- Nicol, D. S. H. W., and Smith, L. F. (1960). *Nature (Lond.)*, **187**, 483.
- Poulsen, J. E. (1953). *Diabetes*, **2**, 7.
- Randle, P. J. (1954a). *Brit. med. J.*, **1**, 1237.
- (1954b). *Lancet*, **1**, 441.
- (1954c). *Ibid.*, **1**, 809.
- (1956). *J. Endocr.*, **14**, 82.
- and Young, F. G. (1956). *Ibid.*, **13**, 335.
- Read, C. H., and Bryan, G. T. (1960). *Recent Progr. Hormone Res.*, **16**, 187.
- Richardson, K. C. (1940). *Proc. roy. Soc.*, **128B**, 153.
- and Young, F. G. (1938). *Lancet*, **1**, 1098.
- Rodriguez-Candela, J. L. (1945). *An. Acad. méd.-quir. esp.*, **29**, 283.
- (1952). *J. clin. Endocr.*, **12**, 245.
- Gofii, P. M., Caldeiro, M. R., and Gonzalez Carreras, P. (1947). *Trab. Inst. nac. Cienc. méd. (Madr.)*, **9**, 89.
- Sanger, F. (1960). *Brit. med. Bull.*, **16**, 183.
- and Thompson, E. O. P. (1953). *Biochem. J.*, **53**, 366.
- and Tuppy, H. (1951). *Ibid.*, **49**, 481.
- Stavitsky, A. B., and Arquilla, E. R. (1953). *Fed. Proc.*, **12**, 461.
- Sutherland, E. W., and de Duve, C. (1948). *J. biol. Chem.*, **175**, 663.
- Thorogood, E., and Zimmermann, B. (1945). *Endocrinology*, **37**, 191.
- Vallance-Owen, J., and Lilley, M. D. (1961). *Lancet*, **1**, 804.
- Yalow, R. S., and Berson, S. A. (1957). *J. clin. Invest.*, **36**, 648.
- Young, F. G. (1937). *Lancet*, **2**, 372.
- (1938). *Proc. roy. Soc. Med.*, **31**, 1305.
- (1939a). *Brit. med. J.*, **2**, 393.
- (1939b). *New Engl. J. Med.*, **221**, 635.
- (1945). *Biochem. J.*, **39**, 515.
- (1953). *Recent Progr. Hormone Res.*, **8**, 471.
- (1959). In *Ciba Foundation Tenth Anniversary Symposium on Significant Trends in Medical Research*, pp. 135–157. Churchill, London.
- (1961). *Mem. Soc. Endocr.*, **11**, 101.

PSYCHIATRIC ASPECTS OF EPILEPTIC AND BRAIN-DAMAGED CHILDREN*

BY

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Brain Damage

The concept of the brain-damaged child that has been steadily gaining ground in the last few years merits careful and critical examination. Contributions to it have been made by a number of authors—for example, Bradley (1957), Strauss and his co-workers (Strauss and Lehtinen, 1947; Strauss and Kephart, 1955), Bender (1956), and, most recently, Eisenberg (1957), whose review of the symptoms may be taken as one of the clearest expositions of the clinical concept. The symptoms said to be most commonly associated with brain damage in children are hyperkinesis, distractibility and short attention-span, lability of mood, antisocial behaviour, low intelligence, and anxiety. These are diffuse symptoms in the sense that they affect large areas of mental functioning, though it is uncertain whether the responsible lesions are diffuse or focal—for example, in the temporal-lobe or mid-brain regions.

The criteria for brain damage appear to be as follows:

1. *History*; which may be of a head injury, encephalitis, anoxia at birth, meningitis, or many another trauma or inflammation of the brain and its coverings. This history may be good evidence of brain damage in the past, but there is the obvious danger of *post hoc, ergo propter hoc*, when it is claimed as the cause of subsequent symptoms.

2. *Neurological Signs and Symptoms such as Hemiplegia or Hemianopia*. These symptoms are signs of damage of a particular part of the brain. Their absence does not

signify the absence of brain damage elsewhere, nor does their presence necessarily imply that the damaged area producing these symptoms may also be responsible for the other psychiatric symptoms of which the patient complains.

3. (a) *Special Investigations: Physical (A.E.G. and E.E.G.)*.—The air-encephalogram is a coarse test of brain damage and sometimes shows normality even when surgery or necropsy has subsequently shown clear evidence of brain damage. The interpretation of the E.E.G. contains even more pitfalls, because there is certainly no E.E.G. change pathognomonic of diffuse brain damage. The findings of an excess of slow activity in an E.E.G. of a patient with a past history of brain damage cannot be regarded as unequivocal evidence of a continued physiological disturbance from that brain damage (see Cobb, 1950). Furthermore, in children there is a very wide variation in standards of normality at any one age, making interpretation still more difficult.

