Decomposing the Corpus of Neuropsychological Tests

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Abstract

This commentary has had two primary concerns: We have attempted to address the difficulties and possibilities of using cognitive tests to measure the underlying biological basis for mental disorders. In particular, we have argued that to understand and interpret test results in psychiatry appropriate models of cognitive brain operations on a process-analytic level rather than on a psychometric level are essential. We have also discussed particular results of Abbruzzese et al.'s study, pointing out alternative interpretations of their perseveration results and some preconditions for demonstrating a true double dissociation. Rather than proceeding from the assumption of a unitary disease entity, we prefer to view "schizophrenia" as heterogeneous in its neuropathological and cognitive processing substrates, reflected in Bleuler's term "the schizophrenias."

Commentary on: Abbruzzese, Massimo and Ferri, Stefano and Bellodi, Laura and Scarone, Silvio (1993) Frontal Lobe Dysfunction in Mental Illness, Psycoloquy: 4,#9 Frontal Cortex (1)

Keywords: cognitive disorder, dorsolateral prefrontal cortex, mental disorder, neuropsychology, obsessive-compulsive disorder, orbitofrontal cortex, psychosis, schizophrenia

I. INTRODUCTION

1.1 In what follows, we present a critique from a cognitive science standpoint of the form of inference to be found in the target article by Abbruzzese, M., Ferri, S., Bellodi, L., and Scarone, S. (1993). For the most part these authors adhere to the conventions of traditional neuropsychological argument. In Table 1 we display their principal findings:

TABLE 1: Neuropsychological test outcome in Abbruzzese et al's study, in terms of whether each of the patient groups performed significantly worse ("*") than the normal group on the four tests: Verbal Fluency, Weigl Sorting, Wisconsin Card Sort: Perseveration Index, and the Object Alternation:

Perserveration Index. Note also that only the VFT and OAT:P reliably distinguished between performances of the two psychiatric groups.

VFT WST WCST:P OAT:P ---------- paranoid schizophrenic * * * = obsessive-compulsive = = = *

Since schizophrenic patients have been reported to perform poorly on almost every available cognitive test, only their normal attainment on the OAT perseveration index holds potential interest. We dispute Abbruzzese et al.'s interpretative assumptions regarding these data as follows: Concerning the conclusions of Abbruzzese et al., we hold that data of the sort presented are in principle incapable of sustaining direct inferences about pathoanatomy. Neither can they afford a neuropsychological "profile" of frontal functioning, since we have no persuasive theory of the critical attributes of these tasks.

II. ON THE EXPLANATION OF BRAIN-BEHAVIOR RELATIONS IN COGNITION

2.1 We take it that the ultimate explanatory objective in the study of brain-behavior relationships is to relate elementary cognitive operations (understood in relation to a detailed model of normal cognitive functioning) to neuroanatomy. In the ideal case, for neuropsychological data to be brought to bear on the resolution of such issues, the necessary preliminaries would include: (i) analysis of the behavioral tasks used into the specific cognitive processes required for performance, as well as any strategic control processes governing the activities of the various specialized processors. (ii) A means of determining what neuropathology was present or a means of inferring the nature of the dysfunction, formulated with reference to a theory of normal functioning and supplemented by a specification of any putative compensatory rearrangements. (iii) In addressing the correspondences between cognitive model and neuroanatomy, we believe that describing the pattern of connectivity amongst the neural elements is likely to be more important than their absolute location in the skull.

2.2. NEUROPSYCHOLOGICAL TESTS: These require from the subject a complex composite of cognitive operations, usually involving specialized processors whose function is governed by relatively nonspecific, strategic control processes. If the "neuro-psychological" tests are to attach to the expectantly dangling "neuro-" anything of psychological substance, one must confront these complexities and provide a systematic explanation instead of attempting to overleap such difficulties in the hope that the test may conduct us directly to its anatomical home. Where the frontal lobes (FLs) are the focus of enquiry these difficulties may be especially severe, if as many have supposed, the FLs largely subserve rather general monitoring, planning and coordinating functions. The FLs have been implicated in many disorders and it seems likely that they play a role in most cognitive skills. Yet neuropsychological tests are sometimes remarkably insensitive to extensive FL damage (Eslinger & Damasio, 1985).

2.3 BRAIN DAMAGE AS A MANIPULATION OF THE SYSTEM: For the privilege of addressing the rich cognitive skills with which humans are endowed and the manner of their breakdown, the

neuropsychologist has to pay a considerable price in terms of uncertainty about the nature and extent of the lesions resulting from accident or the unpredictable progress of the disease processes. By way of compensation, although brain damage cannot be systematically manipulated as an experimental variable, a powerful quasimanipulation can be achieved by selecting cases whose outcome has been the loss of a circumscribed process or skill; in contrast, laboratory manipulations applied to normal individuals merely result in performance that is slightly slower or less accurate.

