Eur Arch Psychiatr Neurol Sci (1985) 235:129–133

European Archives of Psychiatry and Neurological Sciences

A Quantitative Relationship between the Extent of Localized Cerebral Lesions and the Intellectual and Behavioural Deficiency in Children

H. H. Kornhuber, D. Bechinger, H. Jung, and E. Sauer

Neurologische Universitätsklinik Ulm, Steinhövelstrasse 9, D-7900 Ulm, Federal Republic of Germany

Summary. In an investigation of 51 children with localized forebrain lesions (25 left hemispheric, 15 right hemispheric and 11 bilateral) a significant relationship between the extent of the cerebral lesion (quantitatively determined by means of computerized tomography scans) and the IQ was found, but only in children with lesions after age 5: there was a diminution of 3 (British Ability Scales and Porteus Mazes) to 4 (Wechsler test) points of IQ for a 1% increased brain lesion. There was no correlation between the extent of the lesion and IQ in children with early lesions. The IQ of the children with early lesions (up to year 4) was significantly lower than that of children with later lesions of the same extent.

There was a highly significant correlation between behavioural problems (assessed by Conners Teacher Rating Scale) and diminution of IQ. There was a significant correlation between the extent of lesion and behavioural problems in lesions after year 5, but not in early lesions. While the correlation between intellectual-behavioural impairment and the extent of cerebral lesions can be explained by multiple localisation of functions, the lack of this correlation in early lesions and the more severe impairment by earlier lesions may be due to an interaction of brain development with social and lesional factors. Since each part of the brain may act as a stimulus for the development of other parts, even small differences in the lesions (and/or in the environment) may result in large behavioural differences (hypothesis of the developing brain as an amplifier for differences in the stimuli).

Key words: Brain – Intelligence – Behaviour – Cerebral lesions – Training – Development – Education

Introduction

It has been known since the experiments of Lashley in the rat [8, 9] that localized cerebral lesions have two types of results: specific defects, depending on the location of the lesion, and general defects, depending on the extent of the lesion. To date, however, no quantitative measurement of the relationship between intellectual deficit in man and the extent of cerebral lesions exists in the literature although there have been attempts to correlate signs of severity of cerebral pathology (e.g. EEG abnormality) with behavioural data [11]. This may be, in part, due to the fact that in adult man, left hemispheric lesions result in aphasia which renders the measurement of intelligence difficult. In children, however, aphasia disappears rapidly. We decided therefore to investigate the consequences of localized cerebral lesions in children, using computerized tomography scans for the quantitative assessment of lesions.

The results showed that there is a significant relationship between the extent of lesion and the IQ: each percent of cerebral lesion corresponded to a reduction of IQ in the order of 3–4 points. This relationship, however, was apparent only with lesions from year 5 on. The lack of this correlation in children with earlier lesions may be due, in part, to differences in environmental stimuli and their interaction with the developing brain. A preliminary communication has been published [2].

Methods

The children were investigated in Pediatric Neurology (Department of Neurology) at Ulm University between 1982 and 1983. Only children with localized cerebral lesions (according to computerized tomography CT) were included. In this study the extent of the cerebral lesions was calculated by counting the standard matrix pixels included by the lesions over all CT scan slices [15] (Fig. 1) and expressed in percent of the brain. There were 51 children with forebrain lesions: 25 left hemispheric, 15 right hemispheric and 11 bilateral lesions. There were 19 anterior, 13 temporal, 15 posterior and 4 anterior + posterior lesions. While the temporal lesions included only lesions of the temporal lobe, the anterior cases included frontal, fronto-parietal and fronto-temporal lesions, and the posterior lesions included occipital, parietal, parieto-temporal and parieto-occipital lesions. The following investigations were done: standard neurological investigation, CT scan, Hamburg-Wechsler intelligence test (HAWIVA up to 6 years, HAWIK age 6–16 years, HAWIE from 16 years on), Porteus Maze test, British Ability Scales (BAS—our own German translation), short version of the Lincoln-Oseretzky test, Wisconsin Card Sorting, and assessment of the behaviour by means of Conners Teacher Rating Scale.

