Effects of Penetrating Brain Injury on Intelligence Test Scores

Despite centuries of interest in the effect of cerebral lesions on intellectual function, there is no agreement on the localization of those injuries which might produce maximal deficit on tests of "general intelligence." One might suspect that any brain injury of sufficient size produces lasting deficits, in analogy to Lashley's results for rats (*1*). Against this view are recurrent claims that only lesions in certain areas of the brain, especially the frontal lobes, are followed by a loss (*2*). The uncertainty is increased by the large number of reports describing "negative" findings—that is, the absence of demonstrable deficits in test performance, despite the presence of large cerebral lesions, especially of the frontal lobes (*3, 4*).

Most information derives from the testing of patients (often past middle age) with brain tumors or vascular accidents, and from cases of lobotomy, or similar forms of psychosurgery in psychosis, severe neurosis, or intractable pain. Estimates of the decline of intelligence, based on comparison with premorbid scores (*4, 5*) are rendered doubtful by the extent to which the disease process (brain tumor or psychosis) might have affected the patient's performance before operation or continued to affect it afterward. As a result, the information is usually based on abnormal preas well as postoperative scores.

To our knowledge, no studies exist which compare scores obtained on a standardized test of intelligence by patients after stable, localized brain injury, with corresponding scores achieved before injury, and at a time when the patient could be considered physically and mentally intact. Nor are there studies in which a sufficiently long period elapsed after injury to allow maximum recovery from the effects of the brain wounds.

The opportunity for such a study arose in the course of a research program evaluating the effects of penetrating brain wounds on various aspects of behavior. We were able to obtain preinjury scores on the Army General Classification Test (AGCT), a standardized test of "general intelligence" given each man on induction into the armed services, for 62 men who subsequently sustained penetrating brain wounds, and for 50 controls —men who subsequently incurred peripheral nerve injuries of the arm or leg. All men had been injured in World War II, 1 to 3 years after the initial testing.

Approximately 10 years after they had been wounded, these men were retested in our laboratory with a comparable form of the AGCT (First Civilian Edition). Since all men had been given thorough physical and at least cursory psychiatric examinations prior to induction, their preinjury scores can be considered as reasonably accurate estimates of intellectual capacity within the limits of such tests. Localization of the brain wound was based on surgeons' notes at initial *debridement,* subsequent craniotomy, and on roentgenographic evidence—that is, bone defect and retained foreign bodies. In view of the lack of histologic verification of the lesion, all localizations are considered tentative.

The preinjury scores of our control and brain-injured groups were practically identical: the mean of the controls was 106.4, that of the subsequently brain-injured group was 105.0. On the post-injury test, 48 of the 50 controls increased their score, yielding a mean of 119.4, or a mean increase of 13.0 points. This general tendency to gain was an orderly phenomenon, since the correlation (Pearson r) between pre- and postinjury scores was .902, indicating considerable retest reliability.

The 62 brain-injured men were subdivided into groups according to the estimated location of their lesions (frontal, temporal, parietal, occipital, in left, right, or both hemispheres), and the data were subjected to a series of analyses of variance. These analyses demonstrated that lesions of the frontal and occipital lobes did not produce a significant decline in score, and that only groups with lesions of parietal or temporal lobes of the left hemisphere showed a significant decrease. In order to investigate further the role of these regions, a subsequent analysis was performed which compared men with lesions involving either or both of these lobes with those in whom these lobes were spared (*6*). Figure 1 (black bars) shows that, although the group with lesion to the left temporal lobe showed a slight decrease in score, the most striking drop was exhibited by the group with left parieto-temporal lesion. In comparison with the large gain shown by the control group, the group with left parietal lesion showed only a slight increase on retesting; the groups with lesions in left nonparieto-temporal regions or lesions in the right hemisphere showed greater gains, closer to that of the control group.

One might suspect that the inferior performance of the groups with lesions to the left parietal and temporal lobes (either or both) was entirely due to the presence of aphasia. Indeed, when the analyses were restricted to nonaphasics (striped bars), the groups showed somewhat higher scores; and the group with left temporal lesions now exhibited a gain, rather than loss. The left parieto-temporal group alone still showed a loss in score, illustrating the statistically significant inferiority of this group to all other groups.

