present should constantly be minimal. Is it indeed certain that new pus does not form after drilling of a bone? With regard to dosage of penicillin: That advocated by Great Ormond Street does seem to be unnecessarily low. Presumably this is because the series was begun at a time when there was a dearth of the drug, and the dosage was continued because it was found to be adequate. It is a fact that immediate improvement occurred (i.e., within 24 hours) in the majority of the cases. Penicillin must obviously be given to infants by intermittent injection, and the fewer these are the better, provided that an adequate concentration be obtained and the best possible results, for trauma undoubtedly upsets infants and makes handling of them more difficult as regards feeding, etc.

Mobilization does not really form a large part in the treatment recommended. It is rather a suggestion that complete immobilization is unnecessary, and that simple methods are adequate in the acute stage. Certainly there would be little weight-bearing in the infant age group, and it is probable that the risk of pathological fracture is less than the dangers and difficulties involved in plaster immobilization in infants. With regard to positive blood cultures: this may be rather a false percentage, for no doubt some difficulties were experienced in obtaining some or sufficient blood in all cases. This point is not made clear in the article.

It would seem reasonable to accept, with reservations, the principles both of the Great Ormond Street and the Oxford series. It might be wise to use the Oxford method in older patients with severe infections, while the Great Ormond Street technique seems certainly to be adequate in the less severe cases, and in all infants in whom operations should not be undertaken too lightly, and in whom the difficulties of feeding, hydration, and so on have such an important bearing on the ultimate recovery. A dosage scheme dependent upon body weight would appear to be rational and desirable. Such a scheme has been started by Great Ormond Street in this series, and, though it has not yet attained general acceptance, it would seem to be more reasonable than a mere routine dosage for any patient of any weight and any age.—I am, etc.,

London, W.1.

IAN P. TODD.

Myiasis of Palpebral Conjunctiva

SIR,—I was interested to read Dr. T. E. M. Wardill's letter (May 3, p. 615) regarding myiasis of the palpebral conjunctiva. The following case came to my notice recently and may be of interest.

CASE REPORT

A soldier aged 20 reported sick on the evening of May 27 with an inflamed eye of a few hours' duration. He told the medical orderly that he could feel something moving about under the lid, and as the orderly could see something moving on the conjunctival surface he informed me.

On examination an acute conjunctivitis of the affected eye was evident, and on evertng the lids, to my surprise, a number of very tiny maggots were just visible. With characteristic dislike of light they wriggled away into the conjunctival fornices, but eventually half a dozen maggots were removed by wiping the palpebral conjunctiva with cotton-wool. One maggot was examined on the point of a needle, and its form was then clearly discernible. On questioning the man he stated that a fly had buzzed into his eye at 10 o'clock that morning, and the eye had become inflamed in the afternoon.

Following removal of the maggots his eye became more comfortable and the following day was quite normal.

The predominant fly around here is *Musca domestica*, but I am unable to say whether this might be the culprit or not.

I am indebted to Lieut.-Col. D. B. Seymour-Price, R.A.M.C., for permission to publish this case report.—I am, etc.,

M.E.L.F.

P. DRANSFIELD,
Lieutenant, R.A.M.C.

Acute Non-specific Diarrhoea and Dysentery

SIR,—I read the article by Dr. G. R. Kershaw on the above subject (May 24, p. 717) with pleasant anticipation, hoping to gain some new light on this difficult subject. I was disappointed to find that he appears to have drawn his conclusions after a few years' clinical observation, controlled by only a few full pathological investigations, and with an incomplete knowledge of the extensive literature already published. I have been investigating these cases at sea, as occasion offered, over the last twenty years. In most epidemics I have plated out the faeces, picked off non-lactose-fermenters, and put them through the appropriate sugar media, and tested their agglutination with

standard dysentery and salmonella antisera. I have never identified any pathogen from these cases.

In my paper in the *Lancet* (1938) reviewing this subject the question of chills, cold drinks, etc., was considered, but, after careful investigation, was not thought to be the whole answer, and no pathogen was found. My bacterial results were checked by the late Prof. Eyre, of Guy's Hospital. Drinking water was excluded after a research on the *Bact. coli* content of water in ships' tanks extending over a period of 18 months (Royds Jones, 1936). I note that Dr. Kershaw also exonerates water (after two analyses). Knowledge gained during the war when carrying troops in convoy allowed us to go a stage further (Royds Jones, 1943) and exclude food, water, and a food handler "carrier." It was due to this last letter that I was asked to meet the Director-General of Hygiene at the War Office, who kindly supplied me with a large quantity of media for an extensive research. Unfortunately owing to the progress of the war our route was changed, and we never again got these large epidemics in which 400 to 600 men were affected in one night, and I have not been able to proceed further. Incidentally, the majority of men affected were those sleeping in mess decks, both in hammocks and on the deck, and not those sleeping on open decks, where chilling would have been greatest, although some patients came from each.

