differing from my own and I hope you will allow me space in your columns to expand on the subject.

To start with, I agree that it is convenient to divide neurological complications of rheumatoid arthritis into those mediated through systemic features and those caused by local pressure, but when this is done the carpal tunnel syndrome falls into the same group as atlantoaxial subluxation (AAS).

More important than this quibble is to differentiate anteroposterior AAS from vertical AAS, which your recent leading article does not discuss although it was mentioned in its predecessor.¹ The former occurs with merely ligamentous damage whereas the latter, more serious, condition is permitted only when severe and destructive erosive changes have occurred involving the joints of the lateral articular masses of the atlas.² Vertical subluxation is much less common,³ but the neurological sequelae are more frequent.⁴ It is a misconception that rupture of the transverse ligament of the atlas alone allows significant anteroposterior AAS and it has been shown by section of this ligament in the cadaver that only slightly increased mobility occurs.⁵ Clearly the rheumatoid involvement is more widespread and affects other stabilizing ligaments of the atlantoaxial joint. Another and potentially dangerous belief is that the absence of radiological evidence of AAS implies a strong and stable situation and safety for the patient. I feel sure that this cannot be the case and that the rate of subluxation is important as well as its extent. A "last-straw" effect must exist in which a relatively trivial movement causes sudden severe subluxation. Figures for the incidence of vertical AAS are difficult to find, but my own survey³ suggests a figure of 5% when the diagnosis requires the tip of the odontoid process to be above the "foramen magnum line."

Estimates of the incidence of neurological sequelae of AAS must, I agree, depend on many factors. My own survey³ indicated that convincing long-tract signs occurred initially in approximately one-sixth of patients with either variety of AAS, but at the five-year follow-up⁴ the vertical AAS had shown itself as being the more serious, no less than 50% of the patients having an extensor plantar response—detected by a rheumatologist.

Turning to the physical signs, I again find points worthy of comment. The Sharp and Purser test mentioned is not the only physical sign of "specific" diagnostic value and my own attempts at using it produced both false-positive and false-negative results. More direct, and in my hands rather less unreliable, was direct palpation of the posterior pharyngeal wall. However, the points to be made in this respect are that these signs relate only to the anteroposterior AAS, they detect only severe degrees of subluxation, and they are no substitute for the essential x-ray.

I have studied with Dr J Trevor Hughes, of the Radcliffe Infirmary, Oxford, several patients with rheumatoid cervical myelopathy. In our studies a mechanism of interest was compression of the anterior spinal artery at the atlantoaxial and/or C4-5 level, leading to cord ischaemia. In one such case we found no evidence of radicular artery impairment. Preliminary studies have been reported,⁶ but the work is still in progress.

In mentioning the relatively benign natural history of the condition your article quotes a paper which did not set out to contain a follow-up study. It might have been preferable

to have used the three prospective follow-up studies available.^{4, 7, 8} Having made this comment I agree with the principles of management described and perhaps may be allowed to make a final few comments relating to conservative management. (1) An abnormal x-ray appearance alone should not be treated except by advice and observation. (2) Some collars are uncomfortable and useless. (3) Others may be potentially harmful; they should all be critically assessed. (4) A neurosurgical opinion should not be delayed until the patient is a poor operative risk.

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¹ *British Medical Journal*, 1973, **1**, 759.

² Swinson, D R, et al, *Annals of the Rheumatic Diseases*, 1972, **31**, 359.

³ Mathews, J A, *Annals of the Rheumatic Diseases*, 1969, **3**, 260.

⁴ Mathews, J A, *Annals of the Rheumatic Diseases*, 1974, **6**, 526.

⁵ Zinn, W. Personal communication.

⁶ Mathews, J A, *Scandinavian Journal of Rheumatology*, 1975, **4**, suppl 8, p 64.

⁷ Isdale, I C, and Conlon, P W, *Annals of the Rheumatic Diseases*, 1971, **30**, 387.

⁸ Smith, P H, Benn, R T, and Sharp, J, *Annals of the Rheumatic Diseases*, 1972, **31**, 431.

