stability for the articulation. The unique design of the Fenlin prosthesis appears to satisfy all of the complex requirements of a rotator cuff replacement.

It will be recalled that the anatomical musculocutaneous cuff functions not only to stabilize the glenohumeral joint, while allowing it great freedom of motion, but also to fix the fulcrum against which the deltoid can contract and elevate the humerus with power.² The hemiplegic's paralysis of the rotator cuff provokes not only painful subluxation of the joint but also loss of the ability to fix the glenohumeral fulcrum. The deltoid, then, is unable to elevate the humerus and merely shrugs the shoulder. In the hemiplegic who gradually regains muscle strength in the upper extremity the elongated rotator cuff does not decrease to its normal size. Hence shoulder pain continues and is usually accompanied by weakness in elevation of the humerus.

In the Fenlin design a large ball is attached to the scapula, while the socket is secured to the humerus. The geometry of the large ball diminishes the magnitude of mechanical force necessary for the deltoid to initiate elevation. Also, the implant is designed with an inherently stable fulcrum so that the deltoid can elevate the humerus without assistance from the rotator cuff. The positions of the ball and socket are reversed from their normal anatomical location to diminish the potential for dislocation. The other novel feature of this implant is a robust scapular anchor that greatly reduces the likelihood of loosening of the component. Early clinical experience with the Fenlin prosthesis is encouraging. Further trials in hemiplegics appear to be indicated.—I am, etc.,

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¹ Fenlin, J. M. jun., *Orthopedic Clinics of North America*, 1975, **6**, 565.

² Inman, V. T., Saunders, J. B., and Abbott, L. C., *Journal of Bone and Joint Surgery*, 1944, **26**, 1.

Corticosteroids in Treatment of Anaphylaxis

SIR,—The excellent paper by Dr. P. Davies and others (24 May, p. 434) on the value and complications of intravenous pyelography raises an important point in suggesting that patients who are thought to be at risk for anaphylaxis due to possible iodine sensitivity should be given steroid cover. In the absence of a controlled clinical trial of the effect of steroids in acute anaphylaxis it would seem that the commonly held belief that they are the mainstay of the treatment of anaphylaxis is ill-founded. Prophylactic steroid treatment is unlikely to offer a significant degree of protection against acute anaphylaxis mediated by IgE in man. If there is any effect it is likely to be delayed, as in the rat, necessitating pretreatment with a single large dose 12-24 h ahead of the risky procedure. If the patient has type III hypersensitivity steroids could be expected to give complete protection, but this mechanism by itself is unlikely to produce the sudden dramatic emergency with which we are concerned. In patients with dual sensitivity the initial reaction will be no less dangerous, but subsequent management will be easier if steroids have been given.

Though glucocorticoids are unlikely to modify the anaphylactic reaction itself, a very high dose directly reduces capillary permeability and may help to sustain the blood pressure. Here one is seeking the effect which, for instance, is also useful in the treatment of cerebral oedema. For this role a water-soluble phosphate should be activated in the tissues more readily than the hemisuccinate of hydrocortisone, and, since they penetrate tissues much faster than other preparations, dexamethasone or betamethasone phosphates are the best choices. A reasonable dose of either would be 80 mg intravenously. An equivalent dose of hydrocortisone hemisuccinate would be 2.5 g.

Adrenaline, with its blend of α and β sympathetic effects, will help to antagonize the initial catastrophic loss of blood pressure while at the same time antagonizing the intracellular consequences of anaphylaxis. Whatever the risk to the heart, when faced with profound anaphylactic collapse most doctors give adrenaline intravenously, using 0.5 ml of 1/1000 solution, diluted if possible, given slowly. Their choice is almost certainly correct, since in profound shock a peripheral injection would never reach the rest of the body. Perhaps in the future intravenous salbutamol will be judged superior. For the second injection it is logical to give intravenous aminophylline, which potentiates the intracellular β sympathetic effect. Only the third injection should be steroid.—I am, etc.,

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Perforation of Small Intestine and Slow-K

SIR,—While not discounting the possibility that Slow-K may cause small-intestinal ulceration and perforation, I feel that this preparation should not be incriminated on the evidence presented by Dr. M. A. Farquharson-Roberts and his colleagues (26 July, p. 206) and that other aetiological factors should be considered—namely, cardiovascular disease.

Of the large number of causes of small-intestinal ulceration, cardiovascular disease (arteriosclerosis and hypertension) appears to be the commonest, probably producing the damage by localized gut ischaemia.^{1,4} Thus the long history of hypertension and multiple strokes in the 57-year-old patient reported may have been of greater significance than the administration of Slow-K.

I know of no specific histological features associated with iatrogenic ulceration, and histological differentiation of ulceration due to potassium chloride medication from ulceration due to other causes has been found impossible.³ Therefore further details of the "almost certainly iatrogenic" histological picture would be required before the conclusions reached could be accepted.—I am, etc.,

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¹ Teicher, I., et al., *Surgery, Gynecology and Obstetrics*, 1963, **116**, 196.

² Alexander, H. C., and Schwartz, G. F., *Gastroenterology*, 1966, **50**, 224.

³ Wayne, D. M., and Helwig, E. B., *American Journal of Clinical Pathology*, 1968, **49**, 26.

⁴ Carlson, H. C., *Radiologic Clinics of North America*, 1969, **7**, 97.

Pyridoxine and Oestrogen-induced Glucose Intolerance

SIR,—It has been suggested that women using contraceptives should take a pyridoxine supplement to prevent oestrogen-induced changes in tryptophan metabolism.¹ These changes include the production of increased amounts of an intermediate, xanthurenic acid, after a tryptophan load. This compound has been claimed to produce diabetes due to the formation of an inactive complex with insulin.²

The possibility that xanthurenic acid is responsible for oral contraceptive-induced glucose intolerance and gestational diabetes has been examined clinically. The reduction by pyridoxine of excessive xanthurenic acid excretion after a tryptophan load has been associated with an improvement in glucose tolerance.³⁻⁵ However, we do not believe that the two are causally related, as has been suggested, but rather that the beneficial effects of pyridoxine are possibly due to normalization of other tryptophan metabolites. Some women suffer from oestrogen-induced glucose intolerance independent of any abnormalities in xanthurenic acid excretion. Furthermore, we have found that the administration of xanthurenic acid (Sigma, Grade A, 99% pure) 120 mg/kg intraperitoneally in ammoniated saline failed to alter blood glucose levels in male Sprague Dawley or Wistar rats. We repeated the experiments using the special high fat diet which had been used in the original studies on the diabetogenic effects of xanthurenic acid.⁶ Once again xanthurenic acid was found to be inactive (see fig.) In further experiments it was found that xanthurenic acid did not impair glucose tolerance when given one hour before an oral glucose load (3 g/kg given as a 50% w/v solution). In a single experiment in a male albino rabbit xanthurenic acid 100 mg/kg given intravenously did not alter blood glucose concentrations over a period of three hours.

Effect of intraperitoneal injection of xanthurenic acid on blood glucose concentrations in male Wistar rats. Blood samples (0.1 ml) were obtained by heart puncture under pentobarbitone anaesthesia. Results reported by Kotake⁶ are also indicated.

Our failure to demonstrate any diabetogenic effect of xanthurenic acid could not be due to contamination with the 8-methyl ether of xanthurenic acid, which has been reported to be antidiabetogenic,⁷ since the sample of xanthurenic acid used was found