Until recently our sister department in the Western General Hospital operated a 12 channel SMAC II system; neither uric acid concentration nor creatine kinase activity was included in its configuration. Thus SMAC configurations differ.

Doctors should specify the measurements that they wish to have performed or that they recommend should be performed. It is not satisfactory to write SMAC on request forms or to describe the results of investigations by saying, for example, that "the patient's SMAC was normal." Such uses of the abbreviation should be actively discouraged, especially by those concerned in medical education as they make no allowance for the various SMAC configurations, nor for the fact that periodically one or more channels of the machine system may be "down." The "SMAC mentality" encourages an uncritical approach to requesting investigations, a practice so eloquently deplored by the late Dr Richard Asher.²

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SIR,—In their review of the dystonias Professor C D Marsden and Dr N P Quinn try to give an encouraging picture of the treatments now available.¹ It is clear, however, that there is only marginal help for many sufferers and that all avenues still need to be explored.

The authors make strong claims for an exclusively organic aetiology for all of the dystonias that seem at best to be only partially substantiated. While stating that "the manifestations of dystonia . . . are due to brain disease not psychopathology," they confirm that 90% of focal dystonias remain idiopathic. There are apparently no abnormalities that can be shown consistently by computed tomography, nuclear magnetic resonance, or histopathological or biochemical investigations. The claim that abnormal electrophysiological studies "have shown the various forms of dystonia to have a true neurological substrate; they can no longer be considered 'psychogenic' conditions," is surely contentious. Malmö *et al* showed that angry subjects have an isolated focal increase of muscle tone in their arms on electromyography.²

There is evidence that psychological factors play an important part in focal dystonia. Crisp and Moldofsky described seven patients with writers' cramp as tense, striving, sensitive, conscientious, precise, and emotionally overcontrolled people who seemed to have an unresolved ambivalence over writing in their occupations.³ Cottraux *et al* said of their 15 patients with writers' cramp that "all complained of vocational problems, overwork, or conflicts with hierarchy."⁴

Psychological treatments have been used successfully in the management of focal dystonias. Behavioural treatments were first proposed in 1925.⁵ In 1960 Sylvester and Liversedge claimed that 29 out of 39 patients were still improved four years after being treated with a pen that delivered an electric shock when gripped too tightly.⁶ Five years later significant improvement was claimed in six out of seven patients treated with interpretative psychotherapy, relaxation, and desensitisation techniques. More recently Cottraux *et al*, using a combination of electromyography feedback, relaxation, assertion training, and desensitisation, reported that nine out of 15 patients were significantly improved at follow up.⁴ Greenberg and Marks showed substantial improvements in all four of their patients treated with habit reversal.⁷ Finally, in 1988 a trial of 23 subjects with writers' cramp showed no difference between those treated with habit reversal and those treated with relaxa-

tion, but significant improvements were recorded in both groups.⁸

There is a good case to be made for a multifactorial aetiology in the focal dystonias. Basal ganglia disturbance, personality traits, unconscious conflicts, and social stress factors may all play a part. It is too early for psychiatrists to return to their tents. Behavioural treatments are cheap, simple, and non-invasive methods that patients may prefer to drug treatment or injections of botulinum toxin. In addition, learning theory might provide an explanation for the bizarre "task specific" nature of many focal dystonias.

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Self injection treatment for impotence

SIR,—Drs L Q Robinson and T P Stephenson have drawn attention to a potentially fatal error in the dispensing of papaverine.¹ Intracavernosal autoinjection of vasoactive drugs is becoming increasingly used for erectile impotence of various causes. As more patients become established on long term treatment its prescription by general practitioners is likely to continue to increase. This treatment has been in use at our sexual problems clinic for nearly five years. Two almost identical cases of incorrect dispensing have occurred in our patients, both within the past 12 months.

The first patient injected himself with 40 mg of papaveretum intracavernosally after incorrect dispensing by his chemist. The prescription was correctly written by his general practitioner. Fortunately he did not lose consciousness, but his partner was somewhat surprised to find that far from getting an erection he became very sleepy and completely lost interest in sex.

In our second case the patient was aware that something was wrong when the chemist immediately dispensed the prescription, despite my having told him that it would need to be ordered and would take two to three weeks to become available. He discovered the mistake on checking the writing on the box.

In neither of these cases was the prescription itself incorrectly or illegibly written, nor was it written in the format required for prescribing controlled drugs,² of which papaveretum is an example. The fact that two units have each reported two such errors suggests that there is a significant risk of this error occurring. We now give an oral and written warning to all of our patients who are to receive prescriptions from their general practitioner that they should check that the drug dispensed is correct.

The lack of familiarity with intracavernosal autoinjection vasoactive drugs that led to these errors occurs among doctors as well as pharmacists. This has led to problems in two of my patients who

were unfortunate enough to develop priapism after autoinjection. The treatment for papaverine induced iatrogenic priapism described by Brindley³ is simple, quick, and effective. These two patients were unable to attend this hospital for treatment of their priapism and attended their local accident and emergency department. Because this complication is uncommon it is understandable that some doctors might be unaware of its correct treatment. In these two cases there was a considerable delay (12 and 24 hours) before detumescence was achieved. Fortunately there were no long term complications.

It is impossible to insist that patients remain close to the clinic when using intracavernosal autoinjection vasoactive drugs. We therefore give a handout to our patients containing a detailed set of instructions for doctors on the detumescence technique. Patients can thus, if necessary, present at a local accident and emergency department with this handout. Patients wishing to use the drugs on holidays in foreign countries should consider carefully the availability of adequate medical care and language problems in understanding the handout.

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Treatment of subdural haematoma

SIR,—The report by Drs T J Anderson and I M Donaldson recommending conservative treatment of an intracranial haematoma in a woman receiving anticoagulant treatment¹ was, I believe, unhelpful. Treating an intracranial haematoma conservatively is not remarkable. What is difficult is deciding which haematomas require evacuation and which are safe to observe. The criteria for evacuating such a haematoma include a deteriorating level of consciousness, raised intracranial pressure, and appreciable midline shift. The single computed tomographic slice shows considerable midline shift with contralateral ventricular dilatation. It is almost certain that the intracranial pressure was raised, and clinical deterioration occurs in two thirds of such patients with such problems.² This deterioration may be rapid, particularly in patients who already have a reduced conscious level; and once deterioration has occurred evacuation of the haematoma produces satisfactory recovery in only half of the patients.

This risk should be compared with that of operation. The subdural haematoma described¹ was of mixed density, which is typical of those found in patients who have received anticoagulation. The fluid component could have been evacuated through a single burr hole with no significant risk of haemorrhage, even if the patient was fully anticoagulated. In cases in which craniotomy is required and anticoagulation has been reduced or stopped for a few days the risks of a major valve complication (embolus or valve thrombosis) in patients with a Starr Edwards valve are of the order of 10 per 1000 patient years or 1% per year.

Good results will be achieved consistently only if intracranial haematomas are evacuated before clinical deterioration. Ideally the decision to manage these haematomas conservatively should be made by neurosurgeons and the patients should be observed in a unit that is capable of evacuating the haematoma urgently. The woman reported on by Drs Anderson and Donaldson had a high risk