My tentative suggestions at the time were either (1) a virus as the cause, or (2) a saprophyte becoming pathogenic at this temperature, for which there is some experimental evidence—Kligler, I. J. (1936), and Robertson and Weld (1932). I discussed the virus theory with a few first-class virus workers, and they at that time were inclined to discredit the suggestion. However, both American and Canadian workers had published papers suggesting a virus as the cause. Now British authorities are more inclined to favour this theory, as witness several articles published recently and your own annotation (Feb. 1, p. 187) about six months ago. I regret that I cannot give exact references to the recent literature, as I am now at sea.—I am, etc.,

Banbury, Oxford.

H. M. ROYDS JONES.

REFERENCES

- Kligler, I. J. (1936). *Trans. roy. Soc. trop. Med. Hyg.*, **29**, 531.
Robertson, E. C., and Weld, C. B. (1932). *Proc. Soc. exp. Biol. N.Y.*, **30**, 33.
Royds Jones, H. M. (1936). *J. Path. Bact.*, **42**, 605.
— (1938). *Lancet*, **1**, 1407.
— (1943). *Ibid.*, **1**, 538.

Primary Malaria in London

SIR,—The article by Drs. C. Blaxland Levick and M. E. MacGregor (May 31, p. 764) on "Primary Malaria in London Children" prompts me to write and bring to your notice a further case of this illness in an adult which I encountered during the week of publication of the original article.

The patient was a woman, aged 36, residing in Kensington and a Foreign Office Civil Servant employed at Eastcote. I saw her at home first on Thursday, May 22, 1947, when she was complaining of nausea with frequent vomiting, frontal headache, and lacrimation, back pain, and sweating. It was her belief that she had some form of "gastric flu." Her temperature was 101.4° F. (38.5° C.). The following day she was little better, and her temperature had dropped to 99° F. (37.2° C.).

On Saturday, May 24, she was moved to friends in Hampstead who were able to look after her better. She was feeling very much better, apyrexial, and thought she had recovered. Sunday evening, however, she developed a recurrence of her symptoms, which were now very much more definite in character and followed the typical stages of malaria, the temperature rising to 102.4° F. (39.1° C.).

Blood slides which were taken the following day when she was apyrexial proved negative; but those taken next day in the middle of a rigor were kindly examined for me by Dr. H. O. Hughes at the Middlesex Hospital, who reports that the films showed scanty trophozoite ring and amoeboid forms, and gametocytes of *Plasmodium vivax* (benign tertian malaria). She has since been given mepracine and remained free of symptoms.

Her history is interesting. Apart from travelling to and from her work daily she had not been out of London since last Christmas. She was born in India and resided in Calcutta till 1937, when she came to this country, and never had any form of antimalarial therapy given her at any time. The only illness in the past had been German measles, and 3-day fever when she was a schoolgirl aged 12—that is, 24 years ago, and a most unlikely factor in this illness. Her only contact with malaria

was this March, when a friend staying with her had a relapse in her home.

I hope that this case will help further in stressing the fact that primary malaria can be a factor in diagnosis in this country at the present time, where malarial carriers are more numerous and the *Anopheles maculipennis* known to be present.—I am, etc.,

London, W.11.

DAVID A. FERMONT.

Congenital Hepatic-duct Malformation

SIR,—The case of congenital hepatic-duct malformation recorded by Dr. Frank Riggall (June 7, p. 824) emphasizes the fact that children born without bile ducts, or with the bile ducts so malformed that they do not communicate with the intestine, survive much longer than one would expect. In Riggall's case the child lived for ten months, and in a large series of these cases reviewed by Stolkind¹ there was one where the child survived for 15 months. The question of operation is worth while considering in cases of atresia, for it may be found that sufficient of the common duct is present to allow of anastomosis to the duodenum. In these cases jaundice is usually present at birth or shortly afterwards, but according to Ladd and Gross² 2-3 weeks may elapse before the icteric tinge appears, "whereas the stools are always clay coloured or white from birth." Further, they state that if operation is delayed for 4-6 weeks from the date of birth there is little chance of error in differential diagnosis.

Anastomosis of the duct to the duodenum is carried out over a small piece of tubing—somewhat after the method of Sir James Walton³—and silk is used for suturing. Vitamin K and ox bile salts are administered beforehand in the hope of diminishing oozing.

Ladd and Gross emphasize the danger of post-operative disruption of the wound, and advise the use of silk and silkworm gut for stitching the abdominal wall. In 45 of their cases nine patients were found to have a patent hepatic or common bile duct connected with the intrahepatic ductal system but not with the duodenum; 6 out of 9 cases survived the operation and were in excellent health 12, 8, 7, 5, 4, and 3 years respectively after operation.

Dr. Frank Riggall is to be congratulated on taking the trouble of publishing his interesting case in the *British Medical Journal*.—I am, etc.,

London, W.1.

MICHAEL J. SMYTH.

REFERENCES

- ¹ *Brit. J. Child. Dis.*, 1939, **36**, 115.
- ² *Abdominal Surgery of Infancy and Childhood*, Saunders, Philadelphia and London, 1941.
- ³ *A Textbook of the Surgical Dyspepsias*, p. 571. London, 1923.

E.C.T.

