Behaviour after Cerebral Lesions in Children and Adults

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It is generally believed that brain injuries vary in their effects according to the age at which they are sustained, but the nature and extent of these differences remain elusive. A full enquiry requires, ideally, the study of strictly comparable lesions in the young and the old, the opportunity for follow-up examinations extending over decades in both groups and, lastly, the availability of behavioural tasks for which normal attainment is known, and which are equally applicable to the brain-injured child and the brain-injured adult.

The investigations of behavioural changes after brain injury in children and adults, reported here, are far from fulfilling all these requirements. In particular, these studies are obviously deficient with regard to our first postulate, that of comparable lesions. Identical lesions in children and adults are so rare that one is often forced to turn to animal experiments as a supplementary source of information. Admittedly, animal experiments introduce phylogenetic differences in addition to the ontogenetic ones; yet available animal studies can help to indicate some of the directions in which the answers to the ontogenetic question may be sought. Accordingly, we begin this account with a brief consideration of animal experiments, before presenting some of the results obtained in our laboratory in studies of brain-injured adults and children.

Animal Experiments

The principal outcome of earlier experimental studies of animals has been deceptively simple: comparison between the effects of early and late ablations has suggested that the earlier the lesion is made, the less the disability that remains. Tsang (1937), one of Lashley’s students, working with rodents, found that cortical ablations from the brains of infant rats had to be proportionately larger, in order to produce maze-running deficits (three months later) equivalent in severity to those produced by cortical ablations in adult animals. In these studies on rats, the smaller effect of lesions on the infant brain was a matter of degree.

By contrast, Kennard’s comparison (1938, 1942) of motor cortex removals in infant and adult monkeys suggested that there was almost a difference in kind: bilateral removals that rendered the adult
macaque incapable of righting itself were followed in the infant macaque by swift reappearance of postural and locomotor functions, albeit with deficit (Kennard 1942). Even more extensive removals (involving the somato-sensory and motor cortex bilaterally) were found to be compatible with postural control. However, there were some delayed effects (Kennard 1939): during the second and third postoperative months contractures and exaggerated tendon responses developed, and by the end of the first year, spasticity had become marked.

Kennard’s work therefore seems to reveal two possible consequences of early ablation: (1) some effects may be minimal and transient and thus much less disabling than corresponding lesions sustained by an adult; (2) certain other effects may not appear until later in life. In her interpretations, Kennard emphasised the evidence for resiliency and paid less attention to the delayed appearance of deficits. Her general conclusion was that there may be considerable plasticity of the infant brain, and that recovery of motor function, in her own animals, might have involved some reorganisation of dendritic connections. Probably because of these anatomic conjectures, Kennard’s reports have been called into question.

However, closely corresponding results have recently been obtained by Benjamin and Thompson (1959) in a study of sensory function in cats and kittens, following bilateral removal of sensory cortex. After bilateral resection of somatosensory cortex I and II in newborn kittens, no deficit could be detected (six months postoperatively) on a graded series of roughness discriminations, except on the most difficult of the tests. When the same regions were removed from mature cats, there was complete and apparently permanent disability (six months after operation) on all sensory tests employed. These results are thus analogous to those of Kennard, with the exception of the delayed effects of early lesions, which were not found by Benjamin and Thompson. The new results extend the first consequence found by Kennard from motor to sensory functions and from primates to carnivores.

Even more far-reaching are the observations of Doty (unpublished) on removals of visual cortex in kittens and cats. In his adult animals he obtained the usual loss of pattern vision when their striate cortex was destroyed. In newborn kittens, however, no change in pattern vision was found, unless the lesions were extended beyond the primary visual cortex and included some extra-striate foci on the lateral aspects of both hemispheres. Such unusual results suggest, as one possibility, a somewhat different mode of representation in the neonate brain. The relatively greater resiliency of the young would then not reflect a capacity for active reorganisation of function (as Kennard suggested) but rather a less discrete representation for certain functions, or an allotment of comparatively larger cerebral regions for the mediation of certain functions which in the adult occupy a smaller critical area.

