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Commentary on "Cognition and control of action in psychopathology" (D. Widlöcher and M.-C. Hardy-Bayle)

PLEASE ADD NEUROPSYCHOLOGY !

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The questions raised by Widlöcher and Hardy-Bayle (hereafter WHB) in their cogent and well-documented analysis of the cognitive approaches to depressive and schizophrenic states are fundamental both for our understanding of some aspects of these pathological mental conditions and for a critical evaluation of the explanatory power of the present cognitive models of human cognition. The problem raised by WHB can be summarized as follows: over the past 20 years, the experimental approach to cognitive disorders in depressive and schizophrenic states has not provided clear and unchallengeable results pointing to the alteration of specific processing components. On the contrary, the cognitive primary disorders that have been evidenced cannot be adequately captured by modular models of the cognitive architecture since contradictions or replication difficulties have been the rule rather than the exception. At the root of such a critical inventory, WHB postulate that the core of the disorders is general rather than specific and that a general deficit in the temporal regulation of action is present in the depressive stages, and a general planning of action deficit in the schizophrenic states.

While the assumptions put forward by the authors seem reasonable and are based on several items of indirect evidence, we are not entirely convinced by their argumentations and by their critical appraisal of the literature. Indeed we think that some methodological and theoretical problems have not been correctly solved.

We shall limit our discussion to the depressives and most of our argument will be based on a comparison with similar problems in neuropsychology. Finally, we will argue that, in addition to the contacts present between psychopathology and cognitive psychology, what is presently required is a stronger relationship between psychopathology and cognitive neuropsychology.

WHB's position can be examined by answering three questions:
Is the negative evidence sufficient?
Why should one look for a common cognitive disorder?
Are WHB's theoretical suggestions operationalizable?

Is the negative evidence sufficient?

When one looks at the attempts to situate the locus of the mnesic disorder in depressive states, one is impressed by the similarity they present with the series of studies and theoretical interpretations that were produced in neuropsychology with regard to amnesic syndromes. In fact, exactly the same dissociations and the same tentative interpretations were proposed and more or less the same experimental work was performed. Amnesic syndromes have been explained by encoding, retrieval, or consolidation deficits, by a lack of sufficiently deep processing at the encoding stage, by an exaggerated sensitivity to pro- and retroactive interferences, by the influence of contextual parameters, and so on (Van der Linden, 1989). In the same way, many in neuropsychology have pointed to the same weakness in the organization of these studies, and several authors have stressed: the excessive variety of experimental paradigms, the lack of sufficient replication studies, the heterogeneity of the populations, and so on. The consequence of such, almost classic, disorganization of a vast international research enterprise did not lead neuropsychologists to abandon their objectives but progressively to admit that the introduction of the cognitive perspective to neuropsychology required a drastic modification in patient-selection methodology. Under the influence of the work and theoretical contributions of Marshall and Newcombe (1973), Marin, Saffran, and Schwartz (1976), and Shallice (1979), it has been suggested that the identification of an impaired component in a cognitive architecture is only realizable on the basis of selected and contrasted single-case studies. The theoretical reasons for such an imperative

methodological constraint are several and have been discussed in detail by Caramazza (1986). In the context of the present discussion, it suffices to stress that the principal reason for such a single-case approach resides in the uncontrollable heterogeneity of the pathological conditions (the brain lesions) that underly the neuropsychological disorders.

WHB acknowledge briefly in their article that the heterogeneity of psychiatric disorders may be a source of difficulty, but this eventuality is not seriously considered, which is probably because WHB, as did neuropsychologists in the past, have essentially in mind quantitative variations in the expression of a unique underlying deficit. Nevertheless in neuropsychology, it has become evident that variations in disorders generally correspond to qualitative differences in subjacent deficits. Thus, if it is reasonable to postulate that there is heterogeneity within the classic psychiatric syndromes and if that inter-subject variability concerns subjacent (secondary or primary) cognitive disorders, then we are in a situation in which any attempt to locate a deficit in a precise cognitive architecture on a group basis may well be impossible. The lack of coherence in the data gathered with the present cognitive approach in psychopathology is thus not very surprising. The question WHB have to solve is thus the following: What evidence is there for the cognitive homogeneity of the classic psychopathological syndromes?

Why a 'common' cognitive disorder?

Another arguable point in WHB's position is their attempt to be economical in their interpretation. Why are psychopathologists looking for a "common" or "unitary" cognitive deficit subjacent to syndromes in their psychiatric taxonomy? Is it not equally probable that the result of a cognitive analysis may be to divide or to disentangle their present classifications? Actually this is precisely what has occurred in neuropsychology: our classic taxonomies either were divided or simply lost their relevance (Marshall, 1986). The question of economy is of course not unrelated to the problem of syndrome homogeneity, and we sometimes have the impression that, being unable to guarantee the homogeneity of their syndromes on the level of mood or behavioral characteristics, the psychopathologists trying to find another organization principle with the cognitive approach, but this is still a conservative strategy! Even if we can understand the reasons for such a research strategy, at least for practical and clinical purposes, we are not sure it represents the best way to look at cognitive disorders in psychopathology. We think it could be better to bracket the classic taxonomy or to use it only as a first approximation, and to look at the cognitive functioning of well-selected single patients.

