when acid secretion is absent or low—that is, in infancy or old age, and this is clearly a much more severe inflammation than the mild recurring heartburn of middle age. It looks, in fact, as if a fair secretion of acid/pepsin, diluting any bile reaching the stomach, protects the oesophagus against severe damage. Just under half of 25 unselected patients I studied had gastric atrophy, while two-thirds of the group refluxed overnight a fluid whose pH was more than 4, therefore beyond the range of peptic activity.¹⁶

(6) The route of access for surgery is immaterial, and its discussion hardly worth while. There are several safe operations available, and the abdominal approach is not new.^{5 17-19} These procedures have negligible mortality and give excellent results.

(7) One most fascinating newly opened avenue leading to understanding of a type of oesophagitis concerns the complex of gastric atrophy.²⁰ This is a degeneration of smooth muscle as well as of mucosa, widely affecting the foregut, and leaving the stage well set for biliary oesophagitis, as such patients have high resting concentrations of bile in their stomachs.¹⁴

Could I therefore plead for a spring-clean on this complex of diseases and indeed on all diseases when reviewed in leading articles? Our minds are overbombed with traditional and valueless concepts, and it is particularly important that the coming generation of doctors be spared such a blunderbuss of real and imaginary anatomical and aetiological factors in a disease-complex like this one. We have had to traverse a morass to reach the present state of knowledge. There seems little point in cluttering up the minds of our successors with outdated theories and incorrect facts when they have so much to learn anyway. They should surely be entitled to take over from what is known at present.—I am, etc.,

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Treatment of Adder Bite

SIR,—Your interesting leading article on the treatment of adder bites (16 August, p. 730) draws attention to the importance of avoiding infection by making no incisions at the site of the bite. It adds that the application of a tourniquet serves no useful purpose unless there will be a delay exceed-

ing several hours before hospital can be reached.

May I quote from the book by John Visser approved by the executive committee of the Cape of Good Hope College of General Practitioners? This states, under first-aid treatment, "Never apply a ligature after a bite by one of the common adders, but do so after elapid (mamba, cobra, rinkals, etc.) bites." It further explains the reason for this rule: "The ligature or tourniquet is applied to prevent or control the diffusion of venom from the site of the bite. In the case of adder bites, the ligature will, however, aggravate the oedema and anoxia at the site of the bite and thus cause such damage as is 'typically' seen after bites by these species. Ligatures are thus never applied after bites by adders."—I am, etc.,

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REFERENCE

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Spironolactone in Hyperaldosteronism

SIR,—In our view the leading article on "Management of Primary and Secondary Hyperaldosteronism" (23 August, p. 427) and your reply to the letter from Dr. M. J. Tidd (4 October, p. 52) gave a misleading account of the value of spironolactone treatment. We have studied more than 100 patients with hypertension, hyperaldosteronism, and subnormal or low plasma renin concentration (so called "primary" hyperaldosteronism). Forty-five of these have been treated with oral spironolactone in doses ranging from 150 to 400 mg./day (usually 300 mg.) for periods ranging from one month to six and a half years (total 485 months). Spironolactone corrected the electrolyte abnormalities in every patient, and produced a marked fall in blood pressure in most (to 150/90 mm. Hg or less in 24 cases). In the majority of patients the hypotensive effect of spironolactone was closely similar to that obtained by subsequent adrenal surgery; in eight cases spironolactone did not cause satisfactory lowering of blood pressure, and five of these were subsequently submitted to adrenal surgery without any effect on blood pressure.

We can confirm the range of side-effects mentioned in your leading article, but in our experience these are not invariable and rarely incapacitating. The drug had to be stopped in four of our patients, each of whom developed severe anorexia, epigastric discomfort, nausea, and vomiting. Barium meal examination revealed a large gastric ulcer in one of these patients; while this might have been an incidental lesion it is noteworthy that this 64-year-old patient had not experienced similar symptoms before spironolactone, and she had remained symptom-free since the drug was stopped 15 months ago. Our experience is that spironolactone in the treatment of primary hyperaldosteronism provokes fewer and generally less troublesome side-effects than do "conventional" hypotensive drugs in the treatment of other hypertensive patients. We agree that in most patients adrenalectomy is

the treatment of choice after the electrolyte abnormalities have been corrected by spironolactone therapy, but we wish to emphasize that prolonged treatment with spironolactone offers an efficient alternative therapy when surgery is contraindicated. Aldosterone-producing adenomata are sometimes small, and extensive dissection may endanger the adrenal blood supply, thus leading to total adrenalectomy with consequent dependence on corticosteroid replacements. Long-term treatment with spironolactone may well be preferred in cases of this type, and also perhaps in those in whom micronodular changes occur in both adrenal glands.

