

This difference was again significant ($t = 1.8712$; 17 d.f., $P < 0.05$).

These results lend further support to an association between cognitive decline in elderly Down's syndrome subjects and macrocytosis. The explanation for this remains unclear. Increased cell volume is associated with many conditions including Down's syndrome (Eastham & Jancar, 1983). However, it appears that in the deteriorated Down's syndrome subjects this increase is over and above the usual moderate increase in MCV found in Down's syndrome. There was no evidence of Vitamin B₁₂ or folate deficiency in our subjects to account for this further increase. Kedziora (1981) showed that the red cell membrane in Down's syndrome was subject to accelerated ageing and that ultrastructural defects were present. This may go part of the way in explaining macrocytosis in Down's syndrome and it may be that the deteriorated subjects are physiologically more aged than their non-deteriorated peers. These results seem to indicate that further work is needed in determining the exact nature of this relationship.

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References

- HEWITT, K. E., CARTER, G. & JANCAR, J. (1985) Ageing in Down's syndrome. *British Journal of Psychiatry*, **147**, 58–62.
EASTHAM, R. D. & JANCAR, J. (1983) Macrocytosis and Down's syndrome. *British Journal of Psychiatry*, **143**, 203–304.
KEDZIORA, J., KOTER, M., BARTEL, H., *et al* (1981) Ultrastructural modification of the erythrocyte membrane in Down's syndrome. *Acta Biologica et Medica Germanica*, **40**, 423–428.

Chronic Pain Syndrome

DEAR SIR,

Pilowsky & Bassett (1982) have highlighted differences that exist between a 'depressive' group and a group with 'chronic pain syndrome' (where the pain is without adequate somatic explanation). The idea that 'chronic pain syndrome' is a form of masked depression is likely to represent an oversimplification of the issue. However, therapy aimed at relief of depression is frequently helpful in this patient group. In the absence of depressive symptomatology the only symptom change we can measure is pain. The McGill Pain Questionnaire (MPQ) (1975) has both quantitative and qualitative dimensions. We report here on a case of 'chronic pain syndrome' using the MPQ as a measure of change.

The patient, a 41-year-old married man, complained of abdominal pain with a 20-year history. At the start this had occurred in discrete episodes but had been continuous during the past 8 years. Approximately every month pain became 'unbearable' and necessitated some days absence from work. He had been investigated on four separate occasions just stopping short of laparotomy each time. No somatic pathology was ever discovered. He had been referred to various psychiatric services and treated with antidepressants, benzodiazepines, either alone or in combination. He presented here firmly believing he had a physical ailment, taking tricyclics, benzodiazepines and analgesics regularly with no improvement. Therapy included rationalisation of pharmacotherapy (i.e., reduction of benzodiazepines over three weeks from chlordiazepoxide 50 mg daily to chlordiazepoxide 20 mg daily, and replacement of imiprimine, 50 mg daily with amitryptiline to a dose of 150 mg daily), education regarding psychological phenomena in somatic complaints, relaxation training and supportive psychotherapy.

The MPQ was administered prior to treatment and again eight weeks into therapy. Prior to treatment his visual representation of "where pain is felt" included the entire abdominal and thoracic region, the lumbar and sacral area and the vertex of the skull. Pain was felt both internally and externally. After eight weeks the area of pain was confined to the periumbilical region and was felt only internally. Scores on the descriptive aspects of the pain were as follows (the first figure was the score prior to treatment, the second was the score at eight weeks, and the third was the highest possible score).

Total pain rating index (PRI)	36	(16)	(79)
Sensory PRI	19	(8)	(41)
Affective PRI	9	(2)	(14)
Miscellaneous PRI	6	(3)	(17)
Present pain intensity	4	(2)	(5)

These changes on the MPQ reflect a change both in the quality and quantity of pain and were associated with increased well-being, improved sleep pattern and an increase in physical activity.

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References

- MELZACK, R. (1975a) The McGill Pain Questionnaire: major properties and scoring methods. *Pain*, 1, 277–299.
- PILOWSKY, I. & BASSETT, D. L. (1982) Pain and Depression. *British Journal of Psychiatry*, 141, 30–36.

Psychiatric Disturbances in the Shy-Drager Syndrome

DEAR SIR,

The neurological multiple system atrophy (MSA) described in 1960 as Shy-Drager syndrome is caused by a progressive loss of catecholaminergic cells of the intermedio-lateral column of the spinal cord and, to a lesser extent, by a loss of cells of substantia nigra, nucleus coeruleus, nucleus tractus solitarius and preganglionic vagal neurons. A loss of norepinephrine and dopamine within the hypothalamus and limbic system is also observed (Bannister, 1979). This fact might account for the emotional disturbances observed during the course of the illness. I have recently examined a patient whose psychological disturbance preceded the appearance of an uncommon symptom—laryngeal paralysis—and, later, that of the most typical sign, postural hypotension.

The patient was a 59 year old man who eight years previously suffered from dysthymic disorder for about two months. Three years prior to presentation a relapse took place and, in April of the same year, he underwent an operation of arytenopexis because of a unilateral syndrome of Gerhardt (paralysis of the laryngeal abductors).

Two years ago, while still under tricyclic antidepressant therapy, the patient began to show prominent signs of postural hypotension. The tricyclic therapy was discontinued and, after the diagnosis of Shy-Drager syndrome was made therapy with fludrocortisone and hypernatremic diet was begun. Until a couple of months ago the course of the illness proved satisfactory, but in October it was necessary to resume tricyclic antidepressants.

This case is interesting because it is associated with symptoms which may not be recognised as signs of a Shy-Drager syndrome: disturbances of mood and laryngeal paralysis. While the latter is recognised as an early symptom of MSA (Bassich *et al*, 1984), it is

not yet clear to what extent psychological symptoms may accompany and even forerun the neurological signs.

A systematic investigation of the psychological and psychiatric aspects of this syndrome would be of interest.

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References

- BANNISTER R. (1979) Chronic autonomic failure with postural hypotension. *Lancet*, ii, 405–406.
- BASSICH, C., LUDLOW, C. L., POLINSKY, R. J. (1984) Speech symptoms associated with early signs of Shy-Drager syndrome. *Journal of Neurology, Neurosurgery and Psychiatry*, 47, 995–1001.

Schizophrenia and Ethnicity

DEAR SIR,

It is disturbing to find the introduction of apartheid terminology into what may be considered a scientific paper (*Journal*, December 1985, 147, 683–687). The South African authors' aim was a comparison of PSE diagnosed Catego class 'S' schizophrenia in three 'ethnic' groups. Their subjects' ethnicity was described as 'white', 'coloured' and 'blacks'. This unfortunately is an arbitrary racial categorisation perpetuated for political reasons and to facilitate racial segregation by the minority government in South Africa. Does this have any place in scientific literature? What was the basis on which 'coloured' people were so categorised? It may be understandable why researchers working in a particular political climate are influenced by the prevailing dominant ideologies but it must be a matter of concern that the *Journal* saw fit to print an article with politically loaded definitions of little scientific import.

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A HUNDRED YEARS AGO

Part III—Psychological Retrospect

1. English Retrospect. Asylum Reports

Lancashire. Rainhill. A murderous assault was made by a male patient on the head attendant, who fortunately escaped with a wound of the wrist. The patient had secreted a knife from the bakehouse, and intended to kill one of the assistant medical officers. He was committed for trial, and subsequently transferred to Broadmoor. The Commissioners

recommended his being put on trial, "as a warning to other lunatics, many of whom think that they can commit crimes with absolute impunity."

Reference

Journal of Mental Science (1886) October. Vol. XXXII. Pp 424.

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