Diagnosing Measles

SIR,—Now that measles vaccine is generally available it becomes of more importance to make an accurate diagnosis of measles in infancy. A condition that sometimes seems to be mistaken for measles is exanthema subitum or roseola infantum.

In this condition the small child aged 6 months to 3 years presents with a high fever possibly associated with slight coryza and cervical adenitis, but insufficient to account for the height of the fever. On the third to fourth day a generalized maculopapular rash develops with sudden subsidence of the pyrexia. The rash usually does not persist for 24 hours. Needless to add this illness does not prevent the development of measles at a later date.—I am, etc.,

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Aphthous Ulceration

SIR,—Your leading article on the riddle of aphthous ulceration (20 April, p. 131) rightly emphasizes that the aetiology of this common and troublesome disorder is unknown, certainly in the majority of cases. Although you discuss several factors which may play a part and mention some related diseases, you say nothing of the undoubted and close association with cyclical neutropenia. This association emerges most clearly from Reimann's review of the literature which he published in 1963.[1] Oral ulceration occurred in 31 of 42 reported cases and, with fever, was the commonest clinical manifestation of cyclical neutropenia. In contrast, he found no evidence of cyclical neutropenia in patients in whom recurrent aphthous ulceration of both oral and genital mucosa occurs.

In a more recent study Morley, Carew, and Baikie[2] found oral ulceration in 9 out of 20 patients with cyclical neutropenia. In eight of the nine patients cyclical neutropenia was clearly a familial disease; in the ninth patient familial occurrence could not be demonstrated, but the patient was thought to have lymphosarcoma. More important from the point of view of clinicians likely to see patients with recurrent aphthous ulceration is the fact that, in four of our nine patients with oral ulceration it was the only symptom of cyclical neutropenia. We have previously drawn attention[3] to the difficulty of establishing a diagnosis of cyclical neutropenia which can best be done by twice-weekly total and differential white cell counts carried out over three or four weeks. The diagnosis is probably justified in a patient with recurrent mouth ulceration with either chronic noncyclical neutropenia or even without demonstrable neutropenia, if cyclical neutropenia can be demonstrated in a first-degree relative who may himself be symptom-free.

Since our paper on cyclical neutropenia was written we have seen other cases of recurrent aphthous ulceration of the mouth in whom cyclical neutropenia was previously unsuspected but later proved by serial blood counts. We have no way of knowing what proportion of patients with recurrent aphthous ulceration have cyclical neutropenia or the chronic unremitting neutropenia which develops in some cases of cyclical neutropenia after early adult life. This information can only emerge from more frequent investigation of the association by those who see many patients with recurrent mouth ulcers. Morley[4] has suggested that the neutropenia of cyclical neutropenia is an exaggeration of a periodic variation in the level of peripheral blood neutrophils demonstrable in normal individuals and even in patients with chronic granulocytic leukaemia.[5] Oral ulceration in the neutropenic may be due to impairment of mucosal defences by lack of migratory neutrophils. Alternatively, it may be a consequence of an error in the feed-back mechanism which ensures normal replacement of mucosal cells, analogous to the exaggerated time-lag postulated by Morley to explain cyclical neutropenia.—I am, etc.,

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Cerebral Malaria

SIR,—I wish to comment on the article of Professor A. W. Woodruff and Dr. C. J. Dickinson on the use of dexamethasone in the treatment of cerebral malaria (6 July, p. 31), and on Dr. T. Harding's observation thereon (27 July, p. 250).

There is, of course, the possibility—a strong one—that the drug saved this man's life. Nevertheless, Dr. Harding's plea for a controlled trial before a dogmatic statement could be made is understandable, and this should be done. In the meantime however, one does not see what harm could be done by the judicious use of this drug in individual cases of cerebral malaria, which could perhaps save hundreds of lives in many parts of the world. But I do not think it should be used routinely at the onset of the illness. The irreversible cerebral damage feared by the authors does not seem to have materialized. One should in the early case be inclined towards the standard use of parenteral antimalarials, the cautious use of lumbar puncture, drips, and methods for the sustenance of the blood pressure, nutrition, and vital functions.

As regards the rationale for the use of dexamethasone, the authors said it was to reduce cerebral oedema. This drug has a distinct advantage over hydrocortisone in its virtual lack of sodium-retaining properties, and may indeed induce sodium diuresis. Be that as it may, one wonders if the probable reduction of cerebral oedema is the whole picture here. The corticosteroids have been widely used, albeit empirically, in patients who are in extremis, often to counteract presumed suprarenal insufficiency. It seems to me that this might be the action of dexamethasone in this man. There is no evidence from this article that diuresis was induced by the drug, and, in fact, the serum sodium rose, 128 mEq/l. to 137 mEq/l., at a time when progress was continuing satisfactorily. This man probably had little haemorrhages and infarcts in his suprarenals, giving a Waterhouse–Friderichsen syndrome (suprarenal apoplexy).

