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# Memory, emotion and brain injury

*Some lessons from classical “organic” psychiatry*

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## Revised abstract

Brain injuries and brain illnesses provide a very important empirical foundation for the construction of neural network models of brain functioning. If one wants to base such models on neuropsychological or neuropsychiatric data, it is of course essential that the clinical syndromes are described properly. A truly astonishing wealth of relevant observations has accumulated during the last decades. However, it can be argued that the basic understanding of these syndromes by the present generation of psychiatrists and neuropsychologists still suffers from the radical break with traditional European psychiatry which occurred when the new American diagnostic and classificatory system for psychiatry (DSM-III, DSM-IV) was launched and accepted as the new basis for psychiatric thinking. Especially when it comes to modelling brain systems of memory and emotion, the neural network research community should benefit from a review of some facts which were lost in that transition. In the present paper, which is based on a comprehensive update of the classical European diagnostic systems that I participated in some years ago, I focus on three such less well-known facts pertaining to memory and emotion:

- ✚ The classical so-called “frontal lobe syndrome” is not seldom caused by extra-frontal pathology. This fact, which has been known since the 1920’s, clearly motivates a conceptual change. ***Emotional-Motivational Blunting Disorder*** (or ***EMD*** for short) is a construct designed to fulfil this purpose. The DSM alternatives are deficient in that they do not respect next point.
- ✚ It is extremely important to distinguish the emotional *flattening* seen in EMD from the emotional *lability* which is a common effect of many different brain pathologies. Many taxonomies create confusion on this point. Emotional lability is a component of what we call ***Asthen-Emotional Disorder***, or ***AED*** for short. Mental fatiguability and memory disturbances (mainly due to concentration difficulties) are other core symptoms of AED.
- ✚ The use of the seemingly general term “Amnes(t)ic Syndrome” to designate an – albeit very important – subgroup of memory deficits is misleading, considering that AED is the most common cause of memory disturbances in organic psychiatry. It is suggested that one instead use ***Korsakoff’s Amnestic Disorder***, or ***KAD*** for short.

## Background

★ In the early 1970's, the author entered an interdisciplinary cooperation with a leading European specialist in organic psychiatry, Göran Lindqvist. A specialist in both internal medicine and psychiatry, working first in departments of geriatrics, internal medicine and endocrinology, and then for two decades as a consultant psychiatrist in a neurosurgical university clinic, GL amassed an almost unique experience in the organic psychiatric field. Among other things, he was the first to report the occurrence of the Korsakoff's amnestic syndrome after rupture of an aneurysm on the anterior communicating artery.<sup>1</sup>

★ The present author's had had some psychiatric experience already in the 1970's, but his main contribution to the cooperation was his expertise in philosophical issues concerning classification, scientific explanation and the body-mind problem. Together, GL and HM published a comprehensive treatise on organic psychiatry in 1990 – regrettably, only in Swedish!<sup>2</sup> An English summary of it was published in 1993, but the international impact of the book has of course been negligible.<sup>3</sup>

★ The classificatory system in the book (below: the LM system) has its roots in classical European psychiatry: Eugen Bleuler, Manfred Bleuler, Karl Jaspers, Claus Conrad and many others. It is opposed to DSM-III/IV in that it is not a-theoretical.<sup>4</sup> I.e., the disorders are not defined strictly in terms of observable symptoms and signs. The LM system is however not etiologically based either. Indeed, one of the main points made in the book is that the same disorder can be caused by many different etiological agents – as a rule even by differently located brain injuries. Instead, the basic categories are thought of as *hypothetical psychopathogenetic processes*. These processes *typically* have certain observable manifestations, by means of which they can be empirically specified. However, through clinical and theoretical considerations the underlying processes can also be identified in non-typical cases where the usual empirical criteria are *not* fulfilled. Among these cases are, importantly, those where the disorder-process in question interacts with *other*, co-occurring disorders. Multiple diagnoses are thus possible, and indeed they should be the rule rather than the exception.

These principles will now be illustrated through three case histories (abbreviated and translated from Lindqvist & Malmgren 1990).

## Case 1

★ At the age of 50, this female patient noted a progressively raised mental fatiguability, irritability and emotional lability. External signs of acromegaly were noted at the age of 54, and with time she developed diabetes and heart insufficiency. At 64, the symptoms became much worse, and the patient complained of headache and severe fatigue. X-ray therapy directed towards the hypophysis had no certain effect and a **trans-sphenoidal hypophysectomy** was therefore performed when the patient was 66. Her endocrinological status after the operation was satisfactory and overall she improved somatically, but three years later she died from cerebrovascular causes.

★ At the preoperative psychiatric assessment, the patient was tired and emotionally very labile, and she complained about her severe forgetfulness, but the contact with the physician was very warm and natural. However, a few weeks after the operation – when the acute post-operative lowering of wakefulness had regressed – it was obvious that her personality had changed. She showed very little interest in what happened around her. She answered questions very briefly and it was impossible to get a good personal contact with her. Her mood was a shallow euphoria which however turned into irritation when people asked her difficult questions. – A couple of months later, there was some improvement, but she was still not interested in upholding a good emotional contact. She also seemed to have completely lost her sense of hygiene. – Later on, her condition improved gradually, and three years after the operation all the mentioned symptoms had disappeared. This was testified by the patient's close relatives and was confirmed by the clinical examination.