Oral irritation with gentian violet

SIR,—I was interested to read the letter from Dr P Horsfield and his colleagues (28 August, p 529) describing gentian violet causing primary irritation and ulceration in six babies with oral candidosis. Gentian violet is not harmless and similar cases have been described,¹⁻³ while a warning regarding mucous membrane ulceration is given in *Martindale*. Moreover, several patients have been observed with skin necrosis following application of 1% gentian violet in aqueous solution for fungus infections at various sites caused by candida and also dermatophytes.⁵

As Dr Horsfield and his colleagues indicate, adverse reactions to triphenylmethane dyes (gentian violet, brilliant green, etc) should not be mistaken for an exacerbation of the disease for which the treatment was originally prescribed. Mucosal use of gentian violet ($\frac{1}{2}$ -1% in aqueous solution) should be restricted to a maximum of four days with twice-daily applications only. It is difficult to explain why nystatin, presumably the suspension (100 000 U/ml), was not curative. However, amphotericin suspension (100 mg/ml) is an effective safe alternative to the first choice, nystatin.

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¹ Slotkowski, E L, *Journal of Pediatrics*, 1957, **51**, 652.

² Slotkowski, E L, and Redondo, D, *American Journal of Diseases of Children*, 1966, **112**, 40.

³ John, R W, *British Medical Journal*, 1968, **1**, 157.

⁴ *Martindale: The Extra Pharmacopoeia*, ed N W Blacow, 26th edn, p 185. London, Pharmaceutical Press, 1972.

⁵ Björnberg, A, and Mobacken, H, *Acta Dermatovenereologica*, 1972, **52**, 55.

Hypoglycaemia in children undergoing operations

SIR,—The recent article on postoperative hypoglycaemia in small children by Drs Stephen Ware and J P Osborne (28 August, p 499) is a timely reminder of the continued existence of this too little appreciated problem.

One solution has been indicated¹ and more fully developed² and entails administration of oral maltose and metoclopramide preoperatively to the children. This work has been repeated with oral glucose replacing oral maltose, but while the average plasma glucose levels were raised, postoperative hypoglycaemia still occurred in a few children. I had no facilities for measuring insulin levels, but very probably the hypoglycaemia was a rebound phenomenon following hyperglycaemia induced by surgery and the ingestion of glucose, as shown by the insulin levels in several of the cases reported by Drs Ware and Osborne. The recommendation that preoperative starvation should last no more than four hours is sufficient³ but can be difficult when children are placed early on a morning operating list. But for these cases metoclopramide and maltose would seem suitable and effective.

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¹ Fry, E N S, and Ibrahim, A A, *British Medical Journal*, 1974, **3**, 808.

² Fry, E N S, and Ibrahim, A A, *Anaesthesia*, 1975, **30**, 548.

³ Thomas, D K M, *British Journal of Anaesthesia*, 1974, **46**, 66.

SIR,—Hypoglycaemia may occur in children undergoing surgical operations but is often asymptomatic and goes unrecognised. Nevertheless, an asymptomatic hypoglycaemic child may without warning have a convulsion, with consequent brain damage. The paper by Drs Stephen Ware and J P Osborne (28 August, p 499) shows that hypoglycaemia at operation is still a very real risk in children. They report a 10% incidence of hypoglycaemia in the postoperative period, which is a low value compared with other reported studies. Bevan and Burn¹ found that 30% of children starved for operation were hypoglycaemic (blood glucose <3.3 mmol/l (60 mg/100 ml)) at the time of induction of anaesthesia and Kelnar² reported that 50% of children undergoing adenotonsillectomy were hypoglycaemic (blood glucose <2.78 mmol/l (50 mg/100 ml)). Thomas³ studied the effects of shortening the conventional eight-hour preoperative fasting period to four hours. He reported that there was no significant difference between the two groups in the volume or pH of the gastric aspirates just after induction. However, the mean blood glucose level was significantly lower in those children fasted for eight hours, being 2.96 mmol/l (53.3 mg/100 ml) compared with 3.69 mmol/l (66.4 mg/100 ml) in those fasted for only four hours. No hypoglycaemia was seen in the four-hour group, but it occurred in 15% of the eight-hour group, particularly in the very young and small.

The hazard of inhalation of vomit on induction of anaesthesia must be balanced against the dangers of hypoglycaemia produced by prolonged starvation in children. It would seem from Thomas's results that a four-hour preoperative fast is adequate and that prolonging it to eight hours only increases the incidence of hypoglycaemia. Fry and Ibrahim⁴ have suggested the use of metoclopramide to shorten the preoperative fast to one hour. The risks of hypoglycaemia may also be reduced by carefully planning operation lists so that no child is fasted longer than is necessary, the adequate use of intravenous dextrose before and during surgery, and the introduction of