

exaggerating the situation, but, as Dr. Scott and Mr. Malcolm Morrison in a recent article¹ have emphasized, it seems possible that requests for paraclinical investigations are not always entirely rational. Dr. Scott and Mr. Morrison have indicated some questions which the clinician ought to ask himself when requesting an investigation. We would suggest that the clinician asks himself two questions: (1) What information do I expect to gain from this investigation? (2) Will this knowledge affect the management of the patient? If he does not know the answer to the first question, or the answer to the second question is "no," then it would seem likely that the examination should not be carried out.

We hope Dr. Scott's letter will provoke further correspondence as we believe that investigation into these problems is long overdue.—We are, etc.,

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REFERENCE

- ¹ Morrison, M., *World Medicine*, 1969, 5, No. 6 p. 42.

Pathogenesis of Pre-eclampsia

SIR,—I would agree with Mr. A. T. Coopland (13 December, p. 688) that much of the controversy and conclusion regarding the pathogenesis of pre-eclampsia is due to the difficulty in differentiating patients suffering from this disease and those with a chronic renal lesion. The difficulty is most apparent when the patient exhibits proteinuria with some oedema and mild or no hypertension. As has been shown¹ if alpha₂macroglobulin can be demonstrated in the urine the patient is suffering from an organic lesion of the kidney—in our cases, Type II (Ellis) nephritis. In pre-eclampsia this large molecule protein does not appear until the condition is severe, with marked proteinuria and a diastolic pressure above 100 mm. Hg.

This relatively simple test, carried out by the aid of immuno-electrophoresis, serves to distinguish the two types of patient, and will ensure that the population for study is clinically homogeneous.—I am, etc.,

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REFERENCE

- ¹ McEwan, H. P., *Journal of Obstetrics and Gynaecology of the British Commonwealth*, 1969, 76, 809.

Screening Blood Donors

SIR,—Drs. J. E. P. Fitzpatrick and C. C. Kennedy (1 November, p. 299) state that the only means of excluding donors carrying hepatitis virus is by careful questioning. However, Gocke and Kavey recently described a close correlation between transmission of hepatitis by blood donors and the presence of Australia (Au) antigen in their serum.¹

In two cases of hepatitis, both presumably transmitted by blood transfusion, we have

therefore retrospectively studied the donors involved. In both cases one of the donors was found to carry the Au antigen in the blood.

The first patient was a 50-year-old woman who in May 1969 received seven units of blood during removal of a cerebral tumor. Three months later she developed icteric hepatitis. Au antigen was demonstrated in her serum by means of an immune precipitation reaction in agarose.² Four weeks later the test was negative. In October the seven donors were tested for the presence of Au antigen. One donor was found to be Au positive. He had normal liver function tests and a negative history regarding icterus or contact with icteric patients.

The second patient was a 54-year-old woman who in February 1969 received three units of blood during operation for an arterial aneurysm. Four months later she developed icteric hepatitis. Her serum was tested only when she had practically recovered; no Au antigen could be demonstrated. In October the three donors were tested for the presence of Au antigen and here also one of the donors was found to be positive. Liver function tests could not be performed. This donor had suffered from icterus eight years previously.

The finding of two Au-positive donors out of 10 contrasts sharply with the results obtained in an unselected series of 800 consecutive donors, where Au antigen was only found in two. In both hepatitis cases therefore we assume the Au-positive donor to have transmitted the causal agent. It is striking that the donors still carried the antigen five and eight months respectively after having donated the infective blood. They are probably to be regarded as healthy carriers and have been excused from donating blood.

It is unlikely that it will be possible in all cases of hepatitis after blood transfusion to find the infective donor by retrospective testing for Au antigen. Nevertheless it seems worth while to follow this procedure until a time when prospective screening for viraemia has become a matter of routine.—We are, etc.,

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REFERENCES

- ¹ Gocke, D. J., and Kavey, N. B., *Lancet*, 1969, 1, 1055.
² Prince, A. M., *Proceedings of the National Academy of Sciences of the United States of America*, 1968, 60, 814.

Glue Ear

SIR,—The leading article on glue ear (6 December, p. 578) perpetuates the uncertainty and confusion of thought on this subject to such a degree that I feel compelled to contradict several of the statements made.

To quote a few passages: "The viscosity seems to be due to mucus . . ." There is no seems about it. It has been proved by histological staining methods (periodic acid Schiff stain, toluidine blue in polarized light) that the viscosity of the middle ear exudate is due to its high content of mucopolysaccharides. Once this fact has been recognized and understood, one principle of treatment at least becomes clear—namely, that we ought to attempt to liquify the mucus (or break up the polysaccharide molecule) by a suitable mucolytic agent—for example, urea. The high viscosity of mucus interferes with the action

of cilia of the Eustachian tube and of the middle ear mucosa just as much as it does in the bronchi, nose, and sinuses. That this is a fact and not conjecture must again be clearly stated, instead of using the vague phrase "dysfunction of the Eustachian tube." But the uncertainty continues: ". . . this dysfunction may as well be due to mucosal swelling in the tympanic orifice as to some failure at the pharyngeal end." It is also an unequivocally established fact that the Eustachian tube is not blocked at either end.¹⁻³

Even the writer of your leading article acknowledges that removal of adenoids does not prevent or mitigate glue ear. I wholeheartedly agree with this statement. In a large series of patients I have found adenoids in only 20% of cases.

