

# A Counterpoint to Reitan's Note on the History of Clinical Neuropsychology

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In a recent commentary published in this journal, Reitan (1989) struck a cacophonous note, sharply criticizing two reports alluding to the development of clinical neuropsychology. Hartman (1988) characterized neuropsychology as a discipline originally aimed at identifying the "3L's," namely Localization, Lateralization, and Lesion detection. Similarly, Mapou (1988) proposed that neuropsychology should no longer be directed at brain-damage detection as it was initially intended, but rather, should be focused primarily at evaluating specific cognitive domains. Reitan (1989) castigates both authors, trumpeting that "there is a prevalent notion that the beginnings of neuropsychology were devoted rather exclusively to the development of techniques capable of identifying brain lesions" (p. 386), and later chiding that the above conclusions "could not have been reached without a profound ignorance of the history of clinical neuropsychology. Perhaps it is time that the ill-informed resisted the temptation to impose their generalization on the rest of the profession" (p. 387).

## *Neuropsychology in the 1950s*

One can certainly understand how Hartman and Mapou may have come to their conclusions. The tenor of their remarks is in remarkable harmony with the tone established by Reitan in the 1950s. In 1959, Reitan prepared a manuscript containing two reports, "Principles used in evaluating brain functions with psychological tests at the neuropsychology laboratory, Indiana University Medical Center," written by Reitan, and "Hypotheses supported by clinical evidence that are under current investigation," by Reitan and Kløve. Reitan asserted explicitly that "the Halstead Impairment Index serves as a valid and reliable basis for

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inferring the presence or absence of brain damage in individual subjects” (p. 1). Reitan further elaborated that “several studies have yielded results differentiating patients with lesions of the left hemisphere from patients with lesions of the right hemisphere at acceptable levels of statistical significance” (p. 4). For example, “disturbances in simple perceptive abilities . . . often prove to be of valuable aid in lateralizing brain lesions” (p. 5).

Reitan and Kløve discussed lesion localization and the effects of different lesion types on neuropsychological performance. “Absence of any [sensory] suppression argues against an acute, destructive lesion (glioma, cerebrovascular accident, etc.) in the posterior part of the hemispheres” (p. 16). “If both the Category Test and Part B of the Trail Making Test are poorly performed and other tests are on a near normal level, a focal and static lesion of one or both anterior frontal lobes is implied” (p. 17). Even in his commentary in which he lambastes Hartman and Mapou, Reitan (1989) recounts, “the initial approach in validation . . . [was] . . . to focus on the question of brain damage” (p. 388–389). “The next step in validation . . . [was] . . . to see if a patient’s condition could be identified on the basis of the test results alone. . . . Neuropsychological data . . . are sufficiently powerful to overcome the broad range of ‘distracting’ variables and reflect the organic condition of the brain” (p. 389). Thus, Reitan’s own writing firmly established that Localization, and Lateralization, and Lesion detection were major goals of clinical neuropsychology at that time.

Reitan (1989) contends the field of neuropsychology began to develop only when the study of brain–behavior relationships replaced the desire to diagnose brain lesions. “There is a fine line of difference between detecting brain damage or dysfunction and the *clinical* evaluation of brain–behavior relationships” (p. 386, italics mine). Although there is considerable subjective variation present when contrasting scientific and clinical investigations of brain–behavior relationships, the clinical evaluation of brain–behavior relationships and detecting brain damage or dysfunction share a common theme emphasizing the disease process. My informal poll composed of staff physicians and psychologists in neurosurgery, psychiatry, and neurology failed to uncover a single soul who could appreciate this nuance. If such a distinction genuinely exists, it is sufficiently fine that Mapou and Hartman should hardly be chastized for characterizing the early development and practice of neuropsychology as “detecting brain damage” rather than “the clinical evaluation of brain–behavioral relationships.”