3. (b) *Special Investigations: Psychological*.—An adequate survey of the psychological test investigations in brain damage would occupy a lecture in itself (see reviews of Meyer, 1960; and O'Connor, 1958). There would seem to be three main groups of tests. The first refer to the use of special indices in the standard intelligence tests of Wechsler or Binet. Measures of deterioration in adults by comparing the verbal and performance scores are well recognized and standardized. Their application in children is more difficult, since on the one hand wide discrepancies may be found between verbal and performance scores in patients in whom brain damage is not suspected, and on the other no such discrepancies may be found in children with undoubted brain damage. Educated parents can produce a spuriously high verbal score in their backward children.

The second group of tests are those concerned with special psychological functions, of which those related to perceptual anomalies seem to be the most important—for example, the Bender-Gestalt, various form-board tests, etc. Unfortunately, most of these tests have been poorly standardized with inadequate controls. Moreover, often there are no clear correlations between the test results and, on the one hand, the neurological status, and, on the other, personality difficulties and maladjustment (Cruickshank and Bice, 1955).

The third group of psychological tests are the so-called projection tests, of which the Rorschach is the one most frequently used. Many authors have described specific organic signs in the Rorschach test, but these have been equally often denied, and the whole status of this test, and therefore, by implication, of other projective tests, is at the moment under a cloud (Tizard, 1961).

These strictures on the dubious value of most psychological tests should not be regarded as decrying the function of the psychologist himself. In fact, it somewhat increases it in that he or she is essential for the empirical study of any particular case, especially as regards the practical problems of the treatment of a damaged child. By patient investigation of what a child can or cannot do—in, for example, the learning situation—the psychologist makes an essential contribution which cannot be carried out in any other way, but which has a much more dubious connexion with the theoretical problems. For example, the works of Strauss and Lehtinen (1947) and Strauss and Kephart (1955) are deplorably diffuse, irrelevant, and inaccurate, but their insistence on the study of the individual case and their conclusions about teaching methods of handicapped children are important.

There are thus no absolutely unequivocal clinical signs, physiological tests, or psychological tests that can

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prove a relationship between brain damage and any particular aspect of disturbed behaviour. The position is much less certain than in the case of epileptic phenomena, since there are numerous opportunities, clinically and experimentally, of recording the abnormal electrical activity of the nervous system in a fit, at the same time as observing the abnormal behaviour of subjects in the throes of an epileptic discharge.

Clinical Survey of Brain-damaged Children

I have recently begun a survey of the children seen at the Maudsley in whose clinical state organic factors were considered to be important. It at once became apparent that the majority of such children were of below average intelligence, often at the lowest grades. Of such cases I chose only those in which the aetiology was clear-cut, and most of them were fully investigated as in-patients. All those who had epileptic attacks of any sort at any time were excluded.

The adoption of these strict criteria produced relatively few cases, scattered over a number of years. The analysis given in Table I is based on the first 58 obtained from the records.

TABLE I.—*Brain-damaged Children Without Epilepsy (Males, 40; Females, 18)*

Evidence for brain damage:		Age when brain damage suffered:	
History only	30	0-1 year	35
Physical signs	21	2-4 years	8
A.E.G.	4	5-9	7
E.E.G.	3	10+	2
		Not known	6
Intelligence level:		Age when first seen at Clinic:	
Less than 50	8	No.	Expectation from Total Clinic Cases
50-79	21	2-4 years	3 (5%)
80+	29	5-9	21 (36%)
Significant discrepancies	11	10-14	28 (49%)
		15+	6 (10%)

In order to assess the significance of the clinical data I have compared some of the facts with an unpublished dissertation of Dr. Q. Grant. He investigated a consecutive sample of non-organic children referred to the Maudsley Clinic with special reference to the age-and-sex distribution of their symptoms. He based his work largely on the method of McFarlane *et al.* in their excellent longitudinal survey of normal children in California.

In my series males outnumbered females more than 2 to 1, which is about the same as in the clinic generally. The intelligence range showed half the patients to have an I.Q. of less than 80, the usually accepted lower limit of the "normal" range. Only 11 of the children showed anomalous test results, either on the routine psychological tests or on the special tests such as the Strauss marble board, the Benton, etc.

The onset of the brain damage was in more than half the cases under 2 years of age, birth events being by far the largest single cause. Of the rest, meningitis, encephalitis, and head injury were the main causes. In contrast to the age of onset, the ages at which the patients were first seen at the clinic were some years later, mostly between 5 and 14 years, the peak periods for the ordinary clinic population (Table II).