A reason for not searching for a unitary common deficit could also be advanced from data from the psychopathology itself. There are studies whose results seem difficult to interpret in the theoretical framework proposed by WHB, and there are also alternative interpretations of cognitive deficits in depression that WHB have, in our opinion, too quickly dismissed.

First, there are results on cognitive deficits in depression that seem difficult to interpret in terms of reduction of action activation. For example, it is not clear how the results of Colby and Gotlib (1988), showing a short-term memory deficit in depressive subjects only at delays of 20 and 30 sec but normal functioning at a 1 sec delay, would be interpreted by WHB. Moreover, it is difficult to explain why depressive subjects experience difficulties in fluency tasks prompted by semantic category but not in fluency tasks prompted by alphabetic category (Calev, Nigal, & Chazan, 1989).

Second, the authors have too quickly eliminated alternative interpretations of cognitive deficits in depression. There are at least four main interpretations that have to be considered.

The first one suggests that depressives perform better on more automatic than effort-demanding tasks. Several investigators (Cohen, Weingartner, Smallberg, Pickar, & Murphy, 1982; Roy-Byrne, Weingartner, Bierer, Thomson, & Prost, 1986; Calev, Nigal, & Chazan, 1989), using tasks that differed in the amount of required cognitive cognitive effort, found that depressives were more disturbed in more effortful tasks than in less effortful ones.

The second main interpretation holds the view that the effects of depression on memory performance may be mediated, at least partially, by response style (Whitehead, 1973; Larner, 1977; Miller & Lewis, 1977). In such an interpretation, depressives are considered to have an overly cautious response strategy, that leads them to tend not to respond even if they have the correct answer at their disposal in memory.

The third stresses the role of mood state on memory functioning. Mood state would affect memory performances by activating a negative self-schema considered responsible for selective encoding and retrieval of information congruent with the depressed state and by furnishing contextual cues that trigger mood-state-congruent associations in the long-term memory (see for a review, Johnson & Magaro, 1987).

The fourth, recently put forward by Watts, MacLeod, and Morris (1988) explores analytically the role of different kinds of concentration lapses in performance of depressed patients and suggests that different cognitive processes may be altered in function of the type of concentration deficits (mind-wandering or blanking).

Finally, it must be underlined that these hypotheses are not mutually exclusive!

Initiation and planning of action are they operationalizable?

Our third main concern here is the positive contribution of the authors: The alternative way they propose looking at cognitive disorders in psychopathology. We agree with WHB that analysis of disorders (or of exceptional talented subjects) can reveal fundamental aspects of cognition that experimental psychologists with normal subjects may have neglected. Nevertheless, we are less convinced that their two proposals, a failure to initiate action in time and a disorder of action planning, represent sufficiently clear hypotheses capable of framing future research in psychopathology.

According to WHB, the single or central deficit in depression could be "an alteration in the time needed to initiate actions stimulated in normal fashion" and "this disorder does not affect the quality of performance, but rather execution rapidity, it is the slowness with which responses are activated that indirectly alters performance". However, there are ambiguities in WHB's position: in some passages this central disorder is not conceived as altering all information processes, since, when activated, a response is produced normally ("the timing of the motor activity itself is not modified"), whereas in another passage this global disorder is assumed to "affect all the stages of information processing". Either one assumes that the disorder is general and could be identified with a general decline in the information-processing rate (such an hypothesis has been for example unfruitfully put forward as a cause of cognitive decline in aging, see Rabbitt, 1988, for a critical discussion of such an hypothesis) or one supposes that what WHB have in mind is a selective slowdown of the processes related to decision making. If the second hypothesis is correct, the problem remains entirely open: Why does this component (decision making) slow down in the depressive state? If, in aging, a general slowing down of the information-processing rate has been hypothetically linked to a general decline of CNS efficiency, what could be the cause of a selective modification of information processing rate at the sole level of decision making or response initiation in depression? In our opinion when WHB confront this question, they may well be obliged to return to the hypotheses they have prematurely eliminated.

Please add Neuropsychology!

The difficulties that WHB encountered in interpreting the cognitive disorders of depressive and schizophrenic states in line with ideas of cognitive psychology, seem to us very similar to those encountered by neuropsychologists confronted with the interpretation of very similar disorders (at least on a phenomenological level).