★ Before the introduction of DSM-III, the post-operative diagnosis for this patient's condition would have been *frontal lobe syndrome*. However, it is quite improbable that her operation should have done any damage to frontal structures. Instead, the case illustrates the fact that the so-called "frontal lobe syndrome" not seldom has an extra-frontal aetiology. In the LM system, the etiologically neutral name ***Emotional-Motivational Blunting Disorder***, or ***EM disorder*** for short, is therefore used.<sup>5</sup>

What about this patient's condition *before* the operation? Let us have a look at another case to illustrate this.

## Case 2

★ At the age of 37, this male patient started feeling more tired after work than usual. After nine months this fatiguability had become worse, and the patient also experienced concentration difficulties, forgetfulness and some emotional lability. Three months later a **meningeoma at the left frontal convexity** was diagnosed. At the preoperative psychiatric assessment, the patient seemed to be intellectually intact. He showed a natural spontaneity and initiative and his judgment was not impaired, but it was obvious that he easily got tired. This was especially the case when the task demanded continuous attention. He also became uncertain when put to memory tasks. The results on tests for concentration and memory were very weak, but there was no tendency to confabulation on the memory tests. – After the operation, the patient’s condition improved rapidly. He got back into work already after a couple of months, and then reported that his mental condition was the same as before the illness.

★ One interesting thing with this case is that although it was obviously caused by a frontal tumour, its symptomatology has *nothing* in common with the classical frontal lobe syndrome. There is no loss of initiative, no shallow euphoria, no disinhibition. The patient’s judgment and his ability for emotional contact are intact. True, there is an emotional *lability*, but this must not be confused with the emotional *shallowness* of the EM disorder. So how should we classify his disorder? In DSM-IV, the label *Cognitive Disorder Not Otherwise Specified* might be used for this patient’s condition. However, his emotional lability is not included in that concept so one might instead consider *Personality Change Due to Brain Tumor, Labile Type*. But that would not cover his cognitive difficulties.

★ In the LM system, there is a concept that includes both the cognitive and the emotional symptoms: ***Astheno-Emotional Disorder***, or ***AE disorder*** (or ***AED***) for short. Looking back to Case 1, it should be obvious that the *pre-operative* psychiatric condition of that patient was very like that of the present one, only that the emotional lability was more marked in her case. In short, she also had an AED. Indeed, AED is seen with a great multitude of etiological factors like head trauma, tumours of *any* location, endocrinological disturbances, infections, and ischemic and degenerative brain disease of old age. In the last-mentioned context, AED is better known as “Mild Cognitive Impairment”, MCI.<sup>6</sup> But why use a specific term for each clinical context, when there is one which covers them all?<sup>7</sup>

### Case 3

★ This man fell ill at the age of 67 with an attack of temporary loss of consciousness. One week later a similar incident occurred again and now a left hemiplegia remained. A subarachnoidal bleeding was diagnosed, and the patient was operated with **ligation of a ruptured aneurysm on the anterior communicating artery**. This was before the microneurosurgical era and the operation was technically difficult, but there were no somatic postoperative complications.

★ At the psychiatric assessment before the operation the patient was very tired and could not uphold his interest for the questions asked. His judgment and his ability for abstract thought seemed essentially intact, however. His results on memory tests were weak but he did not confabulate and he was fully oriented.

★ One day after the operation there were several marked changes in his mental condition. He was more alert and gave quick and formally adequate responses to questions. However, he was deeply disoriented both spatially and temporally, and did for example not know how old he was. He could not memorize what he was told, but gave some adequate information about his earlier life. – Some tens days later he was moved to another room and then became irritable and more difficult to handle. At the same time he seemed remarkably indifferent and never took an initiative towards talking. – Two weeks after the operation, he could adequately report about the events before the operation, with the exception of the days immediately preceding it. He was still disoriented and did not remember a single one of five presented objects after three minutes. He confabulated readily. This state prevailed for the remaining three weeks at the hospital.

★ After that there was a gradual recovery, but his emotional indifference and lack of interest in his close relatives were a concern for some time. Two and a half months after the operation, his daughter thought that the father was the same as before except perhaps for some remaining indifference. At the psychiatric assessment he was quite oriented and his judgment seemed to be normal. Although the results on some memory tests were as bad as before the operation, other were better and there was no confabulation.