From the results of the subtests, I-values were calculated in order to compare the data from the Wechsler Test with the BAS. Dexterity was tested with a series of behavioural observations (writing, drawing, eating with a spoon, combing, hammering, throwing a ball etc.). The average age of the children

Offprint requests to: H. H. Kornhuber

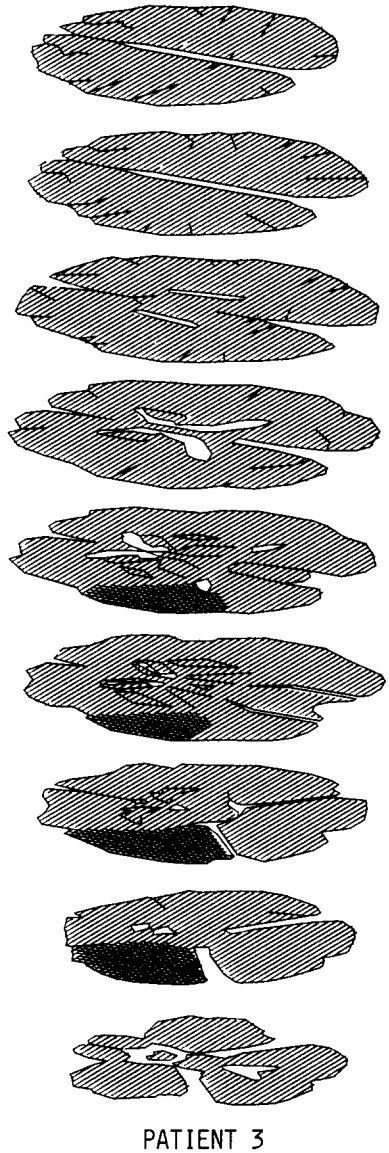

Fig. 1. Left temporal lobe lesion at age 9 years. Extent of the lesion 7% of the brain. Verbal IQ 89, performance IQ 115, full IQ 102 (Wechsler test).

at the time of the investigation was 11.5 years (SD 3.6). The time interval from injury to examination was on average 9.6 years (4.9 to 17.5 years). Although some of the children had aphasia immediately after the injury, none had any clinical sign of aphasia at the time of this investigation. There were 37 children with early (perinatal) and 14 with later (up to age 12 years) lesions. About 40% of both right and left hemispheric lesions had resulted in hemiparesis. Some of the children had epilepsy and were receiving anticonvulsive treatment, which did not affect intelligence [3].

For comparison 42 children without cerebral lesions from the Ulm University Clinic, Department of Pediatrics were investigated. Their average age was 11 years (SD 3.9). Their average BAS IQ was 113, average Wechsler IQ 119.

Results

IQ and Lesion. There was a highly significant negative correlation between the size of the forebrain lesion and the Wechsler IQ in those children in which the lesion had occurred after age 5 ($r = -0.73$, m $= -4.4$, b $= 122.3$, $P < 0.001$). The BAS results confirmed this ($r = -0.49$, m $= -2.6$; $P < 0.05$); in the BAS the significance was smaller partly because one child could not be investigated since the family had moved away. The Porteus Maze data also confirmed this relationship: $r = -0.49$, m $= -2.9$, $P < 0.005$. Thus, with the Wechsler test, 1% increase of forebrain lesion corresponded to 4 points lower intelligence, while with the BAS and Porteus Mazes 1% increased brain lesion corresponded to 3 points IQ. This result was the same for left unilateral lesions as for the total group (Fig. 2), and was not due to a heavier impairment of children with bilateral lesions. For unilateral left hemispheric lesions the Wechsler data were $r = -0.88$, m $= -4.9$, $P < 0.001$.

There was no correlation between the extent of the lesion and IQ in those children in which the lesion had occurred before age 5, most of which consisted of perinatal lesions (Figs. 3 and 4).