As regards the background of the cases, the social-class distribution shows an excess of social class I, which may be due to the greater concern of educated parents for their children, or—less charitably but more plausibly

TABLE II.—*Brain-damaged Children Without Epilepsy (58 Cases)*

Social class	Total	Expected	Environment			
			Normal home	Broken	Institution	Other relatives mentally ill
I	8 (14%)	4.7%	13	13	4	16
II	9 (16%)	16.2%	13	13	4	16
III	18 (31%)	55.1%	13	13	4	16
IV	13 (22%)	10.7%	13	13	4	16
V	8 (14%)	13.3%	13	13	4	16
Not known	2	—	13	13	4	16

	Age of Brain Damage					Age when Seen at Clinic			
	0-1	2-4	5-9	10+	N.K.	2-4	5-9	10-14	15+
Hyperkinesis	8	2	1	0	2	2	8	3	0
Aggression	11	4	2	0	2	2	7	10	0
Neurotic symptoms	15	4	5	0	5	0	10	16	3

—because such parents cannot reach down to the level of these children and so often handle them badly. Only 13 children came from normal homes—that is, with both natural parents present and neither suffering any overt psychological illness or abnormal attitudes to the child. In half of the cases the parents, especially the mother, grossly overprotected or rejected the child, or showed some other abnormal attitude. The interpretation of parents' attitudes is based in most cases on the notes of many interviews between parent and doctor or psychiatric social worker. The bias is therefore that of the whole clinic, and it is open to the objection that psychiatrists think few people are normal. However, in addition, a third of the children were or had been for years in institutions or hospitals, or had come from broken homes with one or both parents missing, step-relations, etc. About one-quarter had close relatives who were psychiatrically ill.

As in the case of epilepsy, the symptoms are extraordinarily varied, and it is clear at once that the classical syndrome of the brain-damaged child does not cover more than a few of the children seen. Of the numerous symptoms present, only three groups are common enough to be analysed. They are: neurotic symptoms, abnormal aggressiveness (temper tantrums, cruelty, etc.), and hyperkinesia or restlessness.

Table III shows that the age of sustaining brain damage in these symptom-groups does not differ from the average of the whole group. However, the ages at which the patients were seen at the clinic show that hyperkinesia is commoner at the younger ages, aggression right through the middle ages, and neurotic symptoms in adolescence. We may compare this distribution with that found for similar symptoms by Grant in the non-organic population of the Maudsley Child Guidance Clinic. He found a peak for over-activity at ages 5-7 years in boys, for aggression at a similar age, and again in adolescence, and for neurotic symptoms from 11 to 13 years. Neurotic symptoms are

TABLE III.—*Brain-damaged Children Without Epilepsy*

	Male	Female	I.Q.		Abnormal Parental Attitude	
			<80	80+	Present	Absent
Neurotic symptoms:						
Present	21	8	11	18	15	14
Absent	19	8	18	9	15	12
Abnormal aggressiveness:						
Present	17	4	8	11	12	7*
Absent	23	14	20	18	17	20
Hyperkinesia:						
Present	12	1	10	3	6	7*
Absent	28	15	19	24	21	22

* All with abnormal homes.

seen in both sexes, but are more common in those with higher (that is, normal) I.Q.s than lower. Abnormal aggressiveness is unrelated to intelligence, but is more common in boys than in girls. Not one of the children with those symptoms came from a normal home. Thirdly, hyperkinesia is most common in low intelligence, in boys much more often than girls, and again none of these children came from normal homes.

Thus the findings of Grunberg in epileptic-behaviour problem-children are closely paralleled by those with brain-damaged children; that is, as regard the type of symptoms, their age distribution, and the prevalence of disturbed environments, the brain-damaged child differs little from the ordinary child psychiatric population, *except* for the factor of low intelligence.

It would appear that the symptoms that bring a brain-damaged child to a psychiatric clinic are very similar to those for which non-brain-damaged children are brought. This type of survey cannot, of course, disclose how common these symptoms (and others) are in brain-damaged children generally, nor whether the brain-damaged child has other symptoms or defects not detected by our child psychiatric methods.

As regards the first point, only surveys of unselected samples or follow-up studies are appropriate. Several studies have shown that head injuries in children produce few long-term ill-effects even when they have been very severe and the patient has been very disturbed immediately afterwards (see Harrington and Letemendia, 1958). On the other hand, children with the permanent neurological defects that have led to their being classified as cerebral-palsied children are often emotionally disturbed. Dunsdon (1952) thought more than one-third were unstable, especially those with an I.Q. below 70; and Floyer (1955) thought nearly half showed some of the psychiatric symptoms said to be typical of the brain-damaged child.