We would thus suggest that, in addition to the relations that have been established between cognitive psychology and psychiatry, it could be promising to introduce neuropsychology as a new partner in the interaction. Indeed, progress in neuropsychology generally has resulted from the theoretically motivated comparison of single cases presenting similarities in their observable deficits. It is by the comparison of well-selected cases of alexias and agraphias that neuropsychologists have tried to penetrate the organization of the different subcomponents of the complex cognitive architectures that permit reading and writing. We think one can enlarge this approach to psychopathology. For example, WHB argue that a disorder of planning could be the core deficit in schizophrenic states, whereas in neuropsychology, on a phenomenological level at least, very similar planning deficits have been described in case of frontal lesions (Luria, 1969). These 'frontal' deficits have been interpreted by Luria in terms very similar to those used by WHB for schizophrenic states. Luria's proposals were also very difficult to operationalize but recently such planning defects have been the focus of theoretical elaboration and empirical verification by Shallice (1982). There is insufficient space here to discuss Shallice's theoretical propositions, but some of the distinctions he proposes in order to define more precisely the nature of the component that intervenes in action regulation and planning could be of interest to psychopathologists. In the same way, even if the slowdown of action initiation is, in its present formulation, difficult to integrate in many cognitive models, some neurological pathology could induce slowdown of action, as occurs in cases of traumatic injury. Not all traumatic patients are depressed, but many do present clinical signs similar to those advanced by WHB in depressive states. There have been many confrontations between psychopathology and neuropsychology in the past but often with the idea of discriminating organic from functional disorders, or more recently, with the aim of establishing neuropsychological profiles of psychopathological conditions. The objective we propose here is somewhat different and consists of comparing with precise information processing models single cases of psychiatric and brain-damaged subjects with the idea that these populations could offer us the opportunity to fractionate the cognitive architecture in different ways. On the both sides of mental pathology, collaboration could be fruitful. We hope this suggestion is a positive contribution to the interesting questions raised by WHB's article.

REFERENCES

Calev, A., Nigal, D., & Chazan, S. (1989). Retrieval from semantic memory using meaningful and meaningless constructs by depressed, stable bipolar and manic patients. *British Journal of Clinical Psychology, 28*, 67-73.

Caramazza, A. (1986). On drawing inferences about the structure of normal cognitive systems from the analysis of patterns of impaired performances: The case for single patient-studies. *Brain and Cognition, 5,* 41-66.

Cohen, R.M., Weingartner, H., Smallberg, S.A., Pickar, D., & Murphy, D.L. (1982). Effort and cognition in depression. *Archives of General Psychiatry, 39,* 593-597.

Colby, C.A., & Gotlib, I.H. (1988). Memory deficits in depression. *Cognitive Therapy and Research, 12* (6), 611-627.

Johnson, M.H., & Magaro, P.A. (1987). Effects of mood and severity on memory processes in depression and mania. *Psychological Bulletin, 101* (1), 28-40.

Larner, S. (1977). Encoding in senile and elderly depressives: A preliminary study. *British Journal of Social and Clinical Psychology, 16,* 379-390.

Luria, A.R. (1969). Frontal lobe syndromes. In P.J. Vinken & G.W. Bruyn (Eds.), *Handbook of clinical neurology, Vol. 2.* Amsterdam: North Holland.

Marin, O.S.M., Saffran, E.M., & Schwartz, D.F. (1976). Dissociations of language in aphasia: Implications for normal functions. *Annals of the New York Academy of Sciences, 280,* 868-884.

Marshall, J.C., & Newcombe, F. (1973). Patterns of paralexia: A psycholinguistic approach. *Journal of Psycholinguistic Research, 2,* 175-199.

Marshall, J.C. (1986). The description and interpretation of aphasic language disorder. *Neuropsychologia, 24,* 5-24.

Miller, E., & Lewis, P. (1977). Recognition memory in elderly patients with depression and dementia: a signal detection analysis. *Journal of Abnormal Psychology, 86,* 84-86.

Rabbitt, P. (1988). Does fast last? Is speed a basic factor determining individual differences in memory. In M.M. Gruneberg, P.E. Morris, & R.N. Sykes (Eds.), *Practical aspects of memory: Current research and issues, Vol. 2.* Chichester: John Wiley & Sons.

Roy-Byrne, P., Weingartner, H., Bierer, L.M., Thomson, K., & Prost, R.M. (1986). Effortful and automatic cognitive processes in depression. *Archives of General Psychiatry, 43,* 265-267.

Shallice, T. (1979). Case-study approach in neuropsychological research. *Journal of Clinical Neuropsychology, 1,* 183-211.

Shallice, T. (1982). Specific impairments of planning. *Philosophical Transactions of the Royal Society of London, B298,* 199-209.

Van der Linden, M. (1989). *Les troubles de la mémoire.* Bruxelles: Mardaga.

Watts, F.N., MacLeod, A.K., & Morris, L. (1988). Associations between phenomenal and objective aspects of concentration problems in depressed patients. *British Journal of Psychology, 79,* 241-250.

Whitehead, A. (1973). Verbal learning and memory in elderly depressives. *British Journal of Psychiatry, 123,* 203-208.