The IQ of the children with early lesions (up to year 4) was significantly ($P < 0.05$) lower (88) than the IQ of the children with later (from year 5 on) lesions (103), although the extent

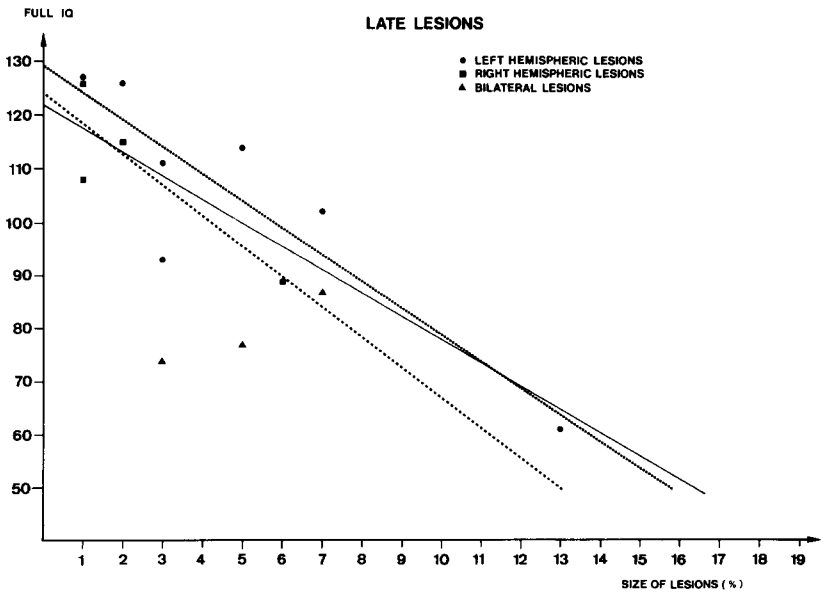

Fig. 2. Relationship between the full Wechsler IQ and the size of the cerebral lesions (percent of the brain) in children with lesions after year 5. The full line is the linear regression for all lesions ($r = -0.73$, $m = -4.43$, $b = 122.3$, $P < 0.001$), the broadely stippled line is the linear regression for right hemispheric lesions ($r = -0.87$, $m = 5.70$, $b = 123.8$), the densely stippled line (which is the uppermost line at the left side of the figure) is the linear regression for left hemispheric lesions ($r = 0.88$, $m = -4.92$, $b = 128.8$, $P < 0.001$)

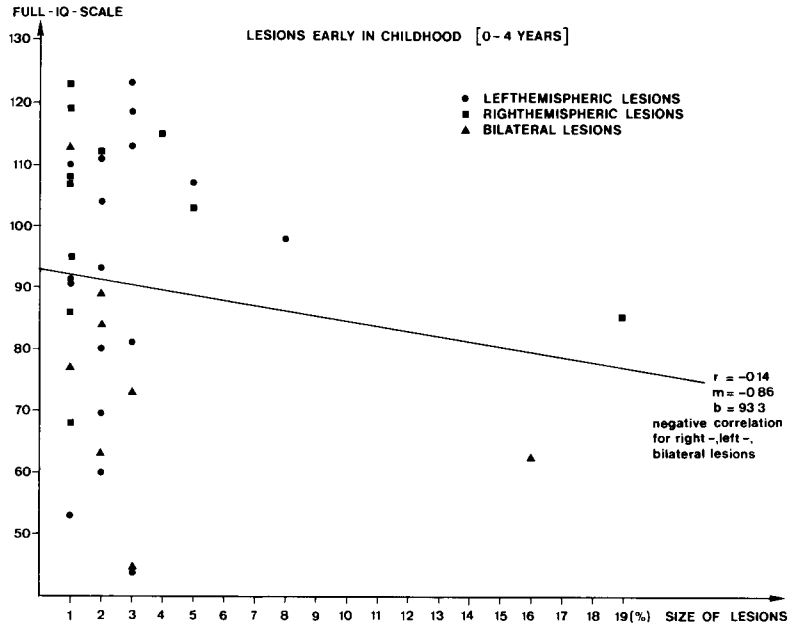

Fig. 3. There is no significant correlation between the size of the lesions and Wechsler IQ in children with lesions before age 5.