Another study bearing on the prevalence of psychiatric symptoms in brain damage is Tizard's (unpublished) survey of mental defectives at home and in institutions, which covered children and adults; and, of course, some of the patients may not have had an acquired brain damage in the sense used here. He found that the only common, important, and easily assessable trait was underactivity or overactivity. 25% of the children were regarded as overactive, 9% as underactive. Only 41% were regarded as normal in temperament. It is also interesting to note that in adult defectives the problem of activity is reversed: 28% are underactive, only 12% overactive—a change similar to that which I have already mentioned in epileptic children.

These surveys can be regarded as no more than preliminary and merely showing how large a problem there is. There are so many variables that must be allowed for. For example, the factors of age, intelligence level, and presence of other physical disabilities in the child and in its environment, and the quality of parent or institutional care, are just a few that spring to mind at once.

The second point I mentioned concerns the possibility of there being psychological symptoms not shown by our psychiatric methods—especially, of course, the possibility of focal disorders of speech, motility, perception, and so on. In the case of children, many people consider that there is no such thing as a focal disturbance of function with a focal lesion of the cerebral hemispheres. This is manifestly untrue in the case of

the simple uncomplicated cerebral palsy which may affect only one limb. Even at higher levels the problem may not seem so simple; for example, with acute lesions such as abscesses or open head injuries aphasia undoubtedly occurs in children, but it never results in a logorrhoea, as can sometimes occur in adults (Guttmann, 1942). Rather the child is more or less speechless, even completely mute. It is obvious that in general an early lesion in childhood may lead to failure to acquire patterns of behaviour, and this may have repercussions in many other ways. (A simple and obvious example is the effect of a peripheral lesion causing deafness that will lead to a failure to acquire speech if it is not tackled early and energetically.)

In the Maudsley material suspected aphasias are very rare—no more than three or four in more than ten years—and psychogenic mutism on the top of brain damage seems commoner. The impression that focal disorders of all sorts are rarely the sequel of brain injury is confirmed by the literature on such disturbances as reading disabilities or so-called congenital auditory imperception, etc. Few, if any, of such cases seem to be related to brain damage unless the disorder is part and parcel of a general mental deficiency. Many of them are inextricably mixed up with a general emotional disturbance, so it is hard to sort out which came first.

It seems that in our present state of knowledge we can say that in a general way the brain-damaged child is handicapped—in the first place, and without any doubt, as regards intelligence. Secondly, such children may suffer from defects of personality that are much less clear-cut, but probably concern all aspects, such as tolerance of anxiety, controllability of primitive impulses, and so on. As our methods of localizing brain damage at the cortical level are so poor, no useful analysis of the effects of different types and sites of brain damage can be made as Nuffield could with epilepsy. In any case, I very much doubt if there are any specific psychiatric entities such as the post-encephalitic, the post-traumatic, and so on, except in so far as there is also focal damage producing focal symptoms at a neurological level, such as a paralysis or a special sense loss, thus adding a particular contribution to the general handicap.

Influence of Environmental Factors Before the Brain Damage

The environmental factor enters into the problem of the effects of brain damage in an even more fundamental way than the handling subsequent to the injury. In the last few years evidence has accumulated showing that the prevalence of premature births and other medical conditions of childhood possibly related to brain damage are not distributed randomly in the population but are concentrated in the lower social classes. For example, Douglas (1960), following up the premature babies in his very big prospective survey, found that they were inferior in their school and social progress but that this inferiority was clearly correlated with the social status of the parents and the general quality of maternal care rather than with the brain damage that may have been presumed to result from prematurity. Fairweather and Illsley (1960) found similarly that among the 66 children in Aberdeen's school for the educationally subnormal there was a high incidence of obstetric abnormalities, but in all these cases the families were from the lower social classes

with poor standards and often low intelligence, so that the authors felt there was no need to postulate birth trauma as a cause of the mental handicap.

In the last few years Pasamanick, Lilienfeld, and colleagues have published several papers on what they call prenatal and paranatal factors in the production of cerebral palsy, epilepsy, and behaviour disorders. They postulate a "continuum of reproductive casualty." On the face of it their work is impressive, with a careful control series for comparison with the cases taken from the same schools. However, the social factors are not adequately controlled. For example, the monograph of Rogers, Lilienfeld, and Pasamanick (1955) on behaviour-problem children shows a table of the number of families known to the Baltimore social service agencies. A highly significant larger number of the white cases, as opposed to the white controls, are known to the agencies. (Of the smaller number of non-whites, three-quarters of both cases and controls are known.) Similar differences appear in the problem children which they call misleadingly "hyperactive, confused-disorganized." Until the social status and quality of parental care of these children is properly controlled, no firm conclusions can be drawn about the significance of the organic factors.