SIR,—Your correspondent who explains (June 14, p. 858) his reactions to E.C.T. is to be highly congratulated for the enlightened description of his experience, and his article should do much towards alleviating the acrimony at present being expressed on the subject. His important contribution lies in the cognizance that the treatment produced a psychosomatic reaction which he himself "identified as the physical reactions to fear," as it is likely that it is through such identity that he escaped from his troubles. His "memory images" are obviously symbolic expression, which would have been better to have undergone elucidation at the time.

I hope I conflict with neither physicist nor psychiatrist when I understand that E.C.T. in this particular case acted by bringing wholly repressed material to a nearer level to conscious appreciation, and that it is probable that it is through such measures that benefit is obtained in all cases that respond to such treatment. Your correspondent's observations emphasize the further benefit to be obtained by the attendance of a psychiatrist to take advantage of the probability of such response.—I am, etc.,

Tipton, Staffs.

L. H. EUNSON.

Causalgia of the Face

SIR,—I read with interest Mr. J. A. W. Bingham's report of two patients suffering from causalgia of the face (June 7, p. 804). Our knowledge of this condition is so incomplete that individual experience is well worth recording. I cannot agree, however,

with his conclusion, "that when sympathectomy relieves causalgic pain and tenderness it does so by interrupting the sensory pathway." It is not proved that sensory (afferent) fibres travel to the spinal cord by way of the sympathetic chain, and secondly one can see that pressure on the superior cervical ganglion can stimulate efferent fibres passing through. "Novocain" block of these fibres at a lower level would produce a temporary paralysis in their peripheral course and distribution and so prevent the occurrence of pain as noted.—I am, etc.,

Salce, Cheshire.

C. H. CULLEN.

Reiter's Disease

SIR,—The annotation on Reiter's disease (Dec. 7, 1946, p. 865) summarizes the present state of the knowledge of this condition very well. A few points noted in four cases by me (subject of communication elsewhere) are worth mentioning. Though you have quoted several writers as correlating the disease with bacillary dysentery, Jackson's contrary opinion, which has also been quoted by you, seems to be confirmed by my four cases, none of which had any relationship with bacillary dysentery. Further, in India we see bacillary dysentery cases literally by thousands, and yet there are no authentic records of Reiter's disease. Had the two conditions been aetiologically related there would surely have been more cases of Reiter's disease in this country.

The categorical statement made by you that "... there is no relation to sexual intercourse" is not wholly correct. Kristjansen¹ described his case in detail in 1930 where the infection was traced to a girl of 16 who for two years had had a yellow vaginal discharge. This girl had also infected another man at about the same time, and the second victim developed only an uncomplicated urethritis lasting three months. In neither man was the gonococcus or any other organism found. The girl herself was examined and found to have had only an inflamed vagina, but gonococci were not demonstrated in her either. The sexual relationship in these two cases is clear. One of my four cases also developed the condition after extramarital coitus.

Neutrophil leucocytosis has been reported in all the cases, but one of my cases had an eosinophilia of 4 to 7% in a total leucocyte count of 11,000 to 13,000. This may mean that allergy is a factor in the disease. This case went through the gamut of bilateral conjunctivitis and polyarthritis, and the eosinophilia persisted over the earlier part of the illness. The patient did not have any parasitic infection or infestation. The possibility of allergy being aetiologically responsible is strengthened by the observation of Forbes² that his case was associated with dental sepsis. Junghans³ tells about a case in conjunction with a furuncle in the upper lip, while Frühwald⁴ described two cases, in one of which the first sign was urethritis and, in the other, joint pain in the left foot. The example of erythema nodosum being the result of a variety of infective processes may be mentioned as a possible parallel to the incidence of Reiter's disease following upon non-specific infections.

In none of the four cases observed was the complement fixation test for gonococci done, but gonococci were not found in any of the exudates by the usual staining methods. The tests were repeated many times and under varying clinical conditions.—I am, etc.,

Bangalore.

P. N. BARDHAN.

REFERENCES

- ¹ Kristjansen, A. (Translation by C. Rasch) (1930). *Ugeskr. Laeg.*, **92**, 275.
- ² Forbes, D. (1946). *British Medical Journal*, **2**, 859.
- ³ Junghans, O. (1913). *Deutsch. med. Wschr.*, **44**, 1304.
- ⁴ Frühwald, R. (1927). *Jadassohn's Handbuch der Haut- und Geschlechtskrankheiten*, vol. 21, p. 478.

Physical Therapy of Mental Disorder

SIR,—It seems to the mind of an ordinary physician a great pity that there should be a need for bitter and violent controversies on the subject of psychiatry; I refer to the article written by Dr. D. W. Winnicott (May 17, p. 688) and to the replies by Drs. W. Malcolm Millar, Dr. A. Spencer Paterson, Dr. A. Lionel Rowson, Dr. A. N. Hardcastle, and Dr. E. E. Feldmesser (June 14, pp. 861 and 862). Whatever the rights and wrongs of it all may be, it seems to me that always should we be able safely to look to, and to accept, the findings and dicta of fully recognized and really experienced psychiatrists.