as anxiety state, depressive illness or schizophrenia.

Patients with co-existent organic disorders were excluded. Obsessional ideas and/or ruminations characterized the disorder in four patients, compulsive rituals in two patients, and combination of both in ten patients.

These patients had been referred to the Department after previous treatment had failed to be of benefit, and in some cases for consideration for leucotomy. There were eight male and eight female patients; the mean age at onset was 22·6 years and the mean duration of illness 8·6 years. Previous treatment had consisted of ECT (six cases), phenelzine (one) and chlordiazepoxide (three), and had been without benefit with the exception of one case in which ECT had brought temporary relief.

The tricyclic drugs prescribed were imipramine (eleven), amitriptyline (three), desipramine (one) and dothiepin (one). Eleven patients received 200–300 mg. daily, two of whom also had ECT. Eight of these patients were significantly improved one to three months later, and two unimproved. Five of the patients who improved had previously failed to respond to smaller doses (75–150 mg.). In no case did the compulsive symptoms remit completely. Thus 13 of the 16 patients (81 per cent) obtained significant short-term relief. Moreover, despite the high dosages employed, side effects caused minor inconvenience in only two patients (blurring of vision and constipation), and were absent in the remaining 14.

In order to assess the long-term outcome, a postal follow-up was carried out. The patients were asked to rate themselves on a global scale of improvement, and the results were as follows:

Recovered	3 (22%)
Marked improvement ..	9 (64%)
Unimproved or worse ..	2 (14%)

Two patients failed to reply. Thus, of the patients who responded to the questionnaire, 86 per cent regarded themselves as significantly improved as compared with their state before treatment. Each patient, with one exception, remained on tricyclic antidepressants in high dosage throughout the follow-up period (mean 4·2 years).

Despite the methodological shortcomings of this inquiry, the findings suggest to us that a prospective controlled evaluation of the long-term effects of tricyclic antidepressants in the treatment of obsessional neurosis is indicated. As the disorder is uncommon in hospital practice, such a trial might be most appropriately carried out as a multicentre project. In addition to evaluating the efficacy of tricyclic drugs information might also be obtained regarding the relative value of different tricyclic drugs, different dosages, oral and intravenous methods of administration, and the long-term effects of medication.

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THE MEANING OF THE SYMPTOM IN NEUROSIS

DEAR SIR,

Lazarus (1971) has pointed out that the 'symptom' presented by patients represents *internal* rather than external problems. While this has always been the view of psychoanalysts, it is only very recently that this view has also been expressed by a behaviour therapist. When a patient presents a symptom, what he is in fact saying is that he has become aware of an external situation which has become anxiety-provoking for him, while remaining unaware of the significance of the symptom in terms of psychopathology. This may only become clear to him as the symptom is being treated. Cautela (1965) first pointed out that patients develop insight *after* systematic desensitization, and it may well be that counteracting the anxiety associated with the symptom allows access to the underlying causes which were responsible for its production and mobilizes the original conflicts involved. This is seen particularly well in phobic patients, in whom desensitization may uncover severe sexual problems. This way of thinking is contrary to standard behaviour therapy teaching, which argues that the symptom alone constitutes the neurosis (Eysenck, 1960).

In behaviour therapy literature, it is frequently stated that behaviour therapy leads to emotional recovery in the patient. If this is so, it would seem to be very important to examine all the facets of the patient's recovery *other* than the symptom being treated. After a course of systematic desensitization it can be shown that many changes occur other than

the change in the specific symptom under consideration (Kraft, 1969). There are large personality changes, which can be shown both clinically as well as on psychometric testing (Kraft and Al-Issa, 1967). These personality changes lead to alterations in interpersonal relationships, and in turn may lead to a great deal of hostility being expressed, both towards the patient and to his therapist (Kraft, 1972a, b).

If behaviour therapy leads to basic personality changes, this may be a possible explanation for the patient's recovery during a course of treatment which appears to be symptom-oriented. This would then offer a link between behaviour therapy and psychoanalysis, as the symptom would then be regarded as the externalization of internal problems.

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ORGANIC OR PSYCHOGENIC STUPOR

DEAR SIR,

The importance of not overlooking psychogenic causes when stupor is encountered in the setting of a general medical ward has been illustrated by Saunders (1), akinetic mutism having been the predominant clinical picture in his depressed patient. Conversely, however, I have been asked to see three patients, within a period of eight months typical general psychiatric practice, in whom akinetic mutism eventually proved to be of organic origin. The neuropathology in each case was undertaken by the Institute of Psychiatry.

A married woman of 58, referred by her family doctor as depressed since her senile father's death eight months before (after caring for him over many years), was admitted to psychiatric hospital. She presented in a state of akinetic mutism, her eyes following people and there being some facial but no limb movement, possibly accompanied by a degree of parkinsonian rigidity. A week later, early papilloedema and developing drowsiness led to her transfer to a neurological centre where she slowly deteriorated and died after 9½ weeks. Autopsy revealed a carcinoma of the lung with secondaries occupying much of the upper halves of the posterior frontal regions of the brain bilaterally (left more than right) disrupting the corpus callosum anteriorly.

A married woman of 64 was first investigated in a general medical ward for a year's history of dementia. A diagnosis of cerebral arteriosclerosis was made after a reasonable number of otherwise negative findings had been established. Reinvestigated in another general medical ward two months later, the patient having moved to a new locality, evidence of pyramidal and extrapyramidal involvement had developed; the diagnosis remained unchanged. I saw the patient in a third general hospital three months later still, by which time akinetic mutism had been present for a month, and a diagnosis of catatonic schizophrenia was being considered. She died of bronchopneumonia a week later in the psychiatric hospital to which she had been transferred. At necropsy, an ependymoma of the pineal region about 2·5 cm. in diameter was found lying mainly in the third ventricle, slightly distorting the thalami and displacing the superior colliculi laterally and downwards, depressing 0·5 cm. of the aqueduct. There was bilateral uncinate grooving, and slight right hippocampal herniation from obstructive hydrocephalus.

A widow of 62 was initially referred by her family doctor to the geriatric service with a tentative diagnosis of presenile dementia of two to three years duration. By the time her relatives eventually accepted psychiatric admission for observation and investigation eight months later, an initially restless, wandering, confused, forgetful, clinical picture without other central nervous signs had gradually over the last six months given way to akinetic mutism and the development of long tract pyramidal and extrapyramidal involvement. She died of pulmonary embolus two weeks after coming under psychiatric care. The only really significant finding on examination of the brain was bilateral degeneration of the thalamus (with medial and postero-medial fibrillary gliosis thereof) and dilatation of the aqueduct.

The relevance to psycho-physiology of both organic and psychogenic factors producing otherwise indistinguishable clinical phenomena should not need emphasizing further; for example, the possible relationship between akinetic mutism and the brain-stem reticular formation or activating system has already been discussed (2).

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