis, who, being a retired member of the staff, was no longer in charge of these patients—indeed, many were by this time under my care.

Sir Denis, in referring to his "highly idiosyncratic notes," now raises a point of some importance, for he implies that no one but he could interpret them accurately. It is of the greatest importance that those dealing with children should recognize that much of what we do must inevitably be judged by those who follow us, and this applies particularly to children with orthopaedic ailments.

Lastly, those children diagnosed early (under 6 months) do not, as it is asserted,

have a significantly worse prognosis than those over the age of 6 months. The incidence of progression is not significantly different, being 19% and 15% respectively. To be sure Sir Denis's hypothesis would be supported only if the ratio was reversed and showed a significant contrast in a larger number of patients. Furthermore, in untreated patients no such feature emerges.

There is no criticism of our findings in untreated patients, so it must be assumed that they are accepted. This is fortunate, for it enables me to reply to his request for "information on how the decision to make no attempt to help a child in danger of disastrous crippling is conveyed to the parents and the referring practitioner." Can Sir Denis deny that a baby with idiopathic scoliosis of infancy has an 80% to 90% prospect of spontaneous recovery? Of those who progress only about half will deteriorate to a degree that requires further treatment, the remainder developing a mild, well-compensated scoliosis. If he cannot deny it could he not simply tell the parents and the family doctor this, adding that there is as yet no form of treatment of proved efficacy capable of influencing this pattern of development during infancy?

It is distasteful to enter into public dispute with a former colleague who has made many notable contributions to other branches of the surgery of childhood. Indeed, had my attention not been drawn to this paper by Sir Denis himself, who challenged me to make a comment in your columns, I would have remained silent.—I am, etc.,

G. C. LLOYD-ROBERTS.

Hospital for Sick Children,
London W.C.1.

Sonne Dysentery

SIR,—In your leading article on Sonne dysentery (16 October, p. 893) you point out that "To have any effect on the spread of infection treatment must be started as soon as possible, because it is the early stages of the disease that appear to be the most infectious. But the single sporadic case may be difficult to diagnose without laboratory examination, which takes time." It is quite true that to isolate and identify the causative organism by conventional cultural means takes from 18 to 48 hours or longer. However, I should like to draw your attention to a paper by Mr. G. V. Heimer and myself¹ entitled "Rapid Diagnosis of Sonne Dysentery by Means of Immunofluorescence." In this paper we described a fluorescent antibody technique for the detection of *Shigella sonnei* in specimens of faeces from patients with acute diarrhoea and compared it with a cultural method. The two procedures agreed in 95.6% of 388 specimens examined. Given the necessary reagents and optical equipment, the technique described is extremely simple to perform and enables a provisional report to be telephoned within one hour of a specimen arriving in the laboratory. We now use the method routinely, and should be pleased to demonstrate it to anyone interested.

Thirty thousand notified cases of Sonne dysentery per annum (and as you rightly say "this represents only a fraction of the total number of infections with *Shigella sonnei* in the community, for many cases go unrecog-

nized") is a national disgrace for a civilized country such as ours. If rapid diagnosis is important in controlling this disease, the time taken for laboratory examination need no longer be a reason for administrative inactivity.—I am, etc.,

C. E. D. TAYLOR.

Diagnostic Reference Laboratory,
Central Public Health Laboratory,
London N.W.9.

REFERENCE

- Taylor, C. E. D., and Heimer, G. V., *Brit. med. J.*, 1964, 2, 165.

SIR,—The valuable contribution by Dr. P. J. Moorhead and Dr. H. E. Parry (16 October, p. 913) on the effect of different antibiotics in Sonne dysentery illuminates the varying patterns of antibiotic resistance with respect to time and place. In this area strains of *Shigella sonnei* have been so frequently streptomycin-resistant that routine use of streptomycin has been largely discontinued. For the past three months 95% of strains isolated from an epidemic in North Wiltshire have been resistant to streptomycin, minimum antibacterial concentrations of representatives being more than 50 $\mu\text{g./ml.}$ Resistance patterns are shown below.

	Strains of <i>Shigella sonnei</i>	Resistant to		
		Streptomycin	Tetracycline	Neomycin
Liverpool :				
1960	120	0		
1963	157	26%		
1964 (8 months)	115	8%		
Salisbury :				
1963–July 1965	69	42%	20%	0
Aug.–Oct. 1965	140	95%	8%	0

The indifferent results of neomycin in terms of bacteriological cure where strains are sensitive *in vitro* is puzzling. We advocate neomycin because clinical symptoms respond to it, because we believe that it stops spread of infection from child to child within a family during the acute stages, because it is almost entirely free from undesirable side-effects, and because it is not excessively expensive.—We are, etc.,

Marlborough, Wilts.

F. D. F. STEEDE.

Public Health Laboratory,
General Infirmary,
Salisbury, Wilts.

P. J. WORMALD.

Diathermy Burn

SIR,—As the Secretary of the Medical Defence Union, Dr. P. H. Addison, referred to me in his letter (23 October, p. 1001), perhaps I might be allowed to explain the position a little more fully.

Before the last war my attention was forcibly drawn to the dangers of diathermy burns from the active electrode when I saw a surgeon accidentally touch a needle electrode during the course of a thoracic operation. The foot switch had not been released and the needle sparked through his glove causing a deep punctured burn of his finger. This apparently trivial injury culminated in the tragic death of the surgeon, and in order to avoid further accidents I suggested that electrodes when not in use should be slipped into a thick red rubber sheath clipped to a towel. This idea was taken up fairly generally and sheaths made of rubber or bakelite were