(The contrast between these surveys and my hospital figure of excess of class I is due to the clinic being a selected sample and illustrates the fallacies of hospital data.)

The sorting out of constitutional *versus* environmental factors is a routine problem in psychiatry, and ordinary family history studies are of dubious value. Twin studies are the only reliable method. Thanks to the extremely complete statistics on the comparatively static population of Sweden, Dencker (1958) was able to collect a large series of twins, one of whom had sustained head injury severe enough to warrant admission to hospital. These twins were, of course, of all ages at the time of the injury and at the time of the examination. The follow-up in most cases was many years after the injury. Dencker interviewed both the proband and the control twin, and in a smaller number of pairs psychological tests were administered to both twins by Löfving (Dencker and Löfving, 1958).

Dencker was particularly interested in the so-called post-traumatic syndrome, and his work suggests strongly that persistent psychiatric symptoms which occur after injury have little relationship to the severity of the injury but a much closer relationship to the previous personality. He showed, for instance, that in most cases the uninjured identical twin tended to have similar symptoms, though usually in a milder degree. Löfving's tests showed up very small differences that were apparent only when the uninjured twin was used as a control. The test results obtained were in most cases within the normal range, but it did appear in general that the injured twin was slightly slower in many forms of responsiveness. In addition to these changes in psychological tests being very slight, there was also no obvious correlation between the severity of the changes and the severity of the general psychiatric picture. Dencker's paper refers to closed injuries only; hardly any of the patients developed epilepsy, and there were very few signs of focal brain damage.

Dencker's work does not adequately analyse the possible effects of environmental pressures in these similarities and differences. Identical twins have

notoriously their own problems and tend to be treated somewhat alike. Interestingly enough, the investigation of Shere, quoted by Cruickshank and Bice (1955) on a small series of identical twins, one of whom had brain injury from birth or other early cause, would suggest that in some cases the uninjured twin showed more evidence of psychological disturbance. The reason for this, she plausibly argues, is the differential handling of the children by their parents, who would tend to favour the handicapped child, so that the non-handicapped one may well feel rejected and react accordingly. These observations are, of course, strongly in favour of the predominant importance of environmental factors.

The assessment of the long-term psychological effects of brain damage is therefore more complicated than it appears at first. The age and previous character of the person suffering the brain injury, and the ways in which he is subsequently handled, are the main factors determining the nature, severity, and duration of symptoms. Only in the case of intelligence, and possibly related traits such as viscosity or slowness, does there seem to be an important direct effect of the brain damage, and then the effects appear to be quantitative rather than qualitative, and proportional to the amount of brain damaged rather than its site and nature.

What we do not know are, firstly, the right personality traits to investigate in any constitutional twin studies designed to throw light on how people react to brain damage; secondly, we do not know the correct formulation of the effects of brain damage even as regards intelligence, since our present psychological tests often give equivocal or negative results. As regards the third factor—namely, the analysis of the effects of environmental handling—we are a bit further on, since the generally accepted concepts of child-parent relationships used in everyday child-guidance work help us not only to understand but also to treat brain-damaged children.

Animal Experiments

Before attempting any further formulation of the effects of brain damage, brief consideration should be given to the results of certain animal experiments, especially those involving the deep temporal-lobe structures and the mid-brain reticular regions, the two areas with which we are particularly interested as regards behaviour and epilepsy.

The idea that the deep temporal-lobe regions may be connected with the expression of the emotions was put forward before the war by Papez (1937), but only in the last few years has the clinical and experimental evidence of this been clearly forthcoming. As in the case of the human observations, the behaviour disturbances in animals seem, in fact, to be related to three fields: firstly, the physiological expression of the emotions; secondly, the recent memory and learning of the new material; and thirdly, consciousness.

The work on the somatic expression of the emotions has recently been reviewed by Ursin (1960). Of the three common patterns of emotional behaviour seen in mammals below man—namely, rage, fear, and pleasure—only the first two are seen as the result of stimulation of the amygdaloid and probably the hippocampal regions. On the other hand, stimulation of comparable areas in the human at operation occasionally may produce the reaction of fear but rarely that of rage, and the responses seen are usually more complex and involve disturbances of consciousness. Williams (1956) found

that the content of epileptic auras is nearly always that of fear or depression and rarely rage. (The rage reactions found in chronic epileptics, as a form of interictal behaviour rather than ictal or pre-ictal behaviour, are different, as already mentioned.)