There is surprisingly little direct evidence from animal studies for the supposed existence of less restricted localisation in the infant brain. A more recent study by Massopust and Daigle (1960), employing evoked potential methods, suggests, however, that certain sensory regions, defined electrophysiologically, may be proportionately larger in the infant than in the adult. In this study the cortical areas involved in response to stimulation of the medial and spinal vestibular nuclei were found to be about 25 per cent larger (in both contralateral and ipsilateral hemispheres) in the kitten than in adult cats. If differential results of anaesthesia and other complicating factors can be ruled out, the results are intriguing. This 25 per cent difference
in area allotted might mean that there is a more widespread representation of certain functions in the young brain, and that this representation becomes restricted as more and more functions are crowded into the maturing hemispheres. However, ablation studies are equally compatible with the view that functional localisation in the infant brain is less specific than in the adult. Either view would make it unnecessary to assume an active process of reorganisation following trauma to the infant brain.

**Limitations of Available Evidence**

Work on subhuman forms has not so far indicated how one should choose between different interpretations, nor have the experiments suggested whether, for some aspects of behaviour, early lesions might be just as disabling as later ones, or even more disabling than lesions sustained at maturity. Such alternative outcomes of early injury are strongly suggested by clinical observations.

In fact, not everyone believes that functions of the young brain are always more diffusely organised and hence more resilient than those of the mature organism. Ritchie Russell (1959), for instance, on the basis of clinical evidence, has urged us to consider the possibility that frontal-lobe lesions in children might be much more devastating to their subsequent development than equivalent lesions of the frontal lobes in the adult. He proposes that the frontal lobes might carry on some essential functions in the child which are no longer crucial at later ages or which can then be mediated almost equally well by other regions.

More recently, infant macaques with bifrontal lesions have been studied in Harlow’s laboratory (Akert et al. 1960). In the adult monkey, lesions of this type produce lasting loss of the capacity for delayed response (Jacobsen 1936), but removals in early infancy leave this response intact. The results may not be as contradictory to Ritchie Russell’s prediction as might appear—there is as yet no unanimity regarding the basis of success or failure on delayed-response tasks—but developmental studies in normal macaques (Harlow 1959) suggest that this response taps some rather primitive capacities, rather than ‘complex functions’. It is therefore possible that the impression of smaller effects of brain lesions in the young might be more valid for certain aspects of sensorimotor function but much less valid for complex intellectual performance. Even in the normally developing child without any brain damage, precocity of sensory and motor development cannot be used to predict future intellectual achievement. In fact, these two aspects of behaviour appear to be essentially uncorrelated, except in the presence of massive brain damage or malformation. Thus, it is conceivable that early injuries might be less disabling than later injuries with regard to certain elementary functions, yet much more disabling with regard to more complex levels of performance. What may be true of one type of behaviour may be quite different for another; structures important for early learning may be dispensed with later on (Hebb 1942, 1949).

Animal experiments have given little indication of such complexity, possibly because the range of behaviour studied has been seriously restricted. By contrast, investigations on brain-injured man may permit one to define empirically, those aspects of behaviour that are similarly affected by early and by late damage, and those that are differentially affected.

**Groups of Patients Studied**

Our investigations have all been concerned with the relationship between the central nervous system and behaviour. To this end we have concentrated on behavioural effects of focal brain wounds
in previously healthy adults and contrasted these effects with those of peripheral nerve injuries. A supplementary means of assessing brain-behaviour relationships has been to study the development of behaviour in the child and the alterations in such development with cerebral damage early in life. Thus, child neurology has been important for diagnosis and prognosis, but even more for studying the normal growth of cerebral function. Through observing aberrations of behavioural development we have hoped to obtain a better view of its true course. Three groups of cases, A, B and C, have been studied.

Group A: adults with brain injury. These have been our principal concern. Systematic studies have been made of sensory, motor and intellectual functions in 232 men who sustained brain wounds in World War II or the Korean campaign. Their performance was compared with that of 118 controls, tested under identical conditions, who had sustained penetrating injuries to one or several peripheral nerves.

Group B: Bellevue and other hospital patients suffering from brain tumours or vascular accidents. These gave us an opportunity to examine the acute stages of rather different types of lesions and compare them with our cases of gunshot wound, to observe the progression or regression of symptoms, and to study the role of retraining in recovery.

Group C: children. This group, which is supplementary to groups A and B, has been studied to show the development of perceptual functions in normal children, in children with brain damage or disease, and in children who have suffered from peripheral sensory loss, such as congenital blindness. We hoped that these studies would throw light not only on normal perceptual development but also on the question whether brain injuries sustained at birth or soon afterwards have a different effect from those sustained later in life.

