(extra 2) and Tianchong (G9). These points are located in the depression 2.5 cm posterior to the mid-point between the lateral end of the eyebrow and the outer canthus, and 2.5 cm superior and posterior to the pinna of the ear. Very precise localization is not required, and identification of the points may be assisted by a history of local tenderness during an attack. A single needle is used and the points are stimulated one after the other. The skin is first cleaned with alcohol and massaged briefly to reduce the pain of insertion. After placement, the needle is rotated backwards and forwards for about 30 seconds and then withdrawn. The point is finally massaged firmly with the tip of a finger for a few seconds.

Four to six weekly treatments will be required before the maximum benefit takes place. Further treatments can then be administered if the symptoms start to recur. The patient can treat himself by lightly scarifying the skin with the point of a sterile needle, or even by firm massage with the thumb nail. This is often effective in aborting an actual migraine episode if carried out in the early stages.

Over a period of about ten years' clinical use I have found this technique most effective and, so far, completely free of side effects. It has considerable advantages in comparison with treatment by drugs, which are expensive, often ineffective and sometimes seriously hazardous. The treatment is admittedly bizarre, and there is no obvious reason why it should succeed. However, conversation with other medical practitioners who use acupuncture, as well as an increasing general acceptance that acupuncture has a contribution to make to Western medicine, have made me feel that I should write about this in the hope that my experiences would be confirmed by a broader sample of doctors and patients – perhaps under placebo controlled conditions.

Yours faithfully

PAUL MARCUS

6 September 1983

Cramp in pregnancy

From Dr Robert Newill
London W1

Dear Sir, At the time when my wife developed pregnancy cramps, I learnt about their treatment with soda mint tablets. These proved extremely successful in my wife and in several other pregnant women who suffered the same complaint.

Unlike riboflavine (Dr Alan Morgan, August *Journal*, p 712), soda mint tablets are still obtainable from chemists, are very cheap and the dosage is six tablets at bedtime. The rationale seems to be that night cramps in pregnancy are due to a build up of lactic acid in the leg muscles, and bicarbonate in the blood neutralizes this. It is certainly worth trying.

Yours sincerely

ROBERT NEWILL

3 September 1983

Organophosphates and *torsade de pointes* ventricular tachycardia

From Dr Z Kiss and Dr T Fazekas
First Department of Medicine, Szeged University Medical School, Szeged, Hungary

Sir, Recently, Ludomirsky *et al.* (1982) published a paper on Q-T interval prolongation with ventricular tachycardias of *torsade de pointes* type observed in 6 cases of acute organophosphorus poisoning (OP). Out of 15 patients with OP, Q-T prolongation was recorded in 14.

To the best of our knowledge, our reports on the largest series of patients were the first in the literature, and stressed the high incidence of arrhythmias and repolarization abnormalities associated with OP (Kiss & Fazekas 1978*a,b*, 1979, 1982*a,b*). Toxic Q-T prolongation, ST segment and T wave anomalies were observed, together with various forms of arrhythmias (56 patients), in our 134 cases poisoned with organophosphate pesticides. Repetitive ventricular tachycardia with *torsade de pointes* phenomenon was seen in 7 patients (Fazekas & Kiss 1979). An example is shown in Figure 1. The tachycardia is initiated by a ventricular extrasystole with 'R-on-T' phenomenon. The toxic repolarization anomaly is seen in the first and second complexes (Q-T = 0.54″, Q-T_c = 35″ after Holzmann-Hegglin). After several periodical reversals of QRS electrical polarity interrupted by multiform ventricular complexes, a spontaneous termination of the episode of the *torsade de pointes* tachycardia with alternating toxic repolarization follows (continuous automatic Cardalarm-Siemens rhythm strip, paper speed 25 mm/s).

In experiments on dogs, cardiac arrhythmias were induced by intravenous administration of the organophosphate pesticide mevinphos (Phosdrin, Shell Co) (Kiss & Fazekas 1978*a*, 1981). We have also reported a case of congestive cardiomyopathy caused by *long-term* organophosphate exposure (Fazekas & Kiss 1980).

