CORRESPONDENCE

Tranquillizers Causing Aggression

SIR,—Your important leading article (18 January, p. 113) has major implications in the prevention and management of child abuse.

We are engaged in research and service in this field. A high proportion of parents in families referred for actual or threatened child abuse are taking drugs at those times that the crises occur. Benzodiazepines and tricyclic antidepressants have been prescribed in the puerperium on the complaints by the mother of "depression" and anxiety about her capacities as mother and spouse. If she is then isolated with an inconsolably crying infant outbursts of aggression occur.

The interaction between drug effects and social stimulation was, as you rightly note, presciently described by Chance[1] some 29 years ago. The clinical implications have been slow to dawn on us because we have tended to treat diseases rather than persons living in specific circumstances.

Many mothers describe how, while taking these drugs, instead of feeling less anxious and depressed they have become more hostile and openly aggressive towards the child, and often to other members of the family. These disturbed mothers have gone to their doctors with complaints which signalled grossly abnormal interpersonal feelings. Their symptoms are frequently treated as isolated anxiety states or depressions, but the frustrating stimuli continue. Thus the stage is set for the mother to react in a paradoxical way with hostility and aggression.

We advocate extreme caution when prescribing tranquillizers and antidepressants for mothers of young children, especially when the complaints include inability to cope with a baby's demanding and frustrating behaviour. It could well be that such complaints are in fact a warning that a child is at risk of abuse, and well-meant prescribing of tranquillizers might help to precipitate just such an event.—We are, etc.,

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1 Chance, M. R. A., *Journal of Pharmacology*, 1946, 87, 214.

SIR,—Your leading article (18 January, p. 113) is a reminder of the dangers of the widespread unnecessary prescribing of psychotropic drugs.

I suspect that not only is the aggression produced by benzodiazepines directed outwardly to others but the instability that they produce may not infrequently lead to self-poisoning in an impulsive act undertaken with uncertain intent. This is a common finding which was confirmed in an unpublished study undertaken at Walton Hospital, Liverpool, and designed by Dr. Eric Birchall. In approximately 550 consecutive cases of self-poisoning 30% of the individuals had a diagnosis of depression and 5% some other treatable psychiatric condition; 25% had a diagnosis of personality disorder and 40% had no detectable psychiatric abnormality, the act of self-poisoning being a reaction to stress. Quite clearly, had 65% of these people not had access to tablets the workload of the accident and emergency department with its attending psychiatrists would have been considerably reduced. Roughly 63% of the people reviewed had been taking psychotropic drugs, in many instances unwisely prescribed in circumstances where brief psychotherapy was the treatment indicated.—I am, etc.,

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Paraquat Poisoning Treated with Immunosuppressants and Potassium Aminobenzoate

SIR,—I would like to report an interesting clinical result in a case of paraquat poisoning.

The patient, a highly intelligent schizophrenic man aged 32 years, was admitted to this hospital on 21 November 1974 having swallowed seven days previously a quantity of Gramoxone (paraquat) estimated at between one-quarter and one-third of a cupful (>20 ml) diluted with orange juice in a suicide attempt. He did not inform his general practitioner for six days, though during this time he had severe inflammation of the tongue, palate, and pharynx.

On admission he was moderately dyspnoeic at rest with bilateral but minimal left-sided crepitations. Chest *x*-ray showed non-specific consolidation in the left lower lung fields and left basal segment, considered to be in keeping with paraquat poisoning. Renal function tests at this time showed intermittent albuminuria and haematuria. Blood urea was 35·8 mmol/l (216 mg/100 ml) and creatinine 928 mmol/l (10·5 mg/100 ml). A qualitative test for paraquat in the urine with sodium dithionite was negative, but seven days had elapsed since its ingestion. Treatment was initially on standard lines with high dosage of steroids, prednisolone 100 mg daily, and prophylactic antibiotics.

His condition steadily deteriorated and on 26 November left lobe consolidation had greatly increased and the lateral aspect of the right lung was showing involvement, with overlying pleural reaction. The changes were now considered compatible with severe paraquat poisoning. He was now very dyspnoeic at rest and his pulmonary function tests showed progressive deterioration with Po_2 falling to 6·65 kPa (50 mm Hg), Pco_2 remaining at 4·79 kPa (36 mm Hg), and pH 7·48. Liver function tests were unremarkable.

