Conclusion

In summary, the chapter on personality development in the textbook under review provides a comprehensive and critical overview of the field. It is well-written, thought-provoking, and offers a wealth of information for both students and practicing clinicians.

A. J. Franks


This large textbook, now in its third edition, is the work of several authors, most of whom have present or past links with the University of Cincinnati. The senior author says in the preface that the book is meant for non-psychiatric physicians. To produce such a work is a laudable aim, but the book has serious shortcomings.

In general it is too long, it has an outdated air, and there is too much preoccupation with psychoanalytic concepts. This last fault is particularly evident in the long, theoretical account of personality development as well as in the chapter on psychosomatic disorders, where the old notion of specific unconscious conflicts underlying specific physical disorders is brought out of the historical museum. There is no mention of epidemiological studies in this field, no mention of the importance of life events in illness in general. Recent references are few.

The same criticism, of lack of reference to important recent work, applies to much of the book. For example, there are several pages on the family dynamics of schizophrenics, but nothing at all on the Danish-American work on the genetics of schizophrenia. There are two chapters on treatment, sensibly divided into one on measures suitable for non-psychiatrists, one on measures suitable only for the psychiatrist. Rather less sensibly, the second chapter is the longer: it contains about 20 pages on psychotherapy, less than two on behaviour therapy. Incidentally, the non-psychiatrist, after being given a very sketchy account of tricyclic antidepressants, is told that he should 'almost certainly' arrange for psychiatric consultation before prescribing them.

J. L. Gibbons

Notices

International Conference on Neurological Epidemiology 16–17 May 1977 (Conference course: 15 May 1977), Gorman Auditorium, Gorman Building, George-town University Hospital, 3800 Reservoir Road, N.W., Washington, D.C. 20007, USA. No registration fees. Details from Dr B. S. Schoenberg, Head, Section on Epidemiology, NINCDS, NIH, Room 7C10A, 7550 Wisconsin Avenue, Bethesda, Maryland 20014, USA. (Phone: 301-496-1714.)

The two-day conference will consider current knowledge in neuroepidemiology and stress the applicability of this information to the practice of neurology and neurosurgery.

Sixth International Congress on Neurological Surgery 19–25 June 1977, São Paulo, Brazil. Details from Congress Office, Caixa Postal 20389, 01000 São Paulo, SP, Brazil.

Letters

Mechanism of the inverted supinatus reflex

Sir,—Estanol and Marin (1976) infiltrated finger flexor muscles with procaine in two patients with an inverted supinatus reflex. They then found that no finger jerk could be obtained by tapping the fingers but that finger flexors still contracted in response to percussion of the styloid process of the radius. This was interpreted as evidence against the latter response being mediated by muscle spindles in the finger flexors and in favour of intraspinal spread of afferent impulses, presumably thought to originate in biceps and brachioradialis. Estanol and Marin quoted earlier work of mine (Lance, 1965; Lance and de Gail, 1965) which demonstrated that the irradiation of reflexes depends on the propagation of a vibration wave from the point percussed to the bellies of muscles participating in the reflex contraction. Gamma efferent block by procaine infiltration renders the muscle spindles less sensitive but still susceptible to excitation by an effective stimulus. The latency for EMG of flexor muscles in Estanol and Marin’s experiments was 33.2 ms when the fingers were tapped and 10.8 ms when the radius was percussed, suggesting that the latter is a more direct and possibly more effective stimulus. In this case the afferent volley from the finger flexors induced by vibration would elicit contraction of those muscles through the monosynaptic pathway, the more readily if alpha motor neurone excitability is enhanced by rostral cord compression. The absence of contraction of biceps and brachioradialis would be explained by compression of the posterior roots at the fifth and sixth cervical segment.

If, as Estanol and Marin propose, the afferent pathway from the C5-6 segments is intact, the absence of the biceps and brachioradialis reflexes must be caused by damage to the anterior roots (unless the lesion interrupts the reflex arc within the spinal cord). Anterior root lesions would be expected to reduce reflex contraction in proportion to reduction in muscle power whereas power is often preserved in biceps and brachioradialis when their reflexes are abolished.

To prove Estanol and Marin’s hypothesis it would be necessary to infiltrate the biceps and brachioradialis with procaine and show that the reflex contraction in finger flexors then disappeared. On both clinical and neurophysiological grounds, the balance of evidence presently available would still appear to favour the ‘peripheral’ rather than ‘central’ explanation of the inverted supinatus jerk.

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References