The concentration in a particular area (left parietotemporal) of those lesions which, in the absence of aphasia, produced a demonstrable AGCT deficit, opposes the view that impairment is

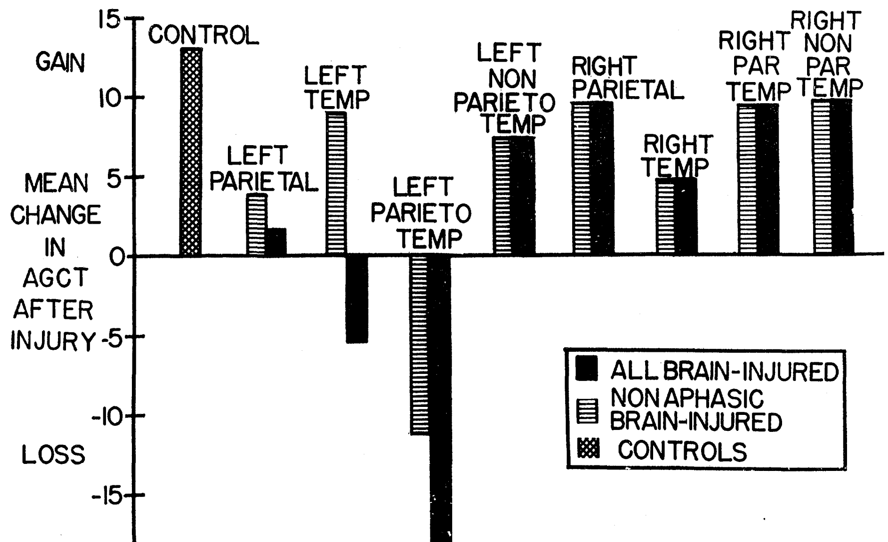

Fig. 1. Mean change in score from pre- to postinjury on Army General Classification Test for controls and for brain-injured subgroups, including and excluding aphasics. The median change and N (parenthetic) for the groups are as follows: Controls (50) 12.5; left temporal (4) −0.5; left parietal (11) 3.0; left parietotemporal (10) −10.0; left nonparietotemporal (9) 14.0; right temporal (9) −1.8; right parietal (7) 10.0; right parietotemporal (2) 9.5; and right nonparietotemporal (10) 12.0. The median change and N for the nonaphasic groups are as follows: left temporal (2) 9.0; left parietal (10) 4.0; and left parietotemporal (6) −9.0.

equal after lesions in any lobe (*1*) or that it should be maximal after injury to the frontal regions (*2*). There is, however, evidence that the present results are limited to the kind of test employed. Using the same population, we found that injury to any lobe of the brain, in either hemisphere, can interfere with performance on certain nonlanguage tasks such as the discovery of hidden figures (*7*).

Performance on a standardized test of "general intelligence," such as the AGCT, thus shows little or no change 10 years after penetrating brain wounds unless the entrance wound included the left parietotemporal region.

SIDNEY WEINSTEIN
HANS-LUKAS TEUBER
Psychophysiological Laboratory, New York University–Bellevue Medical Center, New York

References and Notes

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Radiocarbon Dates of Mankato Drift in Minnesota

The Mankato drift in Minnesota has recently been shown by Wright and Rubin (*1*), on the basis of radiocarbon dates, to be older than the Two Creeks forest bed in northeastern Wisconsin. In a paper (*2*) that was prepared and submitted for publication in June 1956 but which has just been issued, I reached the same conclusion for the Mankato drift of northern Lower Michigan. But Wright and Rubin *1,* p. 626 express the view that the Mankato drift "should be correlated with the Cary (pre-Two Creeks) rather than with the Valders (post-Two Creeks)." And they further state, "We therefore favor the use of the term *Valders* over the term *Mankato* for the last major substage of the Wisconsin." The question was not discussed that, even though the Mankato is older than Two Creeks, it might still be younger than the Cary, which I show in my article to be the case.

