implants inserted as far back as 1956) kindly allowed us to examine them; papers were read and discussed. "Irreparable damage" appeared to belong to the past.

I agree that intraocular implants have no place in the management of the usual run of refractive errors, assuming that appropriate spectacles or contact lenses are available. But the optical correction of aphakia—especially unilateral—is quite another story. Where young children are concerned—and perforating wounds involving the crystalline lens are unhappily not rare—the situation is further complicated by the rapid onset of amblyopia, almost invariably irreversible in later life. Neither spectacles nor contact lenses provide a satisfactory answer, so the insertion of an intraocular lens implant, plus temporary occlusion of the uninjured eye, is the only practicable means of preventing amblyopia and restoring binocularity—as I stressed in 1959¹ and has been stressed by me and others² ever since.—I am, etc.,

London W.1.

D. P. CHOYCE.

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Unconscious Patient's Airway

SIR,—May I comment on Dr. R. Snook's article "Resuscitation at Road Accidents" (8 November, p. 348)? Dr. Snook is to be congratulated on his article, but the omission of one essential point gives rise to some concern.

Stress has been correctly laid on the need for maintaining the airway, and an admirable technique described for passing an endotracheal tube. In some instances and under some circumstances it may not be possible to give the casualty the advantage of intubation. Such unconscious and semiconscious cases, especially if a maxillo-facial injury has been sustained, must be transferred to hospital in the optimum position—that is, in the semi-prone position with a pillow under the chest, the neck extended, the face rotated downwards, and the mouth open so that its contents—blood, mucus, broken teeth, bone,

dentures, foreign bodies, etc.—will tend to fall out rather than fall back and obstruct the airway. This position will reduce but may not preclude the need for suction and intubation.—I am, etc.,

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R. KERR GILBERT.

Control of Rabies

SIR,—I read with interest your leading article on the control of rabies (25 October, p. 185). It is unfortunate that rabies tends to be classed among the esoterica of medicine (even in countries where the prevalence warrants no such assumption) until it strikes uncomfortably close to home. Though a bite from a rabid dog may be the most frequent cause of the disease in man—recognized and proved—infected dogs are only the tip of the iceberg.

To give some idea of the complexity of the problem I will quote some figures for the U.S.A. for the year 1966.¹ Rabies was detected in 26 different animal hosts. Of the 4,198 laboratory-confirmed cases of rabies, wild species accounted for 2,946 (70%) of the cases and domestic animals for the remaining 1,251 (30%) cases. Skunks and foxes accounted for 2,386 cases (81%) of all rabies in wild life, or 57% of all animal rabies. Dogs were the fourth most infected species. Before 1960 dogs had the highest reported incidence of rabies.

Of the 1,251 cases of rabies in domestic animals in 1966, cattle were responsible for 519 (41%) of cases, dogs for 412 (33%), cats for 252 (20%), equines for 43 (4%), sheep and swine for 10 each (<2%), and goats for 5 (<1%). The 412 cases of dogs rabies represent only 9% of all animal rabies (as opposed to 30% ten years previously). One human death due to rabies occurred, a 10-year-old boy who had been bitten by a rabid skunk.

Rabies is fatal in man, but it is far from clear that this is so in all animal species. There is evidence that some animal species are more resistant to infection than others, and also vary in their excretion of the virus on their saliva.²

In addition to the challenge of its epidemiology there is also the further challenge to produce a more sophisticated vaccine than the one in present use.—I am, etc.,

Tehran, Iran.

K. KOOROS.

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Diagnosing Parotid Calculi

SIR,—Not infrequently patients present with a history of recurrent parotitis in whom a calculous aetiology is said to have been excluded by radiological examination. An intra-oral film has not been taken in such a high proportion of these cases that there is obviously a general lack of awareness among both surgeons and radiologists that such a film is often the only way of demonstrating a parotid calculus. As a result, the aetiology of many cases of recurrent parotitis is missed, and parotid calculi are thought to be more uncommon than they really are.

May I make the plea that intraoral films should be a routine part of the radiological investigation of recurrent parotitis in the same way that they are for recurrent swellings of the submandibular gland?—I am, etc.,

London W.1.

DAVID PATEY.

Hiatus Hernia and Reflux Oesophagitis

SIR,—Your leading article on hiatus hernia and reflux oesophagitis (4 October, p. 1) was a curious and sometimes irritating heterogeneity of opinion, tradition, and fact. It failed to point to the main trends in new discovery of these often unrelated conditions, left out the most important work of the past years in its references, and laboured us once again with concepts of which we should be making every effort to rid ourselves.

A leading article is surely meant to explain problems, often complex, in simple terms, and bring readers up to date with the subject.¹ You failed to mention Allison,^{2,3} who pioneered part of the whole modern concept of our understanding of these conditions, and did not refer to Barrett's independent work done at about the same time.⁴ Indeed, the general impression you give is that there is little to add to a review article entitled "Reflux Oesophagitis: Its Diagnosis and Treatment," which was written over 15 years ago.⁵ I would like to comment on some of the main issues:

(1) The mechanism of the sphincter is no longer in serious debate.⁶ It is so apparently weak because it is designed to work in positive pressure surroundings, and, granted this, needs only to contract to about one-sixth of its power to keep the lower oesophagus closed against reflux, so that its margin of safety is great. The oesophagus normally joins the stomach at right angles, as can be seen on an oblique view of any barium swallow; the "obliquity" of this entry angle is a ghost that should have been laid long ago. The diaphragmatic sling of itself is of no importance in this one-way valve.⁷ The phreno-oesophageal ligament is what holds the oesophagus below the diaphragm at its lower end, and, being elastic, allows the upward herniation to take place during vomiting with recoil to normal position immediately afterwards. The pre-aortic fascia may take part as well, though its elasticity has not been so clearly shown.⁸

(2) You completely ignore the quite startling results of a 20-year-long prospective study of the complex of disease—and it is a complex—opening up many new avenues far beyond the narrow confines of this leader, by Eddy Palmer,⁹ however upsetting they may be to our British blinkered view. An up-to-date review must surely refer to such a colossal study.

(3) The correlation between endoscopic oesophagitis and inflammation on biopsy is by no means as close as you suggest.¹⁰ It is an exercise of interest mainly to define the nature of a stricture, but is of much less value in defining reflux and less still inflammation.⁹

(4) In a series of 20 patients I studied personally, weight loss varied from 7 lb. (3.2 kg.) to 4 st. (25.6 kg.). Two only experienced relief in their symptoms, and the patient who lost 4 st. was not one of these. The dearly held and much-quoted concept of the value of weight reduction may just be a myth, representing the longing of a reluctant surgeon or an enthusiastic physician.

(5) Regarding hyperacidity and oesophagitis,^{11,12} it is well known that the most vicious and penetrating inflammation of the oesophagus follows partial or total gastrectomy,^{13,14} and the agent responsible is almost certainly bile salts.¹⁵ The ages at which oesophageal strictures are seen—and labelled benign—are precisely those

Correct position on stretcher to ensure that the tongue falls forwards and does not obstruct the airway.

Alternative position on stretcher during transport of casualty. The illustration in the circle shows the method of attaching bandages to prevent rolling of the head if the patient is unconscious.

(By permission of E. & S. Livingstone Ltd.)

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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East Pondoland,
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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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Newmarket General Hospital,
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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