old symptoms. For similar reasons, she was reluctant to lose weight. She explained to me: 'I must know where you are. If you disappeared suddenly, I would go into a panic. Coming to see you would mean going back, the dependence on you. I would prefer not to. I have a horrible feeling if I came to see you I would do something silly, go to pieces, return to symptoms, like going back to see old boy friends. I prefer to forget about that.'

Quite clearly, the patient may have made a good symptomatic recovery but basic problems of panic and the need for extra marital relationships had not been resolved. It would indeed have been surprising if this had been achieved by systematic desensitization alone.

2. A 20-year-old single male Drinamyl addict (Kraft, 1968) was equally delighted with his treatment result, and was proud to report that he no longer had any need for addictive drugs, though he did admit to smoking hashish when he was on holiday abroad, which was frequent. Whereas he was quite incapable of doing any sort of work at the time when he began treatment, he had now built up a successful business. He told me that he had put on four stones in weight, which he attributed to excessive drinking when he was working at a brewery, but he had no insight into the mechanisms involved. He found the follow-up interview extremely complicated, and on leaving he said that he hoped that there would never be a need for a further visit.

These are just two examples from my series, but they illustrate the sort of treatment result one may expect from behaviour therapy when given alone. Both patients showed symptom substitution in the form of weight gain, and the transference was not resolved in either case. It is for this reason that I have come to the conclusion that behavioural techniques should be used as part of a much wider psychotherapeutic programme, in which the patient is offered psychoanalytically-oriented psychotherapy as well as behaviour therapy.

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## REFERENCES

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## DEATH DUE TO ISONIAZID (INH) AND PHENYTOIN

DEAR SIR,

The dangerous interactions that can occur between

anticonvulsant and antituberculous drugs, particularly iso-nicotinic acid hydrazide, have become widely recognized during the past ten years. INH interferes with the bio-transformation of hydantoinates in the liver, resulting in a rise in serum levels especially in patients who are slow acetylators of INH. The protean manifestations of 'phenytoin encephalopathy' can be difficult to recognize, particularly in chronic epileptics who may have existing neuro-psychiatric syndromes (Johnson, 1975).

We wish to report briefly on a fatal outcome from such a reaction:

A 47-year-old hospitalized subnormal epileptic had been treated with phenytoin and phenobarbitone for 20 years. At the age of 45 she developed tuberculous adenitis and was treated with I.N.H. and streptomycin. Over the next year, she developed a bizarre neuro-psychiatric state with choreiform movements, and died. At a coroner's court death due to 'phenytoin toxicity' was recorded (serum phenytoin 2 days post mortem 8 mg/100 ml). This was attributed to interaction between the anticonvulsant and antituberculous drugs.

Clinicians treating epileptics who are taking anticonvulsant drugs should be aware of this potentially dangerous interaction which can result if antituberculous drugs have to be simultaneously administered. Determination of the patient's acetylator status and regular monitoring of the serum phenytoin levels are essential if dangerous complications of interaction are to be avoided.

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## REFERENCE

JOHNSON, J. (1975) Epanutin and Isoniazid Interaction, British Medical Journal, 152.

## A COMPILATION OF PAPERS FOR THE USE OF POSTGRADUATE STUDENTS OF PSYCHIATRY

DEAR SIR,

Copies are still available of this compilation, which was prepared by the Clinical Tutors' Sub-Committee and was printed on their behalf by John Wyeth Laboratories; those wanting copies should write to John Wyeth Laboratories, Huntercombe Lane South, Taplow, Maidenhead, Berks.

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