Our main investigations have been the outcome of a survey of groups A and C. We have compared the performance of normal children, brain-damaged children, normal adults and brain-damaged adults on the same perceptual tasks. The most serious limitation of these comparisons is that the lesions are less well known and possibly more diffuse in group C than in group A. For this reason, further work comparing groups B and C might be desirable. On the other hand, by working primarily with groups A and C each age-group with brain injury had its own (normal) control group, and the follow-up was long enough (i.e., over 10 years in group A) to show whether the passage of a period of years after brain injury has the same or different effects in children and adults.

We can therefore try to answer these two questions: (1) Do the effects of a brain injury differ according to whether it is sustained early or late in the patient’s life? (2) Do the effects of early brain injury seem different according to whether they are studied soon after the injury or later—i.e., in infancy, or in adolescence or early adulthood? The answers to both questions have depended on the kinds of performance tested, and these different aspects of performance have been defined by adding a distinction based on the work with adults in group A whose brain injuries were sustained at maturity. The after-effects of their lesions seem to fall into two broad classes: (1) specific and localisable effects; and (2) general effects, which appear after a brain injury, irrespective of its site.

Specific and General Effects of Cerebral Lesions

We are all familiar with tasks that bring out permanent and specific effects of focal
lesions: visual-field defects after injury of central visual pathways, or localisable sensory or motor deficits. Changes of this type have been surveyed in great detail and with quantitative measures in group A—i.e., in adults with gunshot wounds (Teuber et al. 1960, Semmes et al. 1960). However, by adding other tasks or analysing the results in a different fashion, we have also obtained evidence of non-specific changes that are found after brain injury in any lobe, regardless of the presence or absence of specific sensory or motor deficits (Teuber 1959).

Hidden-figure Tests

Fig. 1 shows a hidden-figure test adapted by Teuber and Weinstein (1956) from Gottschaldt (1926, 1929). The subject is required to find the figure at the top within each of the lower embedding figures. Men with visual-field defects show increased difficulty in discovering 'hidden' figures. However, while field defects are sufficient to produce some deficit on this task, they are not necessary, since men without field defects perform equally badly.

Fig. 2 shows the mean number of figures correctly traced by controls (C) and by brain-injured subjects grouped according to the location of their wounds of entrance. At the extreme right of the figure one can see that the occipital group (O) was inferior to the controls, but so was the nonoccipital group (NO)—i.e., all brain-injured men whose wounds of entrance fell outside the occipital region. The same was true of the temporals and non-temporals (T and NT), the parietals and non-parietals (P and NP), and the frontals

Fig. 1. Sample page of hidden-figure test. (Adapted after Gottschaldt, 1926, 1929, by Teuber and Weinstein, 1956.)
and nonfrontals (F and NF); all are inferior to the controls, though the slight differences among these brain-injured subgroups were not significant. The same can be said of those with bilateral lesions (BIL) and those with right (R) and left (L) unilateral penetrations. Hence, when the hidden-figure data were analysed according to site of injury or symptoms there was no differentiation among the brain-injured: all did worse than the controls.

Hidden-figure tasks of this type are extremely difficult for children even in the absence of brain injury. Fig. 3 shows one example of a children's version of a hidden-figure task used in our laboratory by Dr. Lila Ghent (1956) with normal children of different ages. She found, as Witkin had done previously (1950), that these tasks are very difficult for children under 6 years. Mixed figures (as in Fig. 4) are somewhat easier for children. However, early brain damage affects the performance of either form of complex-figure test. Thus, Cobrinik (1959) demonstrated that on both tasks brain-injured children, at all ages tested (6–11 years), were significantly inferior in performance to normal controls. This inferiority still existed when brain-injured and normal children had been matched for mental age—i.e., the deficit on the perceptual task was not merely a reflection of lower intelligence. However, the difficulty with the hidden-figure and mixed-figure tasks in the brain-injured children was clearly related to the extent of their demonstrable neurological deficit. Those with severe motor impairment did significantly worse than those with mild or no impairment. Such results suggest that the severity, and possibly the size of the lesion after brain injury in a child finds expression in this difficulty with hidden- and mixed-figure tasks—i.e., with tasks which in the adult revealed the general effects of brain injury but did not differentiate the injury in one area from that in another. By contrast, certain tasks that bring out specific effects of injury in adults—i.e., effects restricted to injury in one but no other region—may be surprisingly insensitive to brain damage in the younger child.