Consideration has also been given to therapy (Kiss & Fazekas 1981). Isoproterenol was effective in 3 patients (electrical pacing in 2) via shortening of Q-T prolongation. As also reported by Ludomirsky *et al.* (1982), we found lignocaine to be ineffective and in some cases ventricular fibrillation developed during its administration.

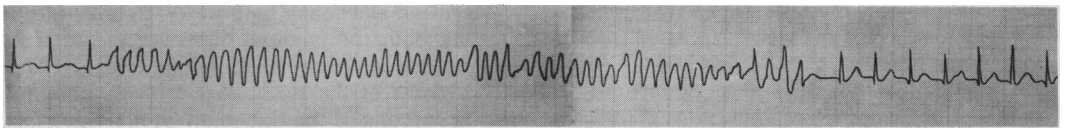

Figure 1. ECG tracing showing *torsade de pointes* phenomenon

In some cases ventricular premature contractions in human organophosphate pesticide poisoning were successfully eliminated with intravenous magnesium sulphate (unpublished data). Although the exact mechanism is not yet known, the magnesium probably counteracts the direct toxic inhibitory effect of organophosphates on Na^+–K^+–ATPase. It may reactivate the membrane Na^+–K^+–ATPase (Shine 1979). Arrhythmias were aggravated by large doses of atropine (from 100 to some thousand milligrams daily), and we therefore recommend that the daily dose should be under 100 mg. It is well known that atropine may be arrhythmogenic not only in toxic but also in ischaemic conditions (Cooper & Abinader 1979).

Amongst our patients, the greatest number of fatal arrhythmias were observed some days after organophosphate exposure, when the patients' toxic clinical symptoms and signs were moderate or absent. Focal myocardial damage (pericapillar haemorrhage, micronecrosis, patchy fibrosis) was demonstrated histologically (Kiss & Fazekas 1982*a*, 1983). These focal areas interspersed with normal myocardium can produce many potential areas of re-entry and inhomogeneity of repolarization, respectively. We suppose that toxic loss of intracellular potassium, extracellular hyperkalaemia and the consecutive partial depolarization may lead to slow response activation in the myocardium with periodical reversal of QRS electrical polarity, i.e. *torsade de pointes* phenomenon. There may be present two (Neumann *et al.* 1982) or several ectopic foci, depending on the actual polarized state of a given region of the toxically-injured myocardium. For these focal changes, critical regional delay or activation wavefront and consecutive macro re-entry are postulated. Secondary sympathetic overactivation due to acetylcholine excess and atropine treatment (because of acetylcholine-induced firing of non-inhibited nicotinic receptors) may be additive factors in arrhythmogenesis in OP.

In conclusion, we would like to stress the high incidence of potentially life-threatening arrhythmias during the late course of OP. The continuous ECG monitoring of patients with OP during the acute and subacute phase should be considered because of organophosphate cardiotoxicity and arrhythmogeneity. As to the efficacy of magnesium sulphate in stopping *torsade de pointes* ventricular tachycardia, further investigations are needed, though there are case reports on its antiarrhythmic effect in magnesium deficiency states (Loeb *et al.* 1968, Levine *et al.* 1982, Dyckner & Wester 1982). Its role in ischaemic heart disease is discussed by Singh *et al.* (1981).

Z KISS
T FAZEKAS
7 July 1983

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Vestibular function tests in children

From Dr S E Snashall
Audiological Department
Farnham Road Hospital, Guildford, Surrey

Dear Sir, The paper under the above title, which you kindly published in the July issue of the *Journal* (p 555), had one important omission which I would welcome the opportunity of correcting.

When the paper was originally presented to the Society on 4 March, it was emphasized that vestibular function tests in children are best performed by staff who have extensive experience of these procedures and also in handling children.