The effects of ablation of the deep temporal regions in animals are actually very varied. Green *et al.* (1957) mentioned 29 often contradictory varieties of behaviour that have been reported. Sexual activity is in the majority of cases increased as the result of these lesions, often with perverse tendencies as well. Green and Kaada both think there is some evidence of localization of function within the amygdaloid region, but there is as yet no convincing human clinical evidence on this point. Several authors (for example, Fuller *et al.*, 1957) have stressed that the circumstances under which the behaviour of the animals with brain lesions is studied is also of importance—a fact which, of course, agrees closely with clinical observation of human cases. In addition to the changes in respect of emotion, Green *et al.* (1957), for example, think that many of the changes in the behaviour seen with amygdala lesions in cats are due to disturbances of consciousness, and he compares their activity with disturbances of behaviour seen in psychomotor epileptics.

Experimenters with a different theoretical bias have been interested in the effects of lesions on the acquisition of new behaviour patterns—that is, learning and conditioning—rather than on the emotional reactions seen with stimulation or frustration. In the past no convincing evidence of any localization of such functions at the cortical level had been obtained, but, following on the observations of Penfield and Milner (1958) and Meyer (1959) on temporal lobectomy patients, Stepien *et al.* (1960) have recently obtained evidence that deep temporal lesions do impair particular types of new learning.

The work on the mid-brain reticular region centres around two closely interrelated problems. Firstly, there are those of consciousness, or level of activity, or awareness of the whole organism; and secondly, the problem of the regulatory mechanisms, particularly of metabolic activity; that is, there are both upward and downward influences from the reticular regions. Furthermore, the level of attention or activation or vigilance of the brain is maintained partly by the inflow of sensory information, but also by cortico-fugal impulses. Thus, the brain exerts some selectivity not only on the extent to which it will respond to incoming signals but also on the strength of the incoming signals themselves, though it is as yet uncertain how far down the sensory pathways central control can be exerted.

Most of the animal experimenters seem as far off a general theory of explanation of the effects of brain damage and epilepsy as are the workers on human material. I personally find very satisfying and stimulating the concepts of central-nervous-system function that derive from so-called "information theory." Pribram (1960) recently reviewed some of the applications of this type of theory. Hyperkinesis, for example, may be related to a failure of the homeostatic mechanisms that are designed to keep the sensory information within manageable limits. The simplest analogy of the homeostat is the thermostat, which keeps the temperature of a room constant. If the thermostat is very sensitive then the heat-source has to be turned on and off every few seconds. When we speak of a

hyperkinetic child as being stimulus-bound we refer to the same sort of close tie between stimuli and the responses thereto. But a thermostat is also adjustable in the sense that the temperature at which the room is maintained can be altered. In the same way, we see that in patterns of sleep, wakefulness, underactivity, and overactivity the whole brain may react more or less to incoming stimuli. Such an idea may perhaps be related to the disturbances of sleep that these children show, since they are very often slow to go off to sleep and then go very profoundly and deeply asleep. This rhythm of sleep and wakefulness is less related to light and darkness and the other rhythms of daily life to which normal children get adjusted.

So far as the clinical effects of epilepsy in these regions are concerned it was, of course, the relationship of bilaterally synchronous and symmetrical spike-and-wave to loss of consciousness without other clinical accompaniments that first led the Montreal school to the concept of the centrencephalic system. The possible connexion of generalized epileptic activity to homeostasis was actually put forward independently by Denis Hill (1952) some years ago in an article comparing and contrasting maturation disturbances in the E.E.G. with what he called homeostatic phenomena. In support may be cited the therapeutic effects of electric shock treatment, provided the patient has a proper grand-mal attack. Possibly also the self-stimulating photosensitive patients have discovered a similar regulating mechanism. (On the other hand, the famous Maudsley patient who deliberately obtained sexual satisfaction culminating in an epileptic attack by the contemplation of open safety-pins had undoubted temporal-lobe epilepsy, so one cannot impute regulatory functions only to centrencephalic epilepsy (Mitchell *et al.*, 1954).)

I do not think that the animal experiments so far provide a clear answer to the question about any extra part that might be played by the lesions that are epileptogenic rather than non-epileptogenic. Green *et al.* found in their extensive study of temporal-lobe lesions in cats that the minority that showed aggressive behaviour had all had epileptic attacks, but also all of them had lesions that were more widespread into the hippocampal regions than with the non-aggressive animals, so that the different size and location of the lesions may account for the differences. I do not know of any convincing experiments showing positive behaviour disturbances related to on-going epileptic discharges in contrast to the negative effects of such discharges, with resultant disorganizations of behaviour and the emergence of simpler patterns, that are the result of the activity of those parts of the brain unaffected by the discharging lesion.

The human clinical material stresses three points that are little discussed by animal workers and may be very important. The first is the age of the subject affected by a lesion; the second is other individual differences; and the third point is the importance of the environment in which the subject is tested for the behaviour under question.

