Acute Gastroenteritis

SIR,—I would like to comment on three aspects of your very concise and topical leading article headed "Acute Gastroenteritis" (13 January, p. 70).

Although certain specific strains of *Escherichia coli* are undoubtedly concerned in the aetiology of gastroenteritis in young children, it is still possible that this disease is primarily a virus infection. At any rate, there is sufficient doubt to keep an open mind. Other infectious diseases such as influenza and ulcerative stomatitis have in the past been thought at first to be due to bacteria, and yet eventually a virus has been found to be responsible.

The paramount importance of adequate fluid replacement is correctly stressed, but surely it is going much too far to state that this can be done only by intravenous infusion. In my experience at least 9 out of 10 children admitted to hospital with gastroenteritis can quickly and effectively be rehydrated via the oral route. Half-strength Darrow's solution containing sodium chloride, potassium chloride, and sodium lactate, given by mouth in small frequent doses, is rapidly absorbed. A regimen of 2 oz. (50 ml.) every 15 minutes for the first two and a half hours, and then 2 oz. every hour for the next 10 hours, will usually result in satisfactory rehydration and restore any electrolyte imbalance. It is only in the case of the very occasional infant who continues to vomit or who is unconscious on admission that intravenous therapy is required.

The sooner correct treatment is started the better. All too often fluids are withheld, foods thickened, or glucose water given, and such measures cannot be too strongly condemned. A suitable tablet included in the *National Formulary* would certainly be an asset for home treatment, but the chances of this being available at the moment of need are perhaps remote. However, it is always possible to add half a teaspoonful of common salt to a pint (500 ml.) of water in order to get a satisfactory solution for commencing treatment at home. You rightly emphasize that in such circumstances there must be close observation, as these small children can deteriorate so very quickly.—I am, etc.,

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Diagnosis of Pyelonephritis in Children

SIR,—Your recent papers (23 December, pp. 697, 702, and 705) and leading article (23 December, p. 691) on this subject call for some comment. The authors emphasize that the correct collection of urine specimens from small children requires patience and time of an order which I believe to be beyond the resources of most of our ordinary nursery services today, and it would be impossible to conduct an adequate follow-up clinic along the lines described. Happily, the answer is in the hands of the clinician, for he can enumerate leucocytes in clean void urine to tell when there may be inflammation of the urinary tract and undertake a search for possible bacterial evidence of an infective cause for this. It has never been satisfactorily shown that bacterial infection of the urinary tract in childhood occurs without leucocyturia, and it is therefore easy to screen all children in and out of hospital to select those "at risk" of having an infection to see if the diagnosis can be confirmed or, more usually, excluded.

When leucocyturia is shown in more than one clean void specimen it *may* be possible to obtain a "clean catch" specimen by inducing reflex micturition, especially in young babies with a full bladder,[1] and this should always be tried. If it fails then bladder puncture should be done. The procedure is easy and has been performed on hundreds of young infants without any complication other than occasional transient haematuria. I have performed bladder puncture on 150 leucocyturic (>50/cu. mm.) infants and found a bacterial growth of $>10^5$ organisms/ml. in 18 and in each of these 18 there was bladder urine leucocyturia which could be immediately ascertained and was of invaluable help in management, especially in the outpatient clinic. All of the remaining specimens were sterile, and only a few of them contained leucocytes, in low numbers.

It is a very easy matter for the clinician to look for significant leucocyturia in all of his patients, and if a "clean catch" urine cannot be obtained from those "at risk" then bladder puncture should be performed. This is a procedure no more serious than venepuncture, which gives the cleanest specimen and most clear-cut answer. Indeed, the clinician-microscopist usually has the satisfaction of telling the diagnosis on the spot. I am, etc.,

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Urinary Tract Infection in Infancy

SIR,—Your statement (23 December, p. 691) that the data of Dr. H. Braude, Professor J. O. Forfar, Dr. J. C. Gould, and Dr. J. W. McLeod (same issue, p. 697) "can now be used as a basis for reporting quantitatively cell and bacterial counts in infants" should not pass without comment.

We should like to make two points. The first concerns the validity of the table of diagnostic levels of cell and bacterial counts. Applying them to collections made in our own wards we find that 7/63 (11%) routine bag specimens from baby girls studied by us[1] had results outside the stated normal limits, as did 2/99 baby boys. All these children were shown by suprapubic puncture to have sterile, cell-free bladder urine. Similarly, Houston's 1963 series[2] describes 77 collections of urine by clean voiding or Paul's tubing from uninfected male children. Of these, 10 (13%) gave results outside Braude and colleagues' stated normal limits. One must therefore doubt the general applicability of these tables to routine urine collections.

The second point we make is: how useful are such tables in clinical work? Do they help in the urgent case? In our experience, no. The chance of a false-positive or false-negative result is substantial.