Once the fact that the Eustachian tube is patent has been comprehended, it becomes illogical trying to ventilate the middle ear through the tympanic membrane by the insertion of a grommet. Fortunately Nature knows better and "the grommets are generally extruded before six months, the membrane healing behind them. They are left in position until they are extruded or deliberately removed because they have become blocked" (*sic!*).

The leading article rightly states that diagnosis of glue ear is difficult and "in some cases it cannot be excluded without diagnostic myringotomy." A far better method is a diagnostic aspiration with a short-bevelled wide bore needle (a Harris lumbar puncture needle). If viscid exudate is present in the middle ear, on attempted aspiration a vacuum will form in the syringe and will tend to bring the plunger back into the barrel, because the "glue" will not go through the needle. I have found this to be a most valuable diagnostic sign of glue ear. The puncture in the tympanic membrane heals quicker than a formal myringotomy incision, in 24-48 hours.

In my experience the most suitable mucolytic agent for the treatment of glue ear is a solution of urea (2 g. of urea/4 ml. of water) prepared immediately before use and sterilized by filtration through a Millipore filter. Boiling would cause hydrolysis. The urea solution is injected under general anaesthesia through the tympanic membrane into the middle ear.³ This injection also automatically increases the hydrostatic pressure in the middle ear which overcomes the additional physical forces causing retention of the exudate (surface tension, capillarity, negative intratympanic atmospheric pressure etc.). This treatment is far more physiological than the use of the grommet, because it aims at re-establishing aeration of the middle ear by the only natural way, namely the Eustachian tube. The children (and the parents) must then be taught how to inflate their Eustachian tubes by the Valsalva manoeuvre to maintain the aeration of the middle ear.

The last but not least confusion of thought is to compare the success or failure of the various methods of treatment of glue ear by the number of relapses. These are a matter of the child's immunity to upper respiratory tract infections. Immunity increases with advancing age and at puberty is usually high enough to prevent further recurrences. It has nothing to do with hormones and nothing whatsoever with the various methods of treatment under discussion. The success of treatment should therefore not be judged by the number of relapses but by the improvement in hearing.

The concluding advice of your leader writer that it is "sometimes wiser to prescribe a hearing-aid to tide the patient over rather than to persist in repeated myringotomies and

grommet insertions" speaks for itself. What an admission of failure and advice of despair!—I am, etc.,

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REFERENCES

- 1 Senturia, B. H., *Proceedings of the Royal Society of Medicine*, 1963, 56, 687.
- 2 Sade, J., *Archives of Otolaryngology*, 1966, 84, 297.
- 3 Bauer, F., *Journal of Laryngology and Otology*, 1968, 82, 717.

** The consensus of expert otological opinion is strongly in favour of the use of grommets or similar devices for the relief of glue ear. This preference is based on the satisfactory results obtained in large published series—for example, Feuerstein, 191 patients;¹ Oppenheimer and Siegel, 300 patients;² which must be compared with Bauer's 18 patients (27 ears)³—both in the short term as judged by immediate improvement in hearing and in the long term by the relapse rate.

Success with the grommet, which acts not as a drain but as a pressure equalizer in the middle ear, points to Eustachian tube dysfunction as an aetiological factor, a view that has received support from such an eminent international authority as Professor Schuknecht, of Boston.⁴ It is known that air may be forced through the tube in these cases from either end, but that does not necessarily mean the tube is functional in a physiological sense. A cautious expression of view in this debatable subject still seems appropriate.—Ed., *B.M.J.*

REFERENCES

- 1 Feuerstein, S. S., *Laryngoscope*, 1966, 76, 686.
- 2 Oppenheimer, R. P., and Siegel, J. R., *GP*, 1967, 35, 105.
- 3 Bauer, F., *Journal of Laryngology and Otology*, 1968, 82, 717.
- 4 Schuknecht, H. F., *Clinical Pediatrics*, 1964, 3, 718.

Herpes Gestationis

SIR,—I read with interest your recent leading article on herpes gestationis (29 November, p. 516). The fact that you say no satisfactory explanation for the development of this dermatosis has been offered and that it closely resembles bullous pemphigoid of the elderly prompts me to report the following case of a patient recently admitted to our wards:

A lady of 82 years presented with a severe bullous eruption of six days' duration affecting mainly the limbs but also the centre of the abdomen. This was diagnosed as pemphigoid. She was also found to have gross abdominal distension of several years' duration. This was shown to be a benign ovarian cyst at operation, when 25 l. of fluid was removed. She died on the third postoperative day of left ventricular failure and bronchopneumonia.

Could abdominal distension be a possible factor in the causation of herpes gestationis?—I am, etc.,

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"Rebreathing Bag"

SIR,—Your leading article (15 November, p. 383) properly raises the question whether anaesthesia in a completely darkened room is ever required or indeed justified. This thoughtful article has great merit, and there-

fore it is the more distressing to note that the writer uses the antiquated and thoroughly misleading term "rebreathing bag."