A preliminary account of some of these observations has been presented elsewhere.¹—We are, etc.,

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Automation in the Laboratory

SIR,—Your correspondent Dr. R. B. Niven (13 September, p. 656) draws attention to the delay in laboratory reports, and suggests British research could produce a system giving results in hours rather than months.

The problem is dealt with in *Abstracts of Efficiency Studies in the Hospital Service*.¹ The method of choice is one using N.C.R. (no carbon required) paper—no typing is carried out—and is similar to the national form now used for exfoliative cytology requests.

The average delay between obtaining results of tests and the dispatch of reports is now negligible. One consequence of this improvement is that fewer telephone inquiries are made to the individual laboratories, which saves the time of the technical and clinical staffs.—I am, etc.,

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REFERENCE

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Fibrinolysis and Toxaemia of Pregnancy

SIR,—The report by Drs. E. N. Wardle and I. S. Menon (7 June, p. 625) is of interest to those who associate pre-eclampsia with alterations in the coagulation and/or fibrinolytic systems. We have emphasized¹ that much of the confusion and lack of basic knowledge regarding the pathogenesis of "toxaemia" stems from the fact that few investigators have clearly defined their test populations. Thus Drs. Wardle and Menon studied 20 patients with hypertension and 23 patients with pre-eclamptic toxemia, "... defined as proteinuria together with oedema

or hypertension or both." There is no way of knowing which of these patients were actually suffering from pre-eclampsia as opposed to essential hypertension or chronic renal disease. McCartney² has shown by electron microscopic study of renal biopsies that the clinical diagnosis of pre-eclampsia is incorrect in primigravidae in 30% and in multiparae in 86% of cases.

Drs. Wardle and Menon refer to cryofibrinogen as "... the early breakdown product of fibrinogen." Although the identity of this substance is not entirely clear, it is more likely to represent an aggregation of fibrin monomer, perhaps in association with molecular fragments of fibrinogen, and as such represents critical evidence of the action of thrombin on fibrinogen. They also state that "toxaemia is increased when there is uterine distension ... [for example, in] accidental haemorrhage." Undoubtedly they intend to imply that toxaemia is increased in incidence rather than severity. However, an association of "toxaemia" and accidental haemorrhage is seriously questioned. Hibbard,³ for example, has produced figures to show that accidental haemorrhage is no more frequent in pregnancies complicated by hypertensive disorders than in normal pregnancies. However, accidental haemorrhage frequently produces severe disseminated intravascular coagulation, often with proteinuria, and, in the absence of hypotension, can resemble pre-eclampsia.

Finally they state that "inhibition of the fibrinolytic system alone never causes fibrin deposition." It is usually unwise to use the word "never" in medicine, and there are many authorities who believe that coagulation is a continuous process in the normal individual, nicely balanced by appropriate fibrinolysis. If this is so, it is conceivable that profound inhibition of the fibrinolytic system could lead to unopposed and progressive intravascular coagulation.

The question of changes in coagulation/fibrinolysis in pregnancy and their relationship to the development of pre-eclampsia is a fascinating one. Further study, preceded by careful selection of cases, may well establish a definite relationship between aberrations in the coagulation and fibrinolytic systems and "toxaemia of pregnancy."—I am, etc.,

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Unnecessary X-rays?

SIR,—For several years, in fact nearly 20, both as a casualty officer and as a country general practitioner with access to x-ray facilities, I have felt that x-rays for traumatic injuries are requested far too often, and often only for legal reasons or because it is customary. This is not a complaint, particularly against my local colleagues and friends, but seems indicative of a slightly woolly approach many people have about fractures. Instead of asking themselves the rather obvious question, is this x-ray going to serve

any purpose in the event that it does or does not show a fracture? will this affect or alter the treatment? they request an x-ray at once.