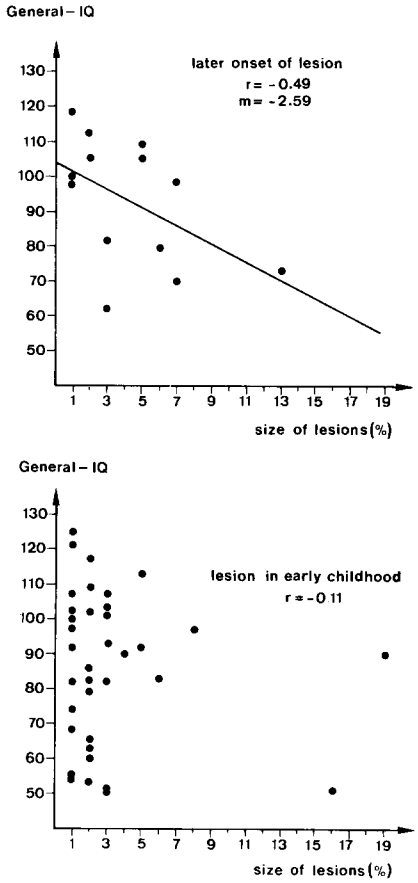

Fig. 4. Relationship between the general IQ (British Ability Scales) and the size of the cerebal lesions in the children with lesions after age 5 *(top)* and in children with early lesions (perinatal to age 4) *(bottom)*

of the lesions was smaller in the early lesions (3.2%) than in the later lesions (4.6%).

Hemispheres and Lobes. There was a difference between the lobes: the IQ was lower in left temporal and posterior lesions than in anterior lesions; however, the differences were insignificant when adjusted for extent of the lesion. On the other hand, there was a significant difference between the hemispheres: left hemispheric lesions impaired intelligence more than right hemispheric lesions of the same extent [5].

With the Wisconsin Card Sorting Test frontal lesions were worse in perseveration (when the task was to change the strategy) than posterior lesions ($P < 0.01$). This was true for both uni- and bilateral lesions and for right and left lesions. Unilateral frontal lesions had 6 perseverative errors, posterior lesions only 2.4, although the average extent of the posterior lesions was 3.4% of the brain while of the frontal lesions only 2.4% of the brain.

Behaviour, IQ and Lesion. For each behavioural disorder observed or mentioned by the parents, 1 point was given in the check list of 39 items of Conners Teacher Rating Scale; thus, the more points, the more disorder of behaviour. There was a highly significant negative correlation between IQ and the sum of the problem points in Conners behavioural rating scale ($r = -0.56$; $P < 0.0001$). In particular factors I (conduct problem), II (inattentive-passive) and IV (hyperactivity) correlated with lower intelligence. There was no correlation between the extent of the lesion and the points in Conners Scale in the early lesions ($r = 0.04$), but there was a significant positive correlation in the children with lesions from year 5 on.

Discussion

In view of the "mass action in cerebral function" in the rat [8, 9] it is not surprising to find a significant quantitative relationship between the extent of forebrain lesions in man and the corresponding impairment of intelligence, although the cerebral organization in the rat (with more important midbrain functions) is different from man (with high corticalization of functions). The "mass action" is probably due to the multiple localization of functions [7] and the multiple connections between cerebral centres. The values of 3 (with BAS) to 4 (with the Wechsler Test) points IQ per 1/100 of the brain found in this investigation offer only a rough estimate because of the