One must also not lose sight of another effect of steroids—reportedly rather marked with dexamethasone—that is, the ability to cause excessive mental stimulation. This property may have also played a part in the remarkable recovery of this man. It would be interesting to compare the results of using more orthodox diuretics, for example, frusemide parenterally, to reduce cerebral oedema in place of dexamethasone in future cases. The paper does not say whether there was much sodium in the drips given to the patient.

Lastly, may I humbly add that I have myself used parenteral hydrocortisone in some serious cases of cerebral malaria, purely on the premises outlined above, and of course in conjunction with the antimalarials, etc., with reasonably favourable results?—I am, etc.,

A. AFYZ HARDY.

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SIR,—Many hospitals and health centres in areas where falciparum malaria is endemic do not at present stock supplies of intravenous steroids and almost invariably operate on a limited budget. If intravenous steroids are to become part of the standard treatment of cerebral malaria, they will have to be obtained at the expense of other drugs. A controlled trial of this treatment is therefore fully justified. The trial of A.T.S. in the treatment of tetanus is an analogous case. Would Dr. J. de Swiet (10 August, p. 377) maintain that this was not justified?—I am, etc.,

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University of Ibadan,
Ibadan, Nigeria.

Renal Damage and Virus Infection

SIR,—Your leading article on renal disease and virus infection (3 August, p. 264) prompts us to record the following case history:

A 51-year-old farmer was admitted to hospital with a history of painless swelling of the ankles and wrists for three weeks and hoarseness for two weeks. Four days before admission he developed a productive cough and became breathless, at first on effort and later at rest. Four months previously he had developed jaundice, with pale stools, with apparent complete recovery in three weeks. On examination he was a thin man, slightly flushed but apyrexial, with a haemorrhagic rash on his feet. Total white cell count was 15,300/cu. mm., of which 81% were neutrophils. Platelets were normal: the sedimentation rate was 17 mm. in the first hour, and his haemoglobin was 95% (13.8 g./100 ml.). He was slightly icteric, with a serum bilirubin of 1.6 mg./100 ml. His liver function tests indicated diffuse hepatocellular damage, but prothrombin time was normal. His liver was enlarged to four fingerbreadths below the costal margin and was not tender, and there was no enlargement of spleen or lymph nodes. Heart sounds were normal, with tachycardia of 112/min.; B.P. 170/128. There was oedema of both feet, but a normal jugular venous pressure. E.C.G. showed sinus rhythm, with non-specific T-wave inversion in V4–V7 leads. The heart contour by *x*-ray showed modest general cardiac enlargement. He had clinical signs of left pleural effusion. This was confirmed by *x*-ray which also showed a mid-zone flare. The

pleural aspirate showed only inflammatory cells, mainly lymphocytes. No malignant cells were seen. He had heavy albuminuria, and the urinary deposit showed many red blood cells with a few hyaline and granular casts. His serum electrolytes were normal, but his blood urea was 179 mg./100 ml., and his serum cholesterol 320/100 ml. The serum albumin/globulin ratio was reversed (albumin 2.2 g./100 ml., globulin 6.4 g./100 ml.), and electrophoresis showed the gammaglobulin fraction to be approximately twice the normal value. Leptospirosis, chemical poisoning, polyarteritis, Henoch-Schönlein purpura, brucellosis, and infectious mononucleosis were excluded. Sera collected 3 and 18 days after admission gave complement fixation titres respectively of 64 and 32 to mumps S (soluble) antigen and 1,024 and 2,048 to mumps V (viral) antigen. Mumps virus was isolated from a specimen of urine collected 17 days after admission.

Following the removal of 1,100 ml. pleural fluid his orthopnoea was completely relieved, but in the following three days he had three episodes of severe bronchospasm, in each case immediately relieved by aminophylline and frusemide intravenously. By the fifth day he was symptomless, and during the following 24 days his blood urea slowly fell to 82 mg./100 ml. and his bilirubin to 0.5 mg./100 ml. The pleural effusion did not recur, and his serial E.C.G.s remained unchanged. However, the oedema of his legs persisted; his urinary protein loss rose to 12 g. daily and the serum albumin fell to 1.4 g./100 ml. He was put on prednisone 20 mg. t.d.s., with a reduction in urinary protein loss to 1 g. within five days. Thirty days after admission he developed severe cellutitis with gross oedema of his right leg, which clinically responded to antibiotic therapy. Following this infection his renal function rapidly deteriorated, the blood urea rising to 250 mg./100 ml. and his serum potassium to 7.1 mEq/l. There were, however, no E.C.G. changes characteristic of hyperkalaemia. Despite all therapy his condition deteriorated over the following week and he died 48 days after admission.

