

BRAIN DAMAGE AND ECT

DEAR SIR,

Some critics of ECT have stated that it produces brain damage (Alpers and Hughes, 1942; Friedberg, 1977). Most reports of brain damage, however, date from before 1956 and are from patients treated without general anaesthesia and muscle relaxation so that the cerebral lesions could well have been a consequence of the seizure and are probably irrelevant to modern practice (Clare, 1979). Experimentally in primates only prolonged or rapidly repetitive induced seizures produced any brain damage; no lesions could be detected with seizures of the type and frequency used in ECT (Meldrum *et al*, 1974).

Unilateral but non-dominant hemisphere ECT is probably as effective in relieving depression as bilateral ECT, while producing less memory disturbance (McKenna and Pratt, 1983). The availability of computed tomography gives the opportunity of comparing the hemispheres after prolonged administration of unilateral ECT, with the possibility of revealing any macroscopic structural change induced by the therapy.

We present 12 patients who received a large number of ECT delivered mainly to one hemisphere. In two patients (cases 4, 5) CT scans were available before ECT had been given; in five others (cases 1, 6, 7, 10, 11) scans had been made very early in the course of ECT after only two to six treatments; in all 12 an

examination was made at the end of the therapy which had lasted over one to forty years. Details of patients and the number, side and type of ECT are given in the Table.

Each scan was assessed independently, without knowledge of its timing relative to the ECT, for any abnormality of the cerebral substance and for the presence and degree of atrophy. Comparable measurements of the maximum transverse diameters of the frontal horn, body and trigone of each lateral ventricle were expressed as a percentage of the maximum transverse diameter of the vault between the inner tables in order to eliminate variations due to magnification as far as possible. The difference in the average of the measurements before and after ECT was expressed as a percentage increase or decrease. (See Table).

Mild atrophy was present in four elderly patients (cases 2, 6, 7, 9) and minimal atrophy in one middle-aged patient (case 10). Minor asymmetries in ventricular size are frequent in normal individuals and occurred in our patients; the larger ventricle was equally often ipsilateral (4 cases) and contralateral (4 cases) to the side on which ECT was mainly administered. Any increase in atrophy over the years was minimal and either bilateral or equally ipsi or contralateral to the treated hemisphere. Bilateral ill defined low density present in the cerebral white matter of one 79-year-old

TABLE

Case/ Initials	Current Age/Sex	Number of ECT, type of machine* and (electrode placement)	Total ECT		Initial or early CT	Final Ct & Intervening Period	Change in Ventricular Size		
			R	L			side of ECT	Opposite side	
1/AB	63/F	33E (B) 17P (B) 106P (R) 169C (R)	325	50	Normal	Normal	4 years	+0.3%	+0.3%
2/PF	76/F	20E (B) 9P (B) 49P (R) 8C (R)	86	29	—	Slight cerebral & cerebellar atrophy	—	—	—
3/VG	73/F	6E (B) 59C (R)	65	6	—	Normal	—	—	—
4/AG	53/M	80C (R)	80	0	Normal	Unchanged	4 years	-0.4%	-0.4%
5/MG	66/F	74C (R)	74	0	Normal	Normal	4 years	+0.4%	+0.5%
6/ZH	73/F	52P (R) 27C (R)	79	0	Mild cerebral atrophy	Unchanged	4 years	+0.7%	+0.7%
7/EH	69/F	18E (B) 69C (R) 10C (B)	97	28	Mild cerebral & cerebellar atrophy	Unchanged	1 year	0.0%	0.0%
8/JH	44/M	29C (R)	29	0	—	Normal	—	—	—
9/KO's	79/F	4E (B) 56C (R) 27C (B)	87	31	—	Cerebral atrophy white matter low density	—	—	—
10/HT	54/F	90P (B) 40P (L) 178C (L)	90	308	Minimal cerebral atrophy	Unchanged	2 years	-0.6%	0.0%
11/HT	51/F	28C (R)	28	0	Normal	Unchanged	1 year	+1.1%	-0.3%
12/CW	38/M	8P (R) 6C (R)	14	0	—	Normal	—	—	—

B = bifrontal:

R L = right or left, mastoid-frontal (Halliday *et al*, 1968)

Each patient had one or two non-dominant ECT in the procedure of establishing language laterality (Pratt and Warrington, 1972)

C = progressive modifications of the City instrument (Gordon, 1981) delivering 1 millisecond pulses ranging from 100–2,000 milliamper the voltage being adjusted to the patient's resistance which varied from 200 ohms to 1,000 ohms.

E = Electron or similar machine (mains transformer with secondary 130 V winding: duration controlled at will by operator probably 1–2 seconds).

P = Plexacon Mark II (Theratronics) capacitor discharge 15–50 joules, diphasic or monophasic: 5 milliseconds square wave with 5 millisecond interval.

patient was entirely consistent with ischaemic change, due to vascular disease affecting the cortical perforating arteries which is commonly present in the elderly.

The absence of CT changes cannot exclude damage due to the therapy but it is encouraging that CT showed no evidence of this occurring with prolonged courses of ECT taking place over widely varying periods of time.

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Book Reviews

Psychotherapy, A Personal Approach. By D. J. SMAIL. London: J. M. Dent. 1978. Pp 150. £3.50.

Focus on Group Psychotherapy: Clinical Essays. By SAUL SCHEIDLINGER. New York: International Universities Press. 1982. Pp 264. No price stated.

These books represent personal views of their authors. In both the final result is an overview of a wide field.

Scheidlinger's collection of essays put together from various American journals and chapters from previously published books, gives a quite wide-ranging survey of group psychotherapy, with an analytic emphasis, as practised in the United States. This is not an integrated book but does bring a number of well thought out essays together. I found those on empathy in groups, the mother group, leadership, scapegoating and combined group and individual therapy to be interesting and provocative discussions of selected literature. The chapters on groups for children and women with severe character disorders are based largely on clinical experience and do not, to my mind, sit well in a book which otherwise raises theoretical issues. Nonetheless, the author's special pleading on behalf of groups for children and adolescents and for the effectiveness of groups in the treatment of very damaged people should be noted.

It is useful to get such a clear presentation of views from across the Atlantic. There are views, however, which do not fully take into account the development of group psychotherapy in the United Kingdom. Scheidlinger tends to refer to the work of Bion and Ezriel as representing the British approach, rather than acknowledging the more recent growth of group-analytic psychotherapy which is becoming the mainstream of group psychotherapy theory and practice in this country.

Whilst Scheidlinger tends to look at specific phenomena such as scapegoating or leadership, developing 'small domain' theories about them, Smail takes time to point out that the non-specific components of the psychotherapeutic relationship may be far more important than the specific theories of any particular school of theory.

In chapters on research in psychotherapy and the scientific philosophy of psychotherapy, Smail reviews the scientific basis of our discipline. In doing this he pursues a well trodden path and does not, to my mind, add substantially to previous discussion. Nonetheless, his language is simple and clear, and he presents issues which should be thought about by all who are involved in the study and treatment of psychological disorder.

These are interesting books to stimulate and provoke: they are not main courses, however, and should