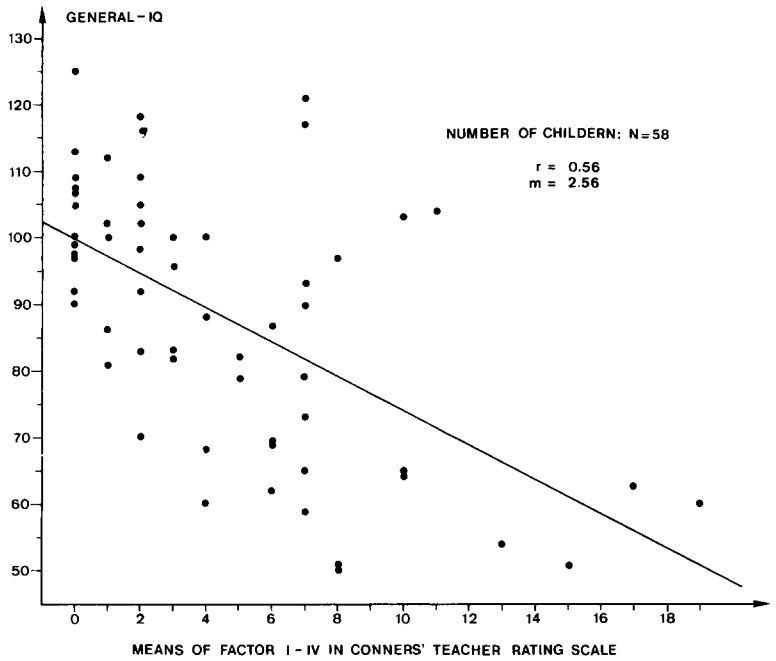

Fig. 5. Relationship between the general IQ (British Ability Scales) and the sum of factors I–IV in Conners Teacher Rating Scale. There is a highly significant negative correlation ($r = -0.56$, m $= -2.56$, $P < 0.0001$).

small number of subjects. This relationship seems to be valid for lesions up to 13% of the brain. The intercept (for children with zero extent of lesion) corresponds to 122 Wechsler IQ; this is in good agreement with our control series without cerebral lesions: their average Wechsler IQ was 119.

In contrast to this finding in lesions after age 5, no significant relationship between the extent of cerebral lesions and intellectual impairment was found in children with early (mainly perinatal) lesions up to year 4. One could argue that this lack of correlation in the early lesions may be due to the fact that the CT scan does not always show the full extent of the functional deficiency. However, this argument holds equally for the later lesions. The cause of the lesions before and after age 5 was probably similar because of the method of case selection starting from the CT scans: in all cases well localized lesions, not diffuse ones were chosen. Judging from the shape of the lesions, there was bleeding or infarction in most cases. Thus, the finding that there was not significant relationship between IQ and lesion size in the early lesions is not trivial; it may be a hint towards more differences of environmental stimuli and training in the early lesions. Environmental differences in infancy may have even more influence on development since the human brain grows rapidly at this age, with billions of synapses being formed: the development of the brain is an amplifier of environmental differences. The same may, however, be true for internal stimuli: small differences in the early lesions may, in the developing brain and its interplay with social environment, result in large differences in intelligence and behaviour.

There is another difference between the early and the later lesions. Early lesions of the same or even smaller extent impair the intelligence more than later lesions. As Fig. 4 shows, this was not due to the fact that only a small minority of the lesions was large; for this was so in both groups. The point is that among the early lesions there were many children with low intelligence despite a relatively small lesion. This was not due to our method of case selection which started from the CT, looking for well localized lesions. Comparable results, although without quantification of the extent of lesions, have been obtained by other investigators [10, 12]. The accumulation of low intelligence children among those with early lesions was not due to bilateral lesions, since among both the early and later lesions, just over one-fifth of the cases were bilateral lesions. Possible explanations for lower intelligence after early lesions are: a cumulative training deficit in the children with early lesions, and an internal deficit of stimuli for growth and connectivity (assuming that each part of the brain acts as a stimulus for the development of the other parts).

