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Psychiatry in the Soviet Union

SIR,—Professor Georgi Morozov (6 July, p. 40) points to the fact that there is a good deal of difference in the way psychiatric diagnoses are made in different parts of the world and he is, of course, right. I suggested in my article (9 March, p. 433) that this explained in part why many British psychiatrists could not accept Soviet practice in respect of political dissenters who are regarded as insane. There were, however, other reasons. For example, few of the dissenters would be regarded in Britain as having committed any serious crime, so that the question of "responsibility" would not arise. Moreover, except in the case of gross abnormalities immediately obvious to a layman, when the accused person may be ruled "unfit to plead," in a British court it is only the *defence* that enters a plea of insanity. I mentioned a number of other apparent disadvantages of Soviet legal procedures in these cases.

However, Professor Morozov's main point was that I had not indicated the favourable outcome of the meeting between western and Soviet psychiatrists at the Serbsky Institute on 15 October 1973. In fact, of the 13 westerners who attended, 12 had first signed a statement containing two main principles: firstly, that the visit could not in itself be held to imply acceptance or rejection of any of the allegations and, secondly, that such difficult and complex problems could not be adequately evaluated in one day. We wished to begin what it was hoped would be continuing discussions. General statements are therefore out of order. Nevertheless, I have no doubt that many of my colleagues were not satisfied, as I myself was not, that all five political dissenters whose cases we heard were so seriously mentally ill during the time of their legal examination as to be unfit to

plead, to conduct their own defence, or to instruct defence lawyers.

In addition to these five cases we asked to discuss the case of Dr. Zhores A. Medvedev (who has given me permission to mention his name). We were told that his compulsory admission to hospital was an error. This frank statement was, for me, one of the most encouraging features of our visit to the Serbsky Institute, since it indicated a willingness to admit mistakes which, if it were matched by an equivalent attitude on the part of western psychiatrists, could lead to constructive discussions. I may have missed a public announcement about an inquiry into this matter. If such an inquiry has been held it seems important that its conclusions should be published together with an account of the action that has been taken to ensure that such an error will not be repeated.

Finally, I should like to endorse Professor Morozov's plea for impartial discussion of these issues. As I suggested in my paper at Yerevan, accusations have been levelled at psychiatrists in many countries and we ought to consider them attentively in order to decide whether, after the stridency and exaggeration have been removed, there does not remain a kernel of truth. The Royal College of Psychiatrists, the American Psychiatric Association, and many other professional psychiatric bodies throughout the world have agreed to set up a committee to examine such issues. A main aim is to discover how best to arrive at a just and humane legal procedure which will spare people suffering from severe mental disorders from being held responsible for actions undertaken under the compulsion of forces they cannot withstand, while at the same time ensuring that those who deliberately and rationally choose to break

what they regard as morally indefensible conventions or laws should not be treated as insane. It is much to be hoped that the All-Union Society will join this endeavour. —I am, etc.,

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Autonomic Neuropathy and Autovagotomy

SIR,—Your leading article on diabetic autonomic neuropathy (6 July, p. 2) ignored gastric secretion. Achlorhydria^{1,2} and associated gastritis^{3,4} after an augmented histamine test are abnormally frequent in diabetics, especially those with a high fasting blood sugar with severe complications.

Vagal function has been studied in diabetics by comparing the peak acid output (P.A.O.) after a chemical stimulus of histamine, Histalog (Hg) (ametazone hydrochloride) or pentagastrin (Pg), and a vagal stimulus, intravenous insulin (I). P.A.O._{pg} was normal in a group of 10 diabetics (seven with neuropathy) but the mean P.A.O._i of these diabetics (19.8 mEq/hr) was significantly lower than in control subjects (33.5 mEq/hr).⁵ The ratio of acid output in response to insulin and to a maximum stimulus estimates the proportion of parietal cells which are vagally excitable.⁶ This ratio was significantly lower in these diabetics (49%) than in controls (87%). One of the 10 diabetics secreted no acid after insulin,⁵ just as if the patient had had a "complete" surgical vagotomy. In another group of 11 diabetics (eight with neuropathy) two failed to secrete acid after insulin.⁷ Evidence of autonomic neuropathy was not sought specifically in these Swedish patients.^{5,6}

In three diabetic patients with autonomic neuropathy (neurogenic bladder, negative Valsalva tests, and absent sweating) the

Sex	Age (Years)	Peak Acid Output (mEq/hr)		$\frac{\text{P.A.O. I}}{\text{P.A.O. Hg}} \times 100$
		Histalog	Insulin	
F	34	18.5	0.88	5
M	66	1.24	0	0
M	63	17.58	9.54	54

results of gastric secretion tests⁸ are shown in the table.

These results could be interpreted as showing absence of impairment of vagal innervation of the stomach in these three patients with peripheral and autonomic neuropathy. Kassander⁹ had originally suggested that "a diabetic patient at some point in the progression of his diabetes may 'vagotomize' himself." Myelin degeneration has been seen in the vagal nerves of diabetics as well as in their peripheral nerves.¹⁰ Taken together these histological abnormalities¹⁰ and the gastric secretory studies^{5, 7, 8} support this concept of the diabetic vagotomizing himself,⁹ analogous to achalasia of the oesophagus. In 32 patients with achalasia vagal nerve function was considered absent in eight because of failure of acid response to insulin but adequate response to maximum Histalog stimulation.¹¹ Autovagotomy should be considered as part of a spectrum of diabetic autonomic neuropathy, so that it is not surprising that the incidence of duodenal ulcer in diabetics is only one-third of that of the normal population.¹¹—I am, etc.,

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Death after E.C.T.