This part of the anaesthesia machine is more properly called a "breathing or reservoir bag," since the one function it should not subserve is that of facilitating rebreathing unless proper precautions are taken for the disposal of carbon dioxide. Indeed, the misconception that rebreathing is one of the functions of this bag has led to fatalities. It is high time, therefore, that this misnomer be discarded.—I am, etc.,

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Peanut or Sweet?

SIR,—In the last few months we have come across several patients under the age of 2 years who had inhaled the kernels of peanuts. In each case there were some difficulties over the extraction of these vegetable foreign bodies, and in one no radiological changes.

Inhalation of peanuts leads to a local inflammatory response which may be severe, and the child may develop bronchospasm. If a history of inhalation is lacking the child could be misdiagnosed as having acute bronchiolitis, bronchitis, or another similar condition. Because of its shape and size, the peanut is easily inhaled and can readily travel down and obstruct a main bronchus. In children under the age of 2 years the difficulties of removal include induction of anaesthesia, passing of a bronchoscope of adequate size to allow instrumentation, and the ease with which the peanut can break when gripped with forceps. With care these difficulties can be overcome.

It has been suggested that children should be given items such as peanuts rather than sweets to prevent early dental troubles. However, we would urge that children in this age group should not be given, or encouraged to eat, peanuts because of the risks involved.—We are, etc.,

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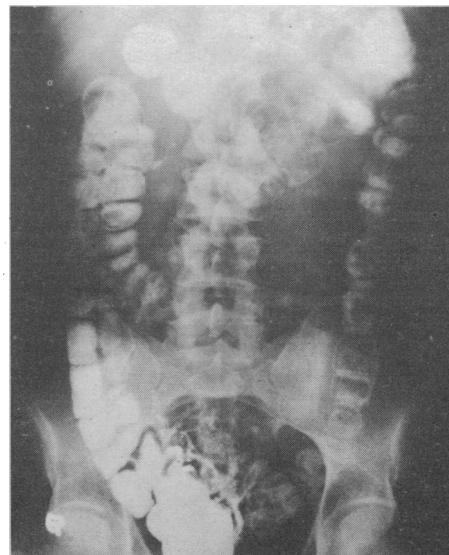
Possible Case of Ischaemic Colitis in a Young Man

SIR,—Radiological changes may occur in the colon following episodes of arterial insufficiency. These lesions—the most characteristic of which is known as "thumb printing"—tend to occur in the distal transverse and the proximal descending colon. In the majority of cases the colon returns to normal in a matter of weeks. As the condition is better known in elderly people, many of whom had overt arterial disease or suffered from diseases which predispose to arterial insufficiency, we think it is of interest to report the occurrence of possible ischaemic colitis in a previously healthy young man. In addition, the natural history of the patient's illness confirms the rapidity with which the radiological changes of ischaemic colitis can disappear.

The patient was a 26-year-old white male. He was perfectly well until the morning of 10

March, when he developed a watery diarrhoea, passing six stools that day. The stools did not contain blood. He had no abdominal pain, but did note a slight discomfort in both groins. Two days later he had abdominal cramps, which were not relieved by the passage of faeces. He now noted that the loose stools contained blood. He also experienced tenesmus and thought that he had passed mucus. The pain became more severe during the night of 13 March and he could not sleep. When seen the next day he did not appear ill. His temperature was 99° F. (37.5° C.) and his blood pressure 120/85 mm. Hg. No abnormality was detected on general examination and no bruits could be heard over any artery. There was tenderness on palpation of the left upper quadrant of the abdomen and along the line of the left side of the colon. Rectal examination, proctoscopy, and sigmoidoscopy to 25 cm. were normal. Rectal biopsy was normal. No pathogens were isolated from the stool. His haemoglobin, white cell count, blood urea, and urine were normal, but the E.S.R. was 29 mm.

Chest x-ray was normal. A straight film of abdomen revealed air in the transverse and upper part of the descending colon, with smooth rounded indentations protruding into the lumen of the descending colon. A barium enema done the same afternoon showed changes which have been described in ischaemic colitis—namely, thumb printing and saw-tooth appearance. (See Fig.)



Barium enema showing thumb printing and saw-tooth appearance.

He was given no specific treatment and the next day he was so well that he requested that he be allowed home because of pressing personal affairs.

It was felt that the diagnosis of Crohn's disease had to be excluded and a small-bowel enema was performed on 21 March, when the patient had no symptoms whatsoever and his E.S.R. was 10 mm. in the first hour (Westergren). The small-bowel enema was normal. The barium was followed through to the colon and no obvious abnormality could be seen. When the barium enema was repeated a week later the colon was normal.

The clinical presentation could have been due to an infective dysentery, Crohn's disease, or ulcerative colitis. However, the radiological features and the rapidity with which the patient's symptoms and radiographic appearances returned to normal, without any treatment, are possibly best attributed to ischaemic colitis.