### *Philosophy of Science*

In his attempt to “lend more reality to current reports,” Reitan flaunts contempt for the epistemological development of neuropsychology. “Theories are conceptual formulations that become necessary because of a paucity of facts” (Reitan, 1988, p. 331). However, the science of brain–behavior relationships is not simply an accumulation of facts (Kuhn, 1966). Reitan fails to differentiate

between models and theories in neuropsychology, and the instruments designed to test the hypotheses that are derived from models/theories. There is a rather conspicuous line of difference between the *scientific* inquiry of brain–behavior relationships, and examining test performance in a clinical population. The former determines relationships between distinct brain structures and their functional interconnections, and how they contribute to psychological and cognitive operations. This relationship is initially hypothesis driven, both in terms of defining brain regions and psychological constructs, and tests are only subsequently selected to assess the behavioral construct of interest. In this context, the psychologist has the flexibility to use the scientific method. In the latter, the relationship of behavior to brain function is limited to correlating tests with brain pathology. However, the starting point is the test, not the hypothesis. Brain–behavior relationships cannot be defined exclusively as those qualities assessed by the Halstead–Reitan battery.

The model of brain–behavior relationships orchestrated by Reitan was tested “through prediction . . . in the individual case, of the biological status on (sic) the brain as discerned through detailed clinical study (including autopsy in many cases) by neurologists, neurosurgeons, and neuropathologists” (Reitan, 1988, p. 337). However, a model of brain–behavior relationships derived from patterns of performance in patients with structural lesions “identified by neurologists, neurosurgeons, and neuropathologists” cannot be easily generalized to patients without visible structural concomitant. Consequently, Reitan is bewildered when Hartman does not directly address the usefulness of the category test for assessing general neuropsychological impairment in patients with, for example, neurochemical lesions. However, Reitan’s model exists independently from whether or not the category test is affected by medication toxicity.

It is imprudent to claim that “neuropsychological evaluation [frequently] presents the only positive evidence [of diffuse or generalized cortical damage]” (p. 387). Without an appropriate model, and appropriate testing of that model, there is no bridge from positive neuropsychological findings to neuronal networks (e.g., physiologic or anatomic). Clinical brain–behavior relationships without positive neurologic findings are simply behavior–behavior relationships, and purely psychometric definitions of function must be relied upon. “The *neuro* part of neuropsychology must also play a role unless the intent is to practice clinical psychology rather than clinical neuropsychology” (Reitan, 1989, p. 386).

Reitan cites the work of both Halstead and himself to indicate the early interest in the “broad range of other variables and conditions that could have an effect on brain function” (p. 388). However, he augments his own contribution since many of the “neuropsychological findings” to which Reitan refers are limited to Rorschach patterns in myxedema (Reitan, 1953), Rorschach findings in essential hypertension (Reitan, 1954a), Rorschach performance in chronic brucellosis (Reitan, 1954b), and MMPI results in affective and brain-damaged patients (Reitan, 1955). Thus, the Halstead–Reitan battery was not administered

to as many diverse groups as one is led to believe when reading Reitan's commentary, and his model of brain-behavior relationships has not been systematically studied in populations without identifiable structural lesions. Interestingly, in the one article cited examining sedative medication effects on standardized psychological tests (Reitan, 1957), the only test employed from the Halstead-Reitan Battery was finger tapping. Reitan appears trapped by his own caveat, in which he cautioned "the information selected to provide the basic structure is sometimes composed of only partially true facts" and "facts are often selected by a theorist because they support his/her preexisting biases or interests and, therefore, do not represent a complete or impartial representation of all available facts" (Reitan, 1988, p. 332).

### CONCLUSION

Reitan was undeniably one of the founders of neuropsychology, and one cannot diminish his contribution to systematized assessment. However, his commentary on "the History of Clinical Neuropsychology" is restricted to the works of Halstead and Reitan. He chooses to ignore many pioneers such as Teuber, Luria, Bender, Hécaen, DeRenzi, and Benton, who conducted extensive research and contributed classical works to the clinical and scientific literature of brain-behavior relationships. The key to understanding clinical neuropsychology's maturation requires appreciation of its movement toward performing different roles depending on the clinical setting, knowledge, and sophistication of referral source, and specific questions to be answered by the neuropsychological evaluation. Just as it is sometimes necessary to revise certain tests that are rendered obsolete due to changes in a population (e.g., Wechsler-Bellevue Intelligence Scale), it is sometimes necessary to modify our scientific models of brain-behavior relationships to accommodate new "knowledge."

As Reitan (1989) himself admonishes, "we should not denigrate knowledge . . . in order to inflate the importance of our current 'intelligence.'" (p. 390) Bravo.

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