It was decided to try the effects of immunosuppressant therapy in this case since there were certain features similar to those of massive pulmonary fibrosis and a possibility that a delayed immune response might account in part for the lung damage. He was started on azathioprine 50 mg four times daily and in addition was given potassium aminobenzoate as an antifibrotic measure. The effect of the immunosuppressant therapy was monitored by frequent blood counts and immunophoresis of gammaglobulins. The latter showed proportional reductions after therapy started, the IgG level being reduced to 5·83 g/l (583 mg/100 ml).

On 23 December the lung appearances began to improve, starting on the right side and to a lesser extent the left, and on 30 December there was further resolution of the lesions. Concomitantly with this his blood urea level began to fall and reached 10·3 mmol/l (62 mg/100 ml), the creatinine level being 61·9 μmol/l being (0·7 mg/100 ml), and Po_2 steadily rose. These changes were all associated with a progressive clinical improvement which has been maintained, though his lung changes have not yet completely resolved.

I hope to publish further details concerning this patient at a later date but feel that while this isolated case may "prove nothing," in view of the usually grave prognosis associated with severe paraquat poisoning, especially when associated with marked pulmonary radiological signs, this patient's response may be of interest to clinicians who may be dealing with such cases, particularly late cases in which the use of adsorption agents such as Fuller's earth and haemodialysis are not applicable.—I am, etc.,

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Septicaemia on the Increase

SIR,—Your leading article on septicaemia (14 December, p. 615) reminds us that "the intravenous tubes now so commonly used are a convenient portal of entry for these infections." One way in which such infection can enter is by accidental contamination of the piercing needle of the giving set when it is removed from an empty bottle and inserted into a full one. This change over is made some 1300 times each week in the surgical wards in Aberdeen hospitals. Therefore the procedure should be as simple and safe as possible.

Currently we use Avon 11 giving sets, as we have done for 16 years since a Medical Research Council subcommittee[1] devised and recommended them, and they have proved highly satisfactory. They have a combined air-inlet and piercing needle, a good grip can be obtained on the rigid filter chamber, a flange prevents the operator's fingers touching the needle which pierces the bung, and nurses find them easy and quick to manipulate. It is therefore surprising to find that official recognition has been withdrawn from this set. There are now only two giving sets listed in the D.H.S.S. schedule of medical and surgical equipment (13 May 1974, p. 1408)—the Avon A 15 and the Travenot F.K.C. 2005. Both have been designed for use with plastic bags containing stored blood and other derivatives and are excellent for this purpose. Because a plastic bag collapses as it empties there is no need to incorporate an air-inlet into these sets. When, however, these sets are used to give fluids supplied in glass bottles a separate air-inlet assembly must be inserted through the bung in order to admit air while fluid leaves the rigid bottle.

The A 11 set seems to have been withdrawn because blood and its byproducts will soon be generally distributed in plastic bags, and it is not safe to penetrate a plastic bag with the piercing needle of the A 11 set. No one disagrees that the A 15 set is essential here. However, our pharmaceutical colleagues expect intravenous electrolyte and dextrose solutions, dextran, amino-acid solutions, and fat emulsions to be supplied in glass bottles for some years to come. In Aberdeen during 1974 the pharmacy has manufactured infusion fluids in 135 000 500-ml glass bottles and half these bottles have been used on the five general and paediatric surgical wards. The transfusion of blood is largely confined to the day of operation, and many patients do not require any blood. On the other hand, most patients who undergo major alimentary-tract surgery or who come in with peritonitis or intestinal obstruction cannot drink for some days. During this time they receive solutions of sodium, potassium, and dextrose, and in my adult ward it is usual to have about eight patients on this treatment at one time. If we have to use the two official sets for all these infusions then every time a bottle is changed two needles must be changed instead of one on some 1300 occasions each week. Not only is this unnecessary work but there is a serious additional risk of contamination, because the separate air-inlet needles supplied are difficult to hold and to introduce through the thick bungs of the bottles. The likelihood of a nurse's fingers contaminating the lower end of this needle is high.

