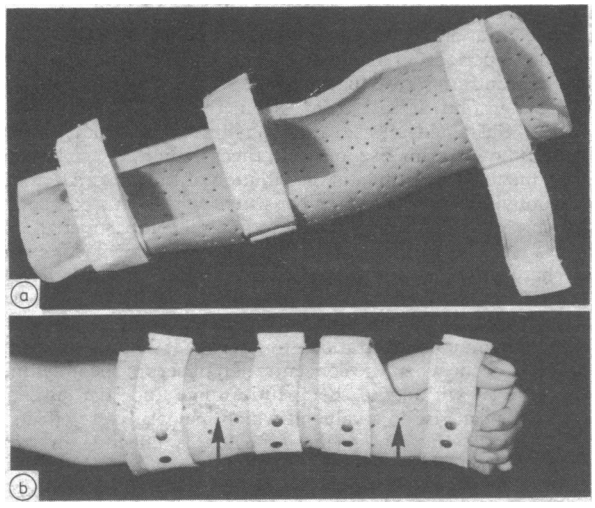

A: Elbow splint. B: Left wrist splint with reinforcing plastic strip arrowed.

The cannula puncture site was covered with a small dressing and the drip tubing held in place with the usual Elastoplast strips.

The splints were used by 100 consecutive patients recruited for a trial to investigate infusion thrombophlebitis.[1] They were enthusiastically accepted by the patients and nurses. The absence of an all-embracing bandage permitted easy inspection of the drip site.

Four splints were made for the investigation. No complications attributable to the splint were encountered, and none of the splints wore out. They were cleaned with a cloth moistened with cetrimide.

Comment

Moulded Plastazote splints are a comfortable and cheap alternative to the traditional board in intravenous infusions.

[1] Woodhouse CRJ. Movelat in the prophylaxis of infusion thrombophlebitis. *Br Med J* 1979;i:454-5.

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North Middlesex Hospital, London N18

C R J WOODHOUSE, MB, FRCS, surgical registrar (present appointment: consultant urologist, Royal Marsden Hospital, London SW3, and senior lecturer, Institute of Urology, London WC2)

Domperidone in the prevention of complete classical migraine

Clinically, migraine is usually associated with a disturbance of various autonomic functions. Objective measurements have suggested that gastric emptying is delayed during migraine attacks.[1,2] Although studies of the autonomic nervous system during the prodromal period have not been performed an autonomic malfunction could possibly precipitate the migraine. Furthermore, since manifold interactions occur within the autonomic nervous system, correcting one defective function could possibly restore the autonomic balance and thus perhaps prevent an attack. In patients with complete migraine physical or psychic changes or both are experienced some 24 hours before an attack.[3] In an open pilot study I found that the attack was prevented when patients were given a high dose (30 mg) of domperidone immediately the warning signals occurred. Domperidone is a peripheral dopamine-receptor antagonist with antiemetic and gastrokinetic properties.[4] The present double-blind study was undertaken to confirm this observation.

Patients, methods, and results

I studied 19 patients with classical migraine, all of whom experienced warning phenomena (primarily sensory or psychic intolerance) which occurred from seven to 48 (median, 24) hours before a severe to incapacitating headache. Each patient was studied during four consecutive (imminent) attacks, two of which were treated prophylactically with domperidone and two with a placebo under double-blind randomised conditions. The patients were given either three 10 mg domperidone tablets or three matching placebo tablets as soon as they became aware of the warning symptoms.

No aura or headache was experienced in 25 out of the 38 (66%) attacks when domperidone was taken and in only two out of the 38 (5%) after the placebo (table). Ten patients, in whom the placebo failed twice to prevent an attack, had no attack on two occasions when treated with domperidone. In all but one patient the attacks were as severe as usual when treated prophylactically with the placebo. The effects of domperidone were not related to either the type of warning symptoms or to the time lag before the actual attack. There were no side effects.

Number of patients with complete classical migraine experiencing attacks after prophylactic treatment with domperidone 30 mg and placebo (n=19)

No of attacks after administration of: Placebo	Domperidone 0	1	2
0	1	0	0
1	0	0	0
2	10	3	5

Statistically significant difference between drug and placebo, $p<0.001$ (Wilcoxon's matched-pairs signed ranks test).

Comment

I know of no investigators who have tried to prevent migraine attacks in patients with early warning phenomena. The present findings show, however, that this can be achieved in most cases with a relatively high dose of domperidone. How domperidone acts in this condition is nevertheless not known. Since it does not cross the blood-brain barrier[4] its effects are unlikely to be mediated by the central nervous system, unless patients with migraine have a defective barrier.[5] As it is a potent dompamine-antagonist domperidone would, however, provoke extrapyramidal symptoms if the blood-brain barrier was defective; no such problems have been reported in patients with migraine. Thus, domperidone probably does not act on the central nervous system, which is often thought to play a part in the pathogenesis of migraine. Whatever the mechanism domperidone, paradoxically, prevents migraine attacks when it is taken up to 48 hours before the attack even though its duration of action is about six hours. Thus, thorough investigation of patients with complete migraine during the day before an attack should give more information on the pathogenesis of the disease.

[1] Volans GN. The effect of metoclopramide on the absorption of effervescent aspirin in migraine. *Br J Clin Pharmacol* 1975;**2**:57-63.

[2] Wilkinson M. The treatment of acute migraine attacks. *Headache* 1976; **15**:291-2.

[3] Blau JN. Migraine prodromes separated from the aura: complete migraine. *Br Med J* 1980;**281**:658-60.

[4] Van Nueten JM, Schuurkes JAJ. Animal pharmacology of domperidone, anti-emetic and gastrokinetic properties. In: Towse G, ed. *Progress with domperidone, a gastrokinetic and anti-emetic agent*. London: Royal Society of Medicine, 1981:21-7.

[5] Harper AM, McCulloch J, MacKenzie ET, Pickard JD. Migraine and the blood-brain barrier. *Lancet* 1977;i:1034-6.

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Populierenlaan 1, B-3620 Lanaken, Belgium

J WAELKENS, MD, general practitioner