It is our experience that paediatricians have not been unwilling to accept diagnosis by suprapubic bladder puncture in small infants and certain urgent cases. Bladder puncture is less distressing than lumbar puncture, and indeed than many venepunctures, and we have not yet experienced (or seen reported) a complication.—We are, etc.,

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2 Houston, I. B., *Arch. dis. Childh.*, 1963, **38**, 600.

Renal Glucose Threshold

SIR,—Professor W. J. H. Butterfield and others (2 December, p. 505) are to be congratulated on the many useful clinical points they make from their very extensive and excellent Bedford survey for diabetes. However, I cannot agree with the deductions which led them to make the main point in this paper. By their own definition the presence of glucose in the urine is not detected below 100 mg./100 ml. Indeed, the manufacturers (Ames Co.) wisely do not claim that Clinistix is reliable below this concentration and are careful to point out the inhibitory effects of ascorbic acid, *p*H, temperature reduction, etc. Because a more sensitive measure of glucose in the urine was not employed (such as paper chromatography) it would have been quite possible for a subject to void a concentration of glucose still undetected by Clinistix but contained in such a volume of urine as to represent more total glucose passed than a subject with a detectable concentration by Clinistix and smaller urine volume. We are not told the volume of urine voided in the test samples.

The oral glucose tolerance test, as applied in this survey, with blood sampling at half-hourly intervals, is probably valid as a test for diabetes mellitus in diabetics. The results should be interpreted with caution when applied in the same way to normal people. This is especially so when it is used to show some other physiological function for which it was never designed as a test. Because of the many factors influencing absorption and utilization of glucose the rise and fall of blood glucose in the normal child and adult can be very rapid or relatively slow, depending on circumstances.

It is not possible to show with confidence peak values of glucose reached in the blood unless a continuous sampling method is used. The authors say: ". . . the *presence* of glucose and not its *concentration* will be considered here. It should be pointed out, under these circumstances, that from the presence of glycosuria we can only deduce that the renal threshold was lower than the peak value found, and that in the absence of glycosuria the renal threshold was higher than the peak value." It should be pointed out that the opposite is just as likely to be true. It is clear that because of the method used the true peak of blood glucose could be missed in many patients, and therefore the renal threshold may have been higher than the value actually found in those patients with glycosuria. It is equally clear, because of the insensitive method used in the urine, that the absence of significant amounts of glucose is not proved[1] (more especially as we do not know the urine volumes). Therefore the renal threshold could just as easily be lower than the peak value actually found by the others.

It is plain that it is difficult to accept the deductions leading to the range of figures quoted as renal thresholds for glucose. Many of the points made in this paper are of the greatest importance in the detection and management of diabetes, and there is no wish to detract from these, but I do not think the renal threshold figures given are necessarily valid.—I am, etc.,

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Source of Pulmonary Emboli

SIR,—Mr. N. L. Browse and his colleagues writing on the management of the source of pulmonary emboli (9 December, p. 596) are to be congratulated on their rational and aggressive approach to this problem. One hopes that their aggression is limited to extensive thrombotic states such as phlegmasia caerulea dolens.

Since December 1966 it has been my policy to precede or follow pulmonary embolectomy (according to the patient's condition) by disobliterating involved veins. In one instance this policy was not followed. That patient suffered a massive pulmonary embolus seven days after a hip operation. A successful embolectomy was performed, delivering an 18-in.-long (45-cm.) clot. For reasons outside our control simultaneous thrombectomy could not be done. The patient succumbed four days later to a second massive pulmonary embolus. It was of similar length and arose in the opposite leg. This case without doubt weighs in favour of pulmonary embolectomy and simultaneous venous thrombectomy. Similarly most cases of pulmonary embolism can be looked upon as an indictment of failure to adopt measures advocated by Mr. Browse and his colleagues.

While the most reliable form of treatment of extensive deep vein thrombosis is by thrombectomy, followed by heparin perfusion and long-term phenindione, one feels compelled to add that:

(1) When ligation or plication is considered, it should be confined to the superficial femoral vein only.

(2) When pulmonary embolism occurs, pulmonary embolectomy should be accompanied by thrombectomy of the affected veins.

(3) It is difficult to condone plication or ligation of the inferior vena cava. These procedures in themselves create conditions favouring further vein thrombosis. Ligation of the inferior vena cava should be limited to recurring septic emboli.[1] Spencer,[2] originator of plication of the inferior vena cava, reporting on long-term results on 39 cases,[3] states that there were six immediate postoperative deaths, a further three within two years, four had immediate and extensive vein thrombosis, nine of the surviving patients had leg oedema, and two had recurrent phlebitis.—I am, etc.,

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Psychotropic Drugs

SIR,—Dr. H. M. Flanagan (13 January, p. 119) is correct in pointing out the increasing use of the label "depression," and not only in psychiatry. It is the "nuciform sac" of the 1960s, taking the place of intestinal stasis or focal sepsis or visceroptosis as a diagnosis of the undiagnosable.