2.4 For studies of disorders such as schizophrenia, the price is especially high, because neither aetiology nor pathoanatomy are known. Moreover, the variation across patients in surface clinical features and in any evidence of underlying pathology is so great as to suggest that treating schizophrenia as unitary at the diagnostic, pathoanatomical or cognitive-defect levels is futile. Although postulating subtypes has since the time of Kraepelin seemed a possible means of salvaging a disease concept, the traditional categories of schizophrenia such as "paranoid" have shown little validity, having neither cohesion at the level of clinical features nor coherence at an interpretative level. At least three mutually compatible explanations of this diffuseness of abnormalities suggest themselves: (i) heterogeneity within the schizophrenic population, (ii) plurality and diversity of abnormal features within the individual and (iii) the nonspecific nature of the schizophrenic cognitive defects.

2.5 METHODOLOGY AND HETEROGENEITY: The assumption that schizophrenia is aetiologically, neuropathologically or functionally unitary is difficult to maintain in face of its extreme heterogeneity. The problems with generalizing about a heterogeneous group can be reduced by adopting a symptomatological rather than nosological approach to the selection of patients and explicanda. In Frith's (1992) carefully pondered version of this approach, nosological categories are only used to control comparisons. For example, the appropriate method for investigating any role of DLPC hypoactivity (PET) in abnormal saccadic distractibility would consist in comparing schizophrenia groups who did and did not exhibit this abnormality. In contrast, the single case-study approach currently favoured in cognitive neuropsychology simply begs the question of how to achieve generalizability, in circumstances of extreme heterogeneity (e.g., Caramazza, 1988).

2.6 Just as the heterogeneity across patients within the same nosological category makes doubtful the notion of a unitary disease (Andreasen & Olsen, 1982), the diversity of abnormalities found in the typical patient obliges us to reconsider the intuition that a particular mental illness must be susceptible to a parsimonious account at a functional level. We need to estimate the amount of explanatory parsimony that might be achievable by postulating particular cognitive defects, each subsuming several surface features. This requires, in turn, assessment of the tendency of signs and symptoms to co-occur (cohesion) and the susceptibility of each cluster to a single-mechanism account (coherence). It is possible that performance is impaired on a broad range of tests, however, because the cognitive processes that are defective are not those relying on specific processors but rather the pervasive, nonspecific processes that coordinate and monitor the activity of other processors.

III. ON THE INTERPRETATION OF ABBRUZZESE ET AL.'S SPECIFIC RESULTS

3.1 Abbruzzese et al.'s main objective is to obtain a more differentiated view of frontal lobe function. To this end, the selection of schizophrenia for positive symptoms (paranoid schizophrenia) seems ill-advised, for the features that have been shown to be associated with frontal dysfunction are negative features such as apathy or features with ambiguous polarity such as distractibility (see Frith, 1992)

3.2 Abbruzzese et al. attempt to use schizophrenic vs obsessive- compulsive group differences in test scores to infer a double dissociation between lesion sites. However, to establish such a dissociation, several preconditions would have to satisfied. The demonstration would be suspect if either group showed equal abnormality in tests that purport to be sensitive to dysfunction in other brain regions. Moreover, it would also need to be established that patients with lesions restricted to nonfrontal regions perform normally on these tests. Contrary evidence abounds. The WCST is sensitive to temporal lobe dysfunction. Performance of VFT induces activity in widely distributed brain regions (PET studies) and is sensitive to a particularly broad range of disorders. The lack of any such control measures in Abbruzzese et al. reveals a general failure to design for falsifiability rather than corroboration.

3.3 Conferring the status of an anatomically decisive result on the WCST/OAT contrast is arbitrary. The performance of the schizophrenic patients was significantly worse than that of the controls on three of the tests and worse than the obsessive-compulsive patients only on the WST. Moreover, the fact that the two tests, set in opposition, both purport to yield measures of perseveration is a reminder of the cognitive opacity of such tests. The perseveration indices are as opaque as the undifferentiated error scores and it is necessary to consider the nature of the tasks and the types of error they allow to interpret particular patterns of stereotyped response (see Freedman, 1990). The frequency with which stereotyped responses occur and the form they take varies between different schizophrenic subgroups, between tasks, and between different ways of presenting a given tasks (Frith, 1992).

3.4. Whereas a true double dissociation would reflect a categorical distinction between processing operations recruited by either test, the obtained pattern of results is at least as likely to reflect variation in one of the pervasive nonspecific requirements of such tests. For example, perfect OAT performance can be obtained with a working memory load extending back a single trial. We suspect this is why it is impaired in schizophrenia. The WCST requires information accrual over a longer background period. At the extreme stands the VFT, which is saturated with working memory and strategic control factors, required for keeping track of output, avoiding response repetition, and locating fresh clusters whilst unpacking those already located.

IV. CONCLUSION

4.1 This commentary has had two primary concerns: We have attempted to address the difficulties and possibilities of using cognitive tests to measure the underlying biological basis for mental disorders. In particular, we have argued that to understand and interpret test results in psychiatry appropriate models of cognitive brain operations on a process-analytic level rather than on a psychometric level are essential. We have also discussed particular results of Abbruzzese et al.'s study, pointing out alternative

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