Summary

I should like to try to summarize my general argument. In the first place, the brain-damaged child is handicapped as regards those functions, included under the general concept of intelligence, with which are bound up memory and learning. The term dementia

is usually applied to older subjects, and implies most typically a loss of recent memory—that is, the capacity to learn new material. Making allowances for the differences produced by age, the difficulty of the brain-damaged child in learning may not be fundamentally different. The poverty of our methods of localizing brain damage makes it impossible to implicate any particular area of the brain, and, in general, mental defect has been associated quantitatively with the amount of cortex damaged. However, the animal experiments make it possible that the deep temporal and mid-brain structures may be more important than others, but the powers of recuperation of the brain are very great.

Secondly, the psychological reaction of the child to being handicapped manifests itself in symptoms that are related to age, and understandable only when the emotional environment of the child is also taken into account. The brain-damaged-child syndrome does not exist in its own right, like an extensor plantar response; but only when the whole situation is considered can the contribution of the intrinsic handicap be assessed. The management of the brain-damaged child requires the fullest and deepest psychiatric approach.

Thirdly, the child who has epilepsy as well as brain damage has an additional handicap in two ways. Firstly, the epileptic activity may intensify the extent to which the brain damage interferes with cerebral activity, particularly when frequent attacks disrupt the activity of the normal parts of the brain. Secondly, the fits are a considerable social and emotional handicap, to which adjustment is difficult.

Epileptic children will long continue to be usefully studied psychiatrically for many reasons. For example, the interference with physiological functions during attacks can be much more carefully studied than can the non-epileptic effects of brain damage. On the other hand, the long-continued handicap of epilepsy imposes a strain on the family situation, the reactions to which illuminate many problems of parent-child relationship found in everyday child psychiatry.

REFERENCES

- Alström, C. H. (1950). *Acta psychiat. scand.*, Suppl. 63.
 Bender, L. (1956). *Psychopathology of Children with Organic Brain Disorders*. Thomas, Springfield, Ill.
 Bingley, T. (1958). *Acta psychiat. scand.*, Suppl. 120.
 Bradley, C. (1957). *Pediat. Clin. N. Amer.*, 4, 1049.
 Bridge, E. M. (1949). *Epilepsy and Convulsive Disorders in Children*. McGraw Hill, New York.
 Chaudhry, M. R., and Pond, D. A. (1961). *J. Neurol. Neurosurg. Psychiat.*, 24, 213.
 Cobb, W. A. (1950). In *Electroencephalography*. Edited by D. Hill and G. Parr. McDonald, London.
 Cohen Committee Report (1956). Cohen Committee Report on the Medical Care of Epileptics. H.M.S.O., London.
 Collins, A. L. (1951). *J. consult. Psychol.*, 15, 392.
 Cruickshank, W. M., and Bice, H. V. (1955). In *Cerebral Palsy*, edited by W. M. Cruickshank and G. M. Raus. Chap. IV. Syracuse Univ. Press.
 Dencker, S. J. (1958). *Acta psychiat. scand.*, Suppl. 123.
 — and Löfving, B. (1958). *Ibid.*, Suppl. 122.
 Douglas, J. W. B. (1960). *Brit. med. J.*, 1, 1008.
 Dunsdon, M. I. (1952). *The Educability of Cerebral Palsied Children*. Newnes, London.
 Eisenberg, L. (1957). *Psychiat. Quart.*, 31, 72.
 Fairweather, D. V. I., and Illsley, R. (1960). *Brit. J. prev. soc. Med.*, 14, 149.
 Floyer, E. B. (1955). *A Psychological Study of a City's Cerebral Palsied Children*. Brit. Council for Welfare of Spastics, London.
 Fuller, J. L., Rosvold, H. E., and Pribram, K. H. (1957). *J. comp. physiol. Psychol.*, 50, 89.
 Gastaut, H., and Fischer-Williams, M. (1959). *Handbook of Physiology*, edited by J. Field. Vol. I, Section I, "Neurophysiology," p. 329. American Physiological Society.