In northern Lower Michigan, as pointed out by Leverett long ago (*3*), the terminal moraine of the Mankato, the Port Huron, transects the last known interlobate deposits of the Lake Border system of late Cary age. Before it was built, the desiccation of the Cary ice had taken place. After a short intraglacial interval, the Cary-Mankato, the Mankato glacier formed from the renourished Patrician center and moved southward across the Straits of Mackinac to its terminal position (see *2,* Fig. 1). Leverett, the revered apostle of glacial-lobe deployment, pointed out that morainic systems which were built following marked readvances like the Port Huron system should be recognized as substages (*3,* p. 31). It is not a "recessional" of the Cary. Likewise, the red drift of the Valders is not a "recessional" of the Mankato.

In Minnesota a similar situation exists. Leverett's map (*4,* Plate 2) shows that immediately south of the Bigstone moraine in northwestern Minnesota, southeast of Ortonville, elongate ridges, resembling crevasse fillings, occur which are transected by the Bigstone moraine (Fig. 1), in the same way that the Port Huron moraine crosses the interlobate pattern of the Lake Border system, except that in Minnesota the transecting moraine is Valders and the deposits out in front are Mankato.

Upon searching for what Leverett might have said about this significant relationship, I find this (*4,* p. 105): "The morainic strips just mentioned [those out in front of the Bigstone moraine] seem to pertain to a very narrow ice tongue that was gradually reduced until it completely melted before the ice sheet readvanced with a broader front to a strong moraine that traverses southern Big Stone County and northwestern Swift County. . . . They complete the series which deployed in the basin drained by the Minnesota River and were followed by a series of moraines formed by an ice lobe that was confined mostly to the Red River Basin." Leverett recognized this as a younger and separate substage of the Wisconsin (*5,* Fig. 5).

Thus, it would appear that the retreat of the Mankato ice gave rise to Lake Agassiz I, that the Valders ice transgressed most if not all of the lake bed, and that the outlet waters of Lake Agassiz II, upon the retreat of the Valders ice from the Bigstone moraine, cut River Warren Valley II. This narrow gorgelike valley is clearly related to the marked change in topography brought about by the deposition of the Bigstone morainal system. As Leverett observed, it is in contrast to the much wider valley of River Warren Valley I, below the terminus of the moraine. Thus, the present beaches are younger than the Valders terminal moraine, although some of the recent topographic maps seem to show short stretches of slightly glacially modified beaches of Lake Agassiz I.

It is also interesting to note that the two valley-train terraces along the Mississippi River in western Illinois, which have been assigned to the early and late Mankato by Leighton and Willman, are but mere remnants of former fills, caused by two successive glacial torrents, the first greater than the second. The new facts throw interesting light on these interpretations. Leighton and Willman have consistently referred the destructive torrents which not only swept away most of the valley trains but sliced the bluffs and headland points in the Mississippi Valley, to the outlet waters of Lake Agassiz I and II and Lake Duluth. More recently Leighton has traced the upper and older terrace that is composed of coarser materials to the Mankato outwash to Dakota County, Minnesota, where it has been mapped as such by Ruhe and Gould (*6*). Now it appears that the lower terrace of finer materials may be Valders and may have come from the ice when it stood at the Bigstone moraine. The terrace at Bonfils, Missouri, across from St. Charles, which was formerly referred to the Mankato, is now known to be Tazewell, from two radiocarbon dates determined by the Washington laboratory. The Tazewell date is consistent with the fact that this terrace remnant has loess on its surface which is essentially lacking on Mankato or Valders terraces.

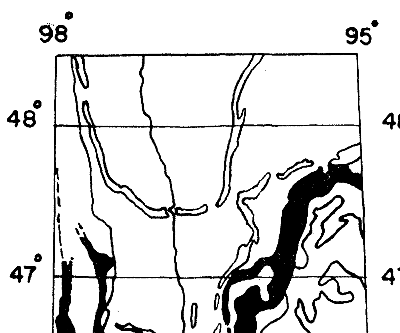

Fig. 1. Bigstone morainal system in the Red River basin, suggested as Valders. The outer portion of Valders is shown in black, Mankato open, Cary dotted, Tazewell diagonally lined, and glacial Lake Dakota horizontally lined [After Leverett (*4,* plate 3) and Flint (*8*)].