This risk is unacceptable in any patient but it is especially dangerous practice among patients on long-term intravenous feeding. The great care taken over the insertion of the necessary central venous catheters and over their maintenance over days and weeks can be frustrated by the use of giving sets never designed to be used with glass bottles. It is no argument to claim that these sets are currently used without trouble. Everyone knows that intravenous lines can become infected and how serious this is; can we exclude faulty technique of needle insertion into solution bottles as one source of infection? Happily we can still purchase A 11 sets and avoid this risk, but for how long will manufacturers continue to make a set which is not officially recognized?

The D.H.S.S. appears to have restricted recognition of the A 15 and F.K.C. 2005 sets on the grounds that it is more economical and less confusing to have only one type of giving set available which will safely enter a bag of blood. This is understandable, but in fact we have to keep both the A 11 and A 15 sets at present, because blood sometimes arrives in bags, and no confusion arises. The major fact which has been overlooked is the very large amount of electrolyte and dextrose solutions used on medical wards, all of which are supplied in glass bottles. I and many of my nursing and surgical colleagues believe that there is an overwhelming case for allowing the A 11 to join the A 15 as an officially recognized giving set.—I am, etc.,

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1 Medical Research Council Blood Transfusion Research Committee, *Lancet*, 1957, 1, 595.

Ischaemic Heart Disease in Young Women

SIR,—Though he gives no figures of his own, Dr. I. McD. G. Stewart (14 December, p. 653) doubts the validity of my assessment (2 November, p. 253) of the prevalence (34%) of hypertension in women who developed ischaemic heart disease under the age of 45, and some clarification appears necessary.

(1) All women classified as having hypertension were recorded by me as having a diastolic blood pressure of 100 mm Hg or more and the lowest reading of three taken over a period of 10 minutes was used for this criterion. Of 50 women so classified, 43 (28 out of 32 with myocardial infarction) had a diastolic blood pressure of 100 mm Hg or more recorded on three or more separate clinical examinations. None were regarded as hypertensive solely on the basis of treatment "at the hands of their family doctor."

(2) In 23% the electrocardiogram showed left ventricular hypertrophy. This was defined in the customary way as $R_{aVL}>11$ mm or $R_{aVF}>20$ mm and $S_{V1}+R_{V6} \geqslant 35$ mm: obviously, S-T and T-wave abnormalities would be of little value in the assessment of left ventricular hypertrophy in patients with previous myocardial infarction.

(3) The prevalence of diastolic hypertension (28%) or of left ventricular hypertrophy in those presenting with angina was only slightly, and not significantly, less than that in those with myocardial infarction. This supports the view that hypertension is an important pre-infarction risk factor in young women.

(4) While a prospective study would probably give more reliable data about the prevalence of each risk factor, Dr. Stewart should consider the magnitude of implementing such a study in women of this age group, in which the incidence of myocardial infarction is so small, and ask himself why no comparable data are available even from the Framingham survey or from the Inter-Society Commission for Heart Disease Resources (Pooling Project)—two of the largest prospective surveys of risk factors in relation to ischaemic heart disease.

(5) The majority of women with diastolic hypertension also smoked cigarettes or had hyperlipidaemia. Thus hypertension should not be regarded as the only risk factor contributing to the early development of myocardial infarction.

At present there are no alternative data from which to deduce the prevalence of diastolic hypertension in young women with ischaemic heart disease, and 28-40% (mean 34%) is as near as we can get.

Finally, it was certainly no surprise to me to find that coexisting hypertension worsened the prognosis in each group. This correspondence may help to emphasize the need for assiduous control of diastolic hypertension in young women.—I am, etc.,

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Tuberculosis of the Spine

SIR,—In your leading article under the above heading (14 December, p. 613) you refer to our work in Nigeria and state that we treated our ambulant patients with "chemotherapy alone, using neither rest, nor splintage, nor operations."

In fact our paper[1] reporting the management of patients suffering from spinal tuberculosis certainly mentioned surgery, albeit of a simple nature. Perispinal abscesses were drained when they were associated with paraplegia and the patient was unable to walk (that is, could not yet undergo ambulant treatment). Believing that the paraplegia is primarily caused by the pressure of the abscess on the cord we contented ourselves with the removal of the inner end of a rib or ribs and emptying the abscess under direct vision (costectomy). Twenty-seven out of a total of 207 patients were