This patient illustrates the generalized nature of virus infections, which may manifest themselves by affecting any organ or system either singly or in combination. Myocarditis is recognized as a possible manifestation of mumps infection,[1] and hepatitis is included in the list of sequelae compiled by Trimble.[2] Nephritis as a complication of mumps has been recognized since 1905,[3] but virological confirmation[4] awaited the development of adequate laboratory techniques. Four fatal cases have been described,[5 6] and recently two non-fatal cases of nephritis following mumps infection have been recorded (23 December 1967, p. 721).[7]

Most of the cases of mumps nephritis previously described have occurred in children or young adults following parotitis. The case reported here is of special interest, as the infection occurred in a middle-aged patient who gave no history of parotitis or neck swelling. Though it is not possible to ascribe the original attack of hepatitis to mumps virus, the serological tests indicate that mumps infection had occurred some weeks prior to hospitalization. It is almost certain that mumps virus was the agent responsible for his final illness.—We are, etc.,

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Undressing the Patient

SIR,—Blood-pressure readings may be recorded both accurately and expeditiously by applying the cuff of the sphygmomanometer over such layers of clothes as will transmit a palpable arterial pulsation.—I am, etc.,

HILARY LORING WEBB.

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Surgical Induction of Labour

SIR,—In your report of the 18th British Congress of Obstetrics and Gynaecology (10 August, p. 368) you state "Professor J. H. M. Pinkerton (Belfast) pointed out that 10% of surgical inductions of labour failed and caesarean section became necessary and did not involve any special risk."

We do not accept that caesarean section after failed induction carries no special risk, and since that Congress have shown that the figure of 10% can be greatly reduced by the routine use of intravenous synthetic oxytocin infusion at amniotomy. Our failure rate was 3.3% in the first 150 cases reported.[1] In the first six months of 1968 a further 184 cases have been induced in this way with only five failures—that is, 2.7%. The foetal and maternal morbidity have remained particularly low and similar to our initial report.

This method of induction of labour is now standard practice in this unit and in our view is the best available. As the figures show, there is a startling reduction in failures.—We are, etc.,

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Primary Non-specific Ulcer of Ileum

SIR,—I was interested to read the report by Mr. M. J. Shah (24 August, p. 474). The following is an account of a similar but more chronic case, again presenting with rectal haemorrhage.

A white male initially presented at another hospital in 1961 (then aged 22 years) with melaena. Following transfusion he recovered and all investigations proved negative. He remained symptom-free until March 1968, when he presented here with further loss of clotted dark red blood. Apart from pallor and tachycardia there were no significant clinical features. Haemoglobin concentration was 4.6 g./100 ml. Again full investigation was negative and he responded to transfusion. While considering an advised laparotomy he was readmitted in June 1968 with further loss of dark red blood. The haemoglobin level on this occasion was 10.8 g./100 ml. Again he responded well after transfusion. At elective laparotomy ten days later a narrowed, thickened segment of ileum, 1.5 cm. long, was discovered about 120 cm. from the ileo-caecal valve. Proximal to this the ileum was dilated for about 20 cm. Resection with anastomosis was performed.

Macroscopically the narrowed segment exhibited an elliptoid ulcer 2 cm. × 1 cm. in size with its long axis lying transversely. There was surrounding induration and puckering of serosa. In the ulcer base were a few haemorrhagic spots. Macroscopically the lesion was a non-specific benign chronic ulcer. There was no evidence of any Meckel's diverticulum or ectopic gastric mucosa.

This case is different from Mr. Shah's in its chronicity. Both are unusual in that they presented purely with bleeding. From the reports quoted by Mr. Shah only two cases were recorded with a history longer than seven years, and all the chronic cases invariably presented primarily as perforation or obstruction, with or without episodes of melaena. It would seem that early laparotomy should be considered in unexplained cases of melaena, especially in the younger age groups.

I wish to acknowledge assistance from Mr. D. H. Mackay, Dr. F. Wemyss Smith, and Dr. C. A. K. Bird.

—I am, etc.,

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Macclesfield.

Seat Belt Injuries

SIR,—Your leading article (6 July, p. 4) reviewed some current literature on the safety and efficiency of seat belts. Very properly, the writer was at pains to point out that most of the reports quoted dealt with American-type (large) automobiles and their usual (lap strap) harness. In passing, it is relevant to comment that under recent federal safety regulations in the United States the addition of a shoulder strap is now specified.

I should like to draw attention to perhaps the largest single survey of this problem (obtainable from Volvo, Sweden), carried out on European cars and European-type lap-and-diagonal) harness and therefore more directly applicable to British conditions. Included in the survey carried out by Mr. Nils Bohlin were 300,000 cars in the 1½-litre class made by one Swedish manufacturer (Volvo) who has included 3-point seat belts in all his cars as standard fitments since 1959. The material is likely to be complete, because under their local five-year guarantee Volvo offer to pay for all accident damage repairs to their cars costing more than £75. Although over 98% of the cars featured this standard harness, only 25% of drivers and, even more surprisingly, 30% of front passengers were actually wearing them at the time of the crunch. In all, the investigation covered 28,750 reported accidents (collisions with repair costs of less than £75 not included), involving a total of 42,813 persons, of whom 2,445 received slight or severe injuries and 57 were killed.

Had safety belts given no protection, one would expect injuries in unbelted drivers and front passengers compared with those strapped to be in a ratio of 3:1, justifying a prediction of 16 or 17 belt-wearers among the 57 killed. In fact, there were only three killed while wearing their belts, and all while travelling at over 65 m.p.h., while in unbelted occupants very severe injuries were reported at speeds as low as 12 m.p.h. Belted, less