- Glaser, G. H., and Dixon, M. S. (1956). *Neurology (Minneapolis)*, 6, 646.
 Green, J. D., Clemente, C. D., and Groot, J. de (1957). *J. comp. Neurol.*, 108, 505.
 Grunberg, F., and Pond, D. A. (1957). *J. Neurol. Neurosurg. Psychiat.*, 20, 65.
 Guttmann, E. (1942). *Brain*, 65, 205.
 Harrington, J. A., and Letemendia, F. J. J. (1958). *J. ment. Sci.*, 104, 1205.
 Hill, D. (1952). *Electroenceph. clin. Neurophysiol.*, 4, 419.
 James, I. P. (1960). *J. ment. Sci.*, 106, 543.
 Jeavons, P. M., and Bower, B. D. (1961). *Arch. Dis. Childh.*, 36, 17.
 Johnson, L. C., Ulett, G. A., Sines, J. O., and Stern, J. A. (1960). *Electroenceph. clin. Neurophysiol.*, 12, 861.
 Lennox, W. G. (1960). *Epilepsy and Related Disorders* (2 vols.). Loveland, N., Smith, B., and Forster, F. M. (1957). *Neurology (Minneapolis)*, 7, 856.
 Masland, R. L. (1960). *Epilepsia (Boston)*, 1, 512.
 Metrakos, J. D., and Metrakos, K. (1960). *Neurology (Minneapolis)*, 10, 228.
 Meyer, V. (1959). *Arch. Neurol. Psychiat. (Chicago)*, 81, 299.
 — (1960). In *Handbook of Abnormal Psychology*, edited by H. J. Eysenck, p. 529. Pitman, London.
 Mitchell, W., Falconer, M. A., and Hill, D. (1954). *Lancet*, 2, 626.
 Morrell, F., Roberts, L., and Jasper, H. H. (1956). *Electroenceph. clin. Neurophysiol.*, 8, 217.
 Nuffield, E. J. A. (1961). *J. ment. Sci.*, 107, 438.
 O'Connor, N. (1951). *J. Neurol. Neurosurg. Psychiat.*, 14, 226.
 — (1958). Chapter in *Mental Deficiency: The Changing Outlook*, edited by A. M. and A. D. B. Clarke. Methuen, London.
 Oumsted, C. (1955). *Lancet*, 2, 303.
 Pampiglione, G., and Falconer, M. A. (1960). *Handbook of Physiology*, edited by J. Field. Section I, "Neurophysiology," 2, 1391. American Physiological Society.
 Papez, J. W. (1937). *Arch. Neurol. Psychiat. (Chicago)*, 38, 725.
 Penfield, W., and Milner, B. (1958). *Ibid.*, 79, 495.
 Pond, D. A. (1952). *J. ment. Sci.*, 98, 404.
 — (1957). *J. Indian med. Prof.*, 3, 1441.
 — (1958). *Modern Trends in Paediatrics*, edited by A. Holzel and J. P. M. Tizard. Chapter 12. Butterworth, London.
 — and Bidwell, B. H. (1954). *Brit. med. J.*, 2, 1520.
 — (1960). *Epilepsia, Boston*, 1, 285.
 — and Stein, L. (1960). *Psychiat. Neurol. Neurochir. (Amst.)*, 63, 217.
 Pribram, K. M. (1960). *Ann. Rev. Psychology*, 11, 1.
 Roger, J. (1953). *Rev. Neuropsychiat. infant.*, 1, 1.
 Rogers, M. E., Lilienfeld, A. M., and Pasamanick, B. (1955). *Acta psychiat. scand.*, Suppl. 102.
 Sherwood, D. L., et al. (1961). In press.
 Siegmund, W., and Pache, H. D. (1953). *Mtschr. Kinderheilk.*, 101, 191.
 Stauder, K. H. (1938). *Konstitution u. Wesens-veränderung der Epileptiker*. Thieme, Leipzig.
 Stepien, L. S., Cordeau, J. P., and Rasmussen, T. (1960). *Brain*, 83, 470.
 Strauss, A. A., and Kephart, N. C. (1955). *Psychopathology and Education of the Brain-injured Child*.
 — and Lehtinen, L. E. (1947). *Psychopathology of the Brain-injured Child*. New York.
 Symonds, C. (1955). *Brit. med. J.*, 1, 1235.
 Tizard, B. (1961). *Psychol. Rev.* In press.
 Tizard, J. (1960). *Brit. med. J.*, 1, 1041.
 Ursin, H. (1960). *Acta psychiat. scand.*, 35, 378.
 Williams, D. (1956). *Brain*, 79, 29.
 W.H.O. Technical Report Series, 130. *Juvenile Epilepsy*. Geneva, 1957.

At a meeting of the court of the University of Leeds the Vice-Chancellor spoke about the practice, gaining ground in the new universities, of providing more broadly based courses each covering two of the traditional subjects of study. "It is thought possible," he said, "that courses of this kind, leading to 'honours' degrees, may well prove able in the coming years to hold their own in the matter of prestige, or even more than hold their own, with the older 'special honours' courses. It is perhaps too soon to tell. But there is increasingly wide recognition to-day both that knowledge does not entirely divide itself up without remainder into the traditional academic fractions, and also that many students will find themselves better educated for their future life and work if they do not confine their studies too rigidly to one specialist line of study. Emphasis is still placed upon study in depth; but the new courses will allow two subjects, each studied in the 'honours' manner, to be pursued to a high level of attainment."