Letters

Factors affecting language recovery in aphasic stroke patients receiving speech therapy

Sir: The Nottingham Aphasia Study[1] describes a large scale investigation of the effectiveness of speech therapy for aphasic stroke patients. Subjects were randomly allocated to either treatment or no treatment groups and monitored for a 34 week period. In addition, there was an opportunity to examine factors affecting response to the treatment offered.

There is extensive literature examining the relationship between age and response to treatment[2–4] with the majority finding that age has no significant effect on recovery. However, Sands, *et al*[5] found that age was the most potent variable influencing recovery in a small sample of relatively young, treated patients. Other studies have indicated that age may be related to the type of aphasia experienced by the patient[6 7] and this may relate to recovery.

The type of aphasia has been found to be important. Basso *et al*[8] found that global and Wernicke's aphasics responded to treatment of 6 months or more duration. Kertesz and McCabe[3] found that the type of aphasia was associated with the amount of improvement and the final language level reached. Sarno and Levita[9] stated that the effect of aphasia type differed at various stages of recovery. In the first 6 months after stroke, fluent patients improved more than non-fluent and in the second 6 months, global aphasics improved most and fluent aphasics least. The severity of aphasia, which is closely related to aphasia type, has also been found to be an important predictor of final language level.[5 10] It is generally agreed that sex of the patient has no relationship to language recovery.[4 11–13] The Nottingham study offered an opportunity to examine the factors affecting recovery.

The assessment procedures and criteria for exclusion from this study are described by Lincoln *et al*.[1] All patients described in this paper were in the treatment group of that study.

There were 104 aphasic stroke patients, aged 38–92 years (mean, SEM = 67·3, 1·07), of whom 59 were men. Severity of aphasia ranged from an overall score of 13·2 to 2·8 on the Porch Index of Communicative Ability[14] (PICA) at 10 weeks after stroke (possible range 0–15·0) and Functional Communication Profile (FCP)[15] from 7·7 to 94·9 (possible range 0–100). On the basis of the Boston Diagnostic Aphasia Examination[16] (BDAE) administered 16 weeks after stroke there were 22 Broca's, 19 Wernicke's, 17 Conduction and 18 Anomic Aphasics. There were 15 patients with severe expressive and comprehension disorders who were classified as Global Aphasics. There were 13 patients who were not classified; of these six did not attend for the 16 week assessment, four did not fit into any recognisable group and three died before classification.

Of the 104 patients available at 10 weeks after stroke, 86 were assessed at 22 weeks and 87 at 34 weeks after stroke. The reduction of numbers was because seven patients died between 10 and 22 weeks and seven died between 22 and 34 weeks of assessment. In addition, eight patients refused to attend the 22 weeks assessment, two were too ill and one had moved away, but seven patients not assessed at 22 week returned for the final assessment at 34 weeks. The Porch Index of Communicative Ability[14] (PICA) and the Functional Communication Profile[15] (FCP) were administered at 10, 22 and 34 weeks after stroke.

In order to examine the factors affecting recovery, analyses of variance were performed on the final Overall PICA and FCP scores at 34 weeks after stroke with initial scores at 10 weeks as covariates.

The effect of two variables was investigated in relation to recovery on the language assessments. These were age (broken down into five age decades) and type of aphasia (Broca, Wernicke, Conduction, Anomic, Global). There were no significant differences between the aphasia types in language scores (PICA $F4,68 = 1·74$ $p = 0·5$, FCP $F4,68 = 1·49$ $p = 0·21$). There were significant differences between the age decades for final scores on the PICA ($F4,68 = 5·12$ $p = 0·001$) with older patients achieving lower levels of overall ability. The mean overall PICA scores at 10 weeks in the five age decades were: 40–49 years 9·89, 50–59 years 9·78, 60–69 years 9·37, 70–79 years 8·35, 80–89 years 7·88. At 34 weeks mean overall PICA scores in the five age decades considered were 40–49 years 11·49, 50–59 years 10·69, 60–69 years 10·90, 70–79 years 8·86 and 80–89 years 8·38. Scores on the FCP at 34 weeks did not differ significantly between the age decades ($F4,68 = 1·05$ $p = 0·39$), though there was a trend for results to follow the same pattern as the PICA. The mean FCP at 10 weeks was: age 40–49 years 52·1, 50–59 years 50·6, 60–69 years 50·3, 70–79 years 42·8, 80–89 years 42·3. The mean FCP at 34 weeks was: age 40–49 years 66·2, 50–59 years 60·4, 60–69 years 59·6, 70–79 years 47·8 and 80–89 years 47·7.