However, it is difficult to share his despondency about it. We should be delighted that medical fashion has for once settled for something whose treatment is fairly simple, reasonably effective, carries relatively little risk to the patient, and above all involves no permanent mutilation.—I am, etc.,

R. ROUSE.

Hythe,
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SIR,—I hope I may be given the privilege of replying to what has proved a long and, I hope, profitable correspondence. It was indeed gratifying to find how many clinicians there were prepared to confirm the value of the monoamine oxidase inhibitor drugs when used in suitable neurotic depressions and anxiety states. But such patients would very rarely agree to enter a mental hospital for prolonged "double-blind sampling," and they were therefore conspicuously absent from the group of severer depressions used to provide the misleading M.R.C. report.[1]

It was also not unexpected to learn that a group of four mental hospital doctors, including Dr. P. Leyburn (14 October, p. 108), working in the Newcastle area, had failed to find as yet the sort of patient benefiting so much from the monoamine oxidase inhibitor drugs, although both their neurological[2] and psychiatric[3] colleagues at the Newcastle General Hospital have already paid written tribute to their efficacy in properly selected patients. One of the troubles about doctors training and working predominantly in our mental hospitals is that they apparently see so many severe depressions, and not enough minor depressive and anxiety states, which are mostly seen in general practice and in general hospital psychiatric units. They also seem to have the idea that you can justifiably treat psychotic depressions with drugs, but neurotic depressions and anxiety states should preferably be given sex talks or group therapy. And if they do use any drugs they mostly use those which they have found to help their severer depressions but are of little use in the neurotic ones, and often even make them much worse.

It is paradoxically the general practitioners who are now learning to their delight that neurotic depressions and anxiety states often do very much better with selected antidepressant drugs than any amount of talk about sex or other matters. So often the time to talk to both the neurotic and psychotic patients about their problems is when they have responded to the drugs and not always during the illness itself. For so often, as the symptoms dissolve under drugs, the problems also resolve with them, and very little supportive psychotherapy is then needed of a commonsense nature.

In my 30 years in psychiatry I have seen the most refined statistics used time and again both to prove and to disprove the value of insulin treatments, E.C.T., leucotomy, and now all the new tranquillizers and antidepressant drugs. One is therefore constantly forced back to the bedside to find out what really works in selected patients and what does not. It seems that people working with statistics often tend to lump both neurotic and psychotic patients together in a very crude manner, and give the drugs equally crudely in their statistical tests. When statistics obviously *contradict* clinical findings I have now learnt to treat them with the very greatest suspicion, especially in psychiatry. I am certain that general practitioners are not quite as foolish as Dr. Leyburn thinks in using the new antidepressant drugs so intensively. For so many of them have found clinically what he does not seem to be able to find by his crude statistics, that the antidepressant drugs, properly used, have very great value for properly selected groups of patients. And they are certainly not mere placebos given for the doctors' own benefit, as he suggests in his last letter (18 November, p. 417).—I am, etc.,

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Use of Epsilon Aminocaproic Acid

SIR,—In congratulating your expert contributor on the concise and helpful account of the use of epsilon aminocaproic acid (23 December, p. 725), may I comment on points raised in connexion with the diagnosis and management of fibrinolytic states, particularly as they occur in pregnancy and labour?

Your contributor remarks that "primary hyperfibrinolysis is difficult to distinguish from other causes of the defibrination syndrome." However, a shortened euglobulin clot lysis time,[1] or dilute blood clot lysis time,[2] does usually indicate pathological fibrinolysis. These tests are rather complicated and time-consuming, which precludes their routine use in clinical practice. Besides, two relatively simple tests are available for the assessment of the fibrinolytic activity in emergency situations. These are a prolonged thrombin clotting time,[3] not corrected by a mixture of patient's and normal plasmas, and a positive agglutination test with the Fi-test reagent (Hyland Laboratories, California), using patient's serum.[4] Both these tests are indirect tests for fibrinolysis (dependent upon the presence of an excessive amount of fibrin degradation products), but the simplicity of these tests should appeal to the clinician.

Your contributor further states that the "Treatment [of defibrination syndrome] should *begin* [my italics] with controlled replacement of fibrinogen and other missing factors. . . ." However, the primary aim of the treatment should be to prevent further infusion of thromboplastins into the circulation and arrest the phase of hypercoagulability. This is achieved in patients with abruptio placentae by amniotomy and prompt delivery.[5] Until this is achieved transfusion of fibrinogen causes further intravascular coagulation or lysis of transfused fibrinogen, depending upon whichever process is operative. If the problem is one of lysis the