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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