Aphasia type was not related to recovery in this study even though clinical subjective impressions are that the more severely language impaired patients such as global aphasics do less well. It is possible that the clinician is aware of spontaneous language recovery occurring in all patients, but because only the less severely impaired attain functional language recovery, the therapist may consider only the latter to be a successful outcome of therapy. This is consistent with a study by Lendrem and Lincoln[17] which showed that all aphasia types show similar amounts of spontaneous recovery but that more able patients such as Broca's, Conduction and Anomic aphasics achieve higher language levels at 34 weeks than Global and Wernickes aphasics. Results on age are consistent with clinical impressions, indicating that older patients improve less. This is contrary to the findings of Lendrem and Lincoln who found that all untreated patients in the study improved by a similar amount regardless of age.

We thank Dr G Mulley, Professor J R A Mitchell and Miss M Edwards for their assistance with the study, Dr D W Lendrem for statistical advice, Mrs A Dodson and Mrs A Dudley for typing the manuscript and the Research Committee of Trent Regional Health Authority for financial support.

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Accepted 2 April 1988

Noradrenaline, adrenaline and tyrosine hydroxylase in adrenal medulla from Parkinsonian patients

Sir: Recent experimental[1 2] and clinical[3 4] studies have suggested that autografting tissue from adrenal medulla into the striatum may improve symptoms resulting from nigro-striatal dopamine degeneration. Parkinson's disease is associated with a severe dopaminergic and noradrenergic deficiency in the brain.[5] Whether the disease affects central dopamine or noradrenaline levels, or peripheral levels as well, in particular those of the adrenal medulla, has not been reported and is of interest with regard to the usefulness of autografts in patients.[3 4]

Nine adrenal glands from subjects with no evidence of endocrinological, psychiatric or neurological disease (mean age: 75·3, SEM 1·9 years; *post-mortem* delay: 18·2, SEM 2·9 hours (range: 6·5–30)) and 12 adrenal glands from patients with Parkinson's disease (mean age: 73·9, SEM 3 years; *post-mortem* delay: 20·6, SEM 1·7 hours (range 10–29)) were examined. The adrenal glands were stored at −70°C, until adrenal medulla was dissected free from the cortex at −15°C, under a dissecting microscope. The whole tissue from each adrenal medulla was crushed into powder on dry ice, and biochemical assays were performed on an aliquot of the structure. Noradrenaline, adrenaline and dopamine were assayed by high performance liquid chromatography with electrochemical detection.[6] The dopamine values are not mentioned as they were too low (in the order of 1 ng per mg tissue) and not reproducible. Tyrosine hydroxylase activity was assayed according to Puymirat *et al.*[7]

The catecholamine content in adrenals from control subjects was in good agreement with studies in monkey[8] (table). Adrenaline concentrations were four times higher than those of noradrenaline. In adrenal glands from patients with Parkinson's disease, the levels of noradrenaline, adrenaline and tyrosine hydroxylase activity were slightly but not significantly decreased compared with control values (table). These observations contrast with the previously reported deficiency in tyrosine-hydroxylase,[9] and suggest that the catecholaminergic systems in the adrenal medulla (unlike those in the brain)[5] are not markedly affected in the disease. Thus the histopathological changes observed in adrenals in cases of Parkinson's disease[10] may not be associated with a massive catecholaminergic degeneration. In monkey, the Parkinsonian syndrome induced by the administration of MPTP (1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine) is not associated with a deficiency in adrenal gland catecholamine content[1 8] a difference from the central effects of the neurotoxin. Therefore, brain catecholaminergic neurons seem to have a specific vulnerability. The present data: (1) emphasise that adrenal autografts in the striatum of patients have the biochemical capacity to substitute a catecholaminergic activity; (2) suggest that the targets of the pathogenic process in Parkinson's disease are mostly restricted to catecholaminergic neurons in the central nervous system; (3) are compatible with an efficiency of adrenal medulla autografts in patients with Parkinson's disease. The mechanism by which these autografts provide a clinical improvement remains unknown: tyrosine hydroxylase might restore dopamine neurotransmission; implanted cells may reinnervate the host striatum; grafted cells might induce some recovery of dopamine neurons.[1 11]

Table *Noradrenaline, adrenaline and tyrosine hydroxylase levels in adrenal medullas from Parkinsonian patients*

	Control (n = 9)	*Parkinson (n = 12)*
Noradrenaline	194, 43	167, 45
Adrenaline	866, 144	644, 168
Tyrosine hydroxylase	71·3, 16·3	48·2, 14·2

Values are expressed in ng/mg tissue for catecholamines and pg/h/mg tissue for tyrosine hydroxylase activity.
n = number of adrenal glands.
Data are the mean, SEM.

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