I have three questions that I ask before requesting an x-ray: (1) If night has intervened before I have seen the patient, has sleep been disturbed by pain? I must admit that I have never seen somebody sleeping easily or without analgesics when he has a bony injury. (2) If the x-ray reveals a fracture will it alter my approach on treatment? And (3) If longitudinal pressure applied at both ends of the bone causes referred pain or not. I find this rule is invaluable if fractures of ribs are suspected. Pressure on the sternum simultaneously with pressure on the dorsal vertebrae will certainly produce a referred pain if there is fracture of a rib.

I am sure many other doctors also apply these kinds of criteria, and I have no desire to denigrate those who request x-ray examinations for every injury, but I feel that the work load on x-ray departments is enough without unnecessary demands being made, and our patients should not be subjected to potentially harmful procedures if avoidable.—I am, etc.,

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Vaccination against Whooping-cough

SIR,—You state in your leading article (8 November, p. 316) that the "vaccines used before 1967 have now been found to be inefficacious in protecting home contacts with *B. pertussis* strains of type 1, 3. . . ." Can this statement be justifiably applied to the Wellcome product on the evidence contained in the recently published report of the Public Health Service Whooping-cough Committee and Working Party (8 November, p. 329)? The report shows that only 9 of the 120 exposed fully immunized contacts reported to have had whooping-cough had received the Wellcome vaccine. The diagnosis rested on the report of "a member of the nursing or sometimes the medical staff of the health department" who visited the home about three weeks after the source patient had been reported and "obtained details of any further patients with paroxysmal coughs. The diagnosis of whooping-cough in these nine children (eight were under 5 years of age) appeared to be based solely on a nurse's (sometimes the doctor's) assessment of whether they had a paroxysmal cough. Although the paroxysmal coughs were bacteriologically shown to be due to *Bord. pertussis* in two-thirds of all exposed contacts in the survey no information is given on the bacteriological confirmation of the diagnosis in any of the nine children immunized with Wellcome vaccine. If the diagnosis of whooping-cough was wrongly made in only one of these nine children then the efficacy of the Wellcome vaccine was underestimated by a factor of 8%.

In view of the fact that the Wellcome vaccine has been marketed at a potency of not less than four international potency units per dose since 1964 in order to meet W.H.O. requirement, while until 1966 the British requirements were 2:1 international potency units, is it justifiable to use such flimsy evidence to cause such serious doubt on the efficacy of Wellcome vaccine? Your leading

article concedes that there is a difference between pre-1968 and current vaccines but does not concede that there were differences between the vaccines used before 1968 even though their method of manufacture and reactogenicity¹ differed.

In the absence of bacterial confirmation the diagnosis of whooping-cough is uncertain, to say the least of it, and the numbers involved are too small to provide a statistically significant comparison of the efficacy of the Wellcome vaccine against the other vaccines used in the trial.—I am, etc.,

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Anaesthesia in the Dark

SIR,—Whether or not anaesthesia in the dark is justifiable (15 November, p. 383) I would suggest as a result of personal experience in 210 myelographies that the most efficient monitoring system is a simple stethoscope with long tubing. One limb is connected to an Ayre's T-piece inserted into the respiratory circuit; the other to a diaphragm on the chest. A stretched rubber diaphragm made out of the finger of a surgical glove gives the best acoustic results. The listening-in, of course, must be continuous while the lights are out.

For blood pressure recording, the most satisfactory illumination has been the replacement of the metal dial in a 7 in. (17.8 cm.) Accoson aneroid manometer by a translucent perspex dial and the insertion behind the dial of two semicircular Beta lights (obtained from Saunders-Roe and Nuclear Enterprises Ltd.). The figures on the dial should be 2 mm. thick, 1 cm. high, and placed at intervals equivalent to 20 mm. Hg.—I am, etc.,

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Voluntary Sterilization

SIR,—Despite the excellent literature distributed by the Simon Population Trust it would seem from the Union's experience that some practitioners still do not appreciate that a man may remain fertile for several weeks or even months after vasoligation. The patient should continue to take contraceptive precautions until two consecutive negative sperm tests have been obtained. Neglect of this precaution may lead to unwanted pregnancies and possible litigation. According to the Simon Population Trust the seminal fluid should be examined not less than 12 weeks after the operation, and a further examination should be carried out about three weeks later. The patient should not be deemed to be sterile until two consecutive examinations have shown complete absence of spermatozoa. It cannot yet be stated with complete finality that spermatozoa will not reappear in the seminal fluid after two consecutive and negative sperm tests. But the chances of this happening, if they exist, are