There are some reports in the literature which support these conclusions. An improvement of intelligence was found in persons who were relocated in childhood from an orphanage to adoptive homes when compared to persons of initially higher intelligence who remained in a less stimulating ortpanage environment for a longer period [13]. Such was not the case for the children of this investigation; all of them lived with their families. Those that had received early physiotherapy tended to have higher IQ, but the difference was insignificant. We know, however, from our epidemiological study of early treatment for language disorders [4] that the results depend on the intensity of training. Furthermore, the relatively small correlation of the mental abilities of normal toddlers with the intelligence of the same children later has been explained by the assumption that babies, as a result of variations in environmental stimulation, acquire varying degrees of ability within the limits of their developmental capacity; tests measure the acquisition of the abilities rather than the capacity [14]. These variations in environmental stimuli are diminished by school, starting from age 6. It is perhaps because of the uniformity of this influence, that the variability of IQ due to environmental factors decreases thereafter.

The correlation found between cerebral lesions and behavioural impairment (Conners Scale) shows that a cerebral lesion is a potential cause of (pseudo)neurosis. Furthermore, the correlation between the impairment of behaviour and intelligence indicates that compensatory encouragement and training may be beneficial for both. Learning and training are capable of promoting mental abilities [6] including the structure of intelligence [1]. Since learning and training, however,

depend on motivation and attention, defects in these dimensions of personality (in consequence of an interaction of early cerebral lesions and environment) may deeply influence intellectual development.

References

1. Amthauer R (1980) Test für Bildung und Beruf. Frankfurter Allgemeine Zeitung, Frankfurt/Main
2. Bechinger D, Jung H, Kornhuber HH, Sauer E (1985a) Quantitative Beziehungen zwischen lokalisierten Hirnläsionen und neuropsychologischen Leistungen bei Kindern. Dtsch Med Wochenschr 110:196–197
3. Bechinger D, Jung H, Kornhuber HH, Sauer E (1985b) Epilepsie und neuropsychologische Störungen bei Kindern: Größe und Lokalisation der Hirnläsionen. In: Kruse R (Hrg) Epilepsie 1984. 25. Tagung der Deutschen Sektion der Internationalen Liga gegen Epilepsie, Einhorn-Presse Verlag, Reinbek, S 298–304
4. Bechinger D, Kornhuber HH (1985) Neue Aspekte der Aphasieforschung. In: Rothe M, Frank G, Grziwotz-Buck P, Stöckle O, Walther T (Hrsg) Zentralbedingte Kommunikationsstörungen. Verlag Wartenberg & Söhne, Hamburg, S 133–142
5. Bechinger D, Kornhuber HH, Jung H, Sauer E (1986) Higher density of information processing capacities in the left cerebral hemisphere of man. Arch Ital Biol (in press)
6. Kern B (1930) Wirkungsformen der Übung. Münster, Helios Verlag
7. Kornhuber HH (1983) Wahrnehmung und Informationsverarbeitung. In: Der Mensch, Kindlers Enzyklopädie, vol III. Kindler, Zürich, S 597–620
8. Lashley KS (1929) Brain mechanisms and intelligence. University of Chicago Press, Chicago
9. Lashley KS (1931) Mass action in cerebral function. Science 73:245–254
10. Lehmkuhl G, Kotlarek F, Schieber PM (1981) Neuropsychologische und neurologische Befunde bei Kindern mit angeborenen umschriebenen Hirnläsionen. Z Kinder- Jugendpsychiatrie 9:126–138
11. Matarazzo JD (1972) Wechsler's measurement and appraisal of adult intelligence. 5th edn, Williams and Wilkins Co, Baltimore
12. Schneider R, Remschmidt H (1977) Der Einfluß des Schädigungszeitpunktes auf Wahrnehmung, kognitive und soziale Entwicklung hirngeschädigter Kinder. Z Kinder- Jugendpsychiatrie 5:317–345
13. Skeels HM (1966) Adult status of children with contrasting early life experiences. Monogr Soc Res Child Dev 31:(no. 3, serial no. 105)
14. Stott CH, Ball RS (1965) Infant and pre-school mental tests: review and evaluation. Monogr Soc Res Child Dev 30:no. 3
15. Wallesch CW, Kornhuber HH, Brunner JR, Kuntz T, Hollerbach B, Suger G (1983) Lesions of the basal ganglia, thalamus, and deep white matter: differential effects on language functions. Brain Language 20:286–304

Received April 25, 1985