known possible causes of atelectasis and complications representing a list of possible causes, though I attribute one reference under discussion to authors I should not have—namely, Kaneko in Prys-Roberts.

As to a mysterious further 116 patients. This is an error in editing on my part, and of course should have read 297 patients, being divided into a control group of 181 not given bicarbonate together with 116 receiving bicarbonate as stated in the summary. Fifty-two patients received halothane, but the importance of this is in the Table, which shows that these patients did not have a thiopentone induction unlike the other 235, so I thought it reasonable to separate them. The confusion between Venus and Astrup bicarbonate is only apparent because of the positioning of the table.

In conclusion, I should like to repeat how grateful I am for the criticisms of Drs. Conway and Leigh, and I hope this letter has helped to elucidate my paper for them and clear up some of their queries.—I am, etc.,

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Hepatic Carcinoma and Haemoperitoneum

SIR,—Dr. Marjorie L. Penwill's letter (17 October, p. 181) makes it sound as if haemoperitoneum in patients with malignant hepatoma is uncommon. In a series of 189 patients with primary hepatocellular carcinoma seen by us six patients presented as haemoperitoneum. Many of the patients with acute intrahepatic haemorrhage were not well enough to be transferred to us for treatment, so that the six patients mentioned form a very conservative estimate of the incidence of this presentation of the disease.

At post-mortem examination 15% of the patients treated by us were shown to have died of haemoperitoneum.—We are, etc.,

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Clofibrate and the Fibrinolytic System

SIR,—I agree with Dr. G. R. Fearnley (10 October, p. 114) that the action of clofibrate on fibrinolysis described in "Today's Drugs" (12 September, p. 632) requires qualification, but I do not agree with his comments.

A number of reports indicate that Atromid (clofibrate plus androsterone) or Atromid-S (clofibrate alone) increases fibrinolytic activity¹⁻⁵ and reduces raised levels of plasma fibrinogen^{3,4,6-8} in patients with ischaemic heart disease and hypercholesterolaemia. Both of these effects appear to be persistent. Statistically significant changes were apparent over a 9-month period of observation in patients treated with Atromid-S and over a 17-month period of observation in patients treated with Atromid.⁴ We have noted, however, that if an infective or thrombolytic episode occurs in patients receiving these preparations then

the temporary rise in fibrinogen and depression of fibrinolysis which results from these conditions still occurs. Similar observations were noted if patients receiving Atromid-S were subjected to surgical procedures. The pathways causing changes in these parameters following such stimuli probably differ from those producing changes in the chronic situation.⁹

As mentioned in "Today's Drugs" Atromid-S has no effect in reducing plasma fibrinogen or increasing fibrinolytic activity when these are in the normal range before treatment is instituted. This probably accounts for the results obtained by Sweet *et al.*,¹⁰ who obtained no effect on fibrinolysis or plasma fibrinogen levels in their patients treated with this preparation.

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REFERENCES

- 1 Srivastava, S. C., Smith, M. J., and Dewar, H. A., *Journal of Atherosclerosis Research*, 1963, 3, 640.
- 2 Hocking, E. D., Chakrabarti, R., Evans, J., and Fearnley, G. R., *Journal of Atherosclerosis Research*, 1967, 7, 121.
- 3 Gajewski, J., Skorykow, A., Ciszwicka-Sznajderman, M., Ignatowska, H., and Sznajderman, M., *Polskie Archiwum Medycyny Wewnętrznej*, 1964, 34, 1647.
- 4 Cotton, R. C., and Wade, E. G., *Lancet*, 1969, 1, 263.
- 5 Frandoli, G., Gibelli, A., and Giarola, P., *Clinica Terapeutica*, 1966, 38, 27.
- 6 Cotton, R. C., and Wade, E. G., *Journal of Atherosclerosis Research*, 1966, 6, 98.
- 7 Chakrabarti, R., Fearnley, G. R., and Evans, J. F., *Lancet*, 1968, 2, 1007.
- 8 Ogston, C. M., Ogston, D., and McAndrew, G. M., *Current Therapeutic Research*, 1965, 7, 437.
- 9 Thorp, J. M., Cotton, R. C., and Oliver, M. F., *Progress in Biochemical Pharmacology*, 1968, 4, 611.
- 10 Sweet, B., Rifkind, B. M., McNicol, G. P., and Gale, M., *Journal of Atherosclerosis Research* 1965, 5, 347.

Problems of Rectal Prolapse

SIR,—In the leading article on the management of prolapse of the rectum (14 November, p. 381) I was surprised to see no reference to the anal stimulator described by Hopkinson and Lightwood.¹

In my experience this device has become the treatment of choice in the first instance for all patients suffering from rectal prolapse. When it is effective it controls both the prolapse and the incontinence that goes with it. It is a very simple treatment and inexpensive. Even if it is not successful it is difficult to see how it could ever be harmful. Since this invention was described I have not had to subject any patient to surgery for rectal prolapse.

I regard the stimulator as a most excellent device and feel that it should be brought to the attention of your readers.—I am, etc.,

PETER H. LORD.

London W.1.

REFERENCE

- 1 Hopkinson, B. R., and Lightwood, R., *Lancet*, 1966, 1, 297.

Problems of Rectal Prolapse

SIR,—I would take issue with the writer of your leading article on rectal prolapse (14 November, p. 381) on two counts. In the first place, the evidence that rectal prolapse is really an intussusception starting at the

rectosigmoid junction is certainly not conclusive. A fundamental feature of any intussusception is that the precise site of origin of the intussusception becomes the leading point of the intussusception and remains so as the condition advances. In rectal prolapse in children, on the other hand, as the condition advances from a prolapse of about 1 cm. to perhaps 5 or 6 cm. the leading point becomes progressively a more proximal part of the rectum.

Secondly, I think a review of the surgical management of rectal prolapse is incomplete without reference to the injection of a sclerosing solution, such as saturated saline, into the pararectal tissues.¹ While it remains true that most cases of rectal prolapse in children settle down spontaneously with simple conservative measures such as strapping, there is undoubtedly a group which needs something more, and I would strongly recommend the injection of saturated saline into the pararectal tissues before proceeding to other surgical methods. The precise site of injection is of fundamental importance. A lumbar puncture needle is introduced about 5 cm. from the anal margin and is passed upwards until it can be felt by an index finger in the rectum to lie very close to the rectal wall. About 5 ml. of saturated saline is injected at each side and also posteriorly, and it is of the utmost importance to make sure that the injection does not raise a bleb under the finger which might lead to necrosis of the overlying mucosa. If such a bleb is felt, the needle is withdrawn slightly and the bleb massaged away at once before injecting more saline slightly further away from the finger in the rectum.—I am, etc.,

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REFERENCE

- 1 Kay, N. R. M., and Zachary, R. B., *Journal of Pediatric Surgery*, 1970, 5, 334.

Estimation of Immunoglobulin E (IgE)

SIR,—It is now established that reaginic antibodies belong to the class of immunoglobulins designated as IgE. These antibodies become fixed to the surface of mast cells in various tissues. When the allergen becomes attached to the antibodies, histamine and other amines are liberated from the mast cells giving rise to the allergic reaction (Type I allergy).