SIR,—In her report of the death of a 55-year-old woman following electric convulsion therapy (E.C.T.) Dr. Joan R. Gomez (6 July, p. 45) raises three points deserving consideration.

(1) The dangers of "cardiotoxic drugs" such as the tricyclic antidepressants used in conjunction with E.C.T. must be weighed against their advantages in preventing the fairly high incidence of early relapse (with risk of suicide) in patients treated with E.C.T. alone,^{1, 2} particularly as giving additional E.C.T. after symptomatic relief has not been shown to prevent relapse.² A number of studies do, however, suggest that concomitant tricyclic administration reduces the incidence of relapse.^{3, 4}

(2) Many investigators have commented unfavourably on the dosage and mode of atropine administration prior to E.C.T.⁵

Though Rich and Pitts,⁶ in a series of over 1,500 E.C.G.-monitored E.C.T.s, failed to demonstrate "anything approaching a life-threatening vagal-induced arrhythmia" using 0.65-2.5 mg atropine by the subcutaneous route, pharmacological evidence suggests that the commonly used dosage of atropine—that is, 0.65 mg—is of placebo value only in preventing vagal inhibition.

(3) Acute anxiety resulting in sympathetic/parasympathetic stimulation can undoubtedly be responsible for cardiac arrhythmias; some anaesthetists in general surgical and dental practice are using such compounds as lorazepam intramuscularly as sole premedication with satisfactory results. It should be remembered, however, that most currently available effective anxiolytics also have anticonvulsant properties which might be antagonistic to the convulsant stimulus.—I am etc.

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SIR—I was most interested by Dr. Joan R. Gomez's letter (6 July, p. 45) reporting the death of a woman apparently from vagal inhibition due to the combination of electric convulsion therapy (E.C.T.) and amitriptyline and by her suggestion that the standard premedicating dose of atropine (0.6 mg) is inadequate. After I had had a couple of nasty experiences (a cardiac arrest in a grossly obese man who was fortunately easily resuscitated and a profound bradycardia in a young, apparently healthy man) I made it my standard practice to give atropine 1.2 mg intramuscularly at least an hour before E.C.T. and with this regimen I found no further problems of this type, though I was averaging 50-60 E.C.T.s a week in a predominantly middle-aged and elderly population. Since peripheral circulation is often relatively impaired in patients under stress, it would be interesting to know by which route atropine was given to Dr. Gomez's patient and how long before the fatal E.C.T. I think one reason why the 0.6 mg dose remains standard is because nearly all of us, doctors and nurses alike, tend to think of "drying up secretions" as being the main reason for giving atropine. Actually, if one stops to think logically about it, it is obvious that if one passes an electric current through the brain all the cranial nerves will be overstimulated and therefore one should use a dose of atropine sufficient to paralyse the vagus in order to avoid cardiac arrest.

Regarding Dr. Gomez's last question, surely the usual effect of anxiety on the

heart is to produce a tachycardia, but it would be reasonable to suppose that this in itself may make the heart more vulnerable to further stimuli and also it is possible that the tachycardia might stimulate the vagus in an attempt at homeostasis. In any case, as the patient was 55 at the time of her re-admission it is probable that the E.C.T. she received for her puerperal illness would have been given without anaesthesia and so she may well have been far more apprehensive than are most patients prior to their first E.C.T.—I am, etc.,

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Tests of Hearing in School

SIR,—In your leading article (6 July, p. 3) doubt is expressed about school screen audiometric tests and it is suggested that perhaps impedance measurements for middle ear function could replace them. The article also gives incorrect information about the standard procedures.

School screen audiometry is carried out at 20-dB levels, which is now the generally accepted standard, and all frequencies from 250 to 8,000 c/sec should be tested. Children should be tested as soon as possible after entry into the infants' school and again at the junior stage, but it is desirable to test all children, if at all possible, three times during their stay in the primary school. The value of this test is amply confirmed by direct experience from areas where school screen audiometry is practised properly. Extensive literature is available which clearly shows the importance of school screen audiometry, which is one of the most valuable methods of detecting children with previously unknown hearing losses, both conductive and sensorineural. Without this test many children with hearing losses would remain undetected.

From time to time, in order to check the results of school screen audiometry, groups of children who have failed the screen test and subsequent threshold audiometry test are examined by me at audiology clinics. Recently, in one group of 36 children I found 21 with a conductive loss and two with a sensorineural one; four had wax in their ears which needed syringing and nine had normal hearing. Of those with conductive loss, only three had already had E.N.T. treatment; six were referred by us for treatment for serous otitis media; seven needed further investigation and almost all of them needed some help and were visited at the school by a peripatetic teacher of the deaf. Of the two with a sensorineural hearing loss, one had a moderate high frequency one and one almost total unilateral deafness which had so far been undetected. Of the nine children with normal hearing, several had histories of variable conductive losses and needed further follow-up. Other checks have given similar results.

There is no doubt whatsoever that any school health service without a properly organized audiometry programme neglects a very important aspect of school child health. The suggestion that impedance measurements should replace school screen audiometry reveals a lack of understanding of the functions of these tests. Many children are detected by school screen audiometry who would not be detected by the impedance