## Amnesia is not one disorder

★ *Amnesic* (or *Amnestic*) *Disorder* (or *Syndrome*) is a common designation for this patient's condition after the operation. The first point to be made from a conceptual point of view is that these names invite a confusion between the kind of memory disturbance that prevailed *after* the operation and the memory problems that the patient had *immediately before*. With a high probability, the latter problems were at least partly due to a concurrent astheno-emotional (AE) disorder, i.e. they were of the same nature as the memory difficulties that we saw in Case 1 and Case 2. Since memory difficulties due to AED are more common in clinical practice than such due to the so-called Amnestic Syndrome, it is better to have a more specific name for the latter. We have therefore suggested the use of ***Korsakoff's Amnestic Disorder***, or ***KAD*** for short. Korsakoff described *all* the essential features of the syndrome in the 1880's, including the remarkable fact that implicit learning can be preserved.<sup>8</sup> Historically, the term "Korsakoff's syndrome" has been associated with a specific aetiology of the syndrome (alcoholism), and sometimes with symptoms other than the amnestic ones. However, in the absence of clear evidence to the contrary one may presume that the *amnesia* is essentially the same in alcoholic cases with dysfunction of the mammillary bodies or the medial thalamus as in, for example, bilateral hippocampal damage. (How else could Korsakoff's descriptions fit the famous case of HM so well?) That different locations may produce *some* clinical differences is obvious, but this does not entail that we should use different diagnoses.

★ The second point to be made from Case 3 is that it reminds us that Korsakoff's amnestic disorder is not seldom transient. The paradigm example of this is of course the so-called "Transient Global Amnesia" (TGA), which has all the characteristics of KAD.<sup>9</sup> We also believe that the common "memory gap" that often remains after a head trauma is a manifestation of an (otherwise) rapidly transient KAD.

★ The third point is the co-existence in this case of *three* organic mental syndromes: Korsakoff's Amnestic Disorder, Astheno-Emotional Disorder and Emotional-Motivational Blunting Disorder. During the acute post-operative phase, only the KAD and the EMD were visible. However, the memory deficits early and late in the course were probably mainly due to an AED. It may be presumed that the latter disorder was present even when the KAD and the EMD dominated the clinical picture.

## Dementia and the Dysexecutive Syndrome

★ *Dementia* is an essentially socio-functional concept that comprises many different clinical pictures and many different somatic aetiologies. We have found it fruitful to look at the symptomatology of the dementias in terms of the three “big” disorders AED, KAD and EMD. Nearly all dementia cases can be analyzed as containing more or less of each of these three components. The composition often varies over time and at the beginning only one of the disorders may be manifest. A dementia from cerebrovascular causes usually starts with AED (“MCI”) symptoms and is long dominated by more and more severe symptoms of this kind. Alzheimer’s disease is more prone to begin with discrete symptoms of EMD or even KAD, and even when AED are the first symptoms the other two components usually soon make their appearance. This is of course not surprising in view of the common location of the Alzheimer disease process to temporo-frontal areas. It goes without saying that beside manifestations of the three mentioned disorders, most dementia cases also show diverse focal neurological symptoms and signs such as aphasia.

★ The *Dysexecutive Syndrome* is a controversial construct which is defined differently by different authors. It is however thought by all of its proponents to comprise certain disturbances of higher cognitive (“executive”) functions and, importantly, to be “frontal” in nature.<sup>10</sup> Some of the definitions of the concept indeed reminds one of the classical “frontal lobe syndrome”, but other definitions hardly mention emotion and motivation. In our view, the concept is the result of not realizing that frontal damage can give rise to two distinct organic mental disorders: astheno-emotional disorder (AED) and emotional-motivational blunting disorder (EMD). Of these, AED occurs in a lot of other locations of the brain pathology, and even with frontal damage it is more common than EMD. EMD, in turn, does not occur only with frontal lesions but also as a consequence of pathologies of extra-frontal, limbic structures.<sup>11</sup>

★ A crucial issue in the differentiation of AED and EMD is the distinction between *emotional lability* and *emotional (and motivational) shallowness*. EMD leads to a change in the patient’s personality because her basic emotional attitude towards others and her ambitions about her own future are affected. In contrast, *a patient with AED (only) is the same person as before*, intellectually as well as motivationally and emotionally, but more prone to mental fatigue and emotional over-reactions.



## Neural networks for organic psychiatry?

★ Much effort is now concentrated on analyzing internal and external hippocampal circuits in order to understand the amnesic syndrome on a neural network level. There is a popular view to the effect that amnesia = hippocampal damage, but most researchers of today surely know that non-hippocampal lesions, for example in the mammillary bodies or medial thalamus, may cause a full-blown Korsakoff's amnesic disorder (KAD). Not so many are aware that most organic memory disturbances are *not* due to KAD. Both facts must be taken account of in the neural network theory.

★ Contemporary speculations about the neural networks underlying emotion often focus on conditioned fear and its supposed anatomical locus in the amygdalae. However, another equally urgent task is to understand the brain's innate motivational and emotional circuits on a detailed network level. The clinical evidence – especially, of course the evidence pertaining to the emotional-motivational blunting (EM) disorder – shows that these circuits include a much wider area, indeed the whole of the (fronto-)limbic system. The scientific community's interest in this system has been rather low during the last decades. This is probably partly due to the abovementioned misunderstandings about “frontal” symptoms, and partly to the way emotional disturbances are categorized in DSM-III/IV.

★ Astheno-emotional (AE) disorder poses a great challenge for neural network theorists. The way AED manifests itself is a paradigm case of graceful degradation: top intellectual and emotional capacity are kept very long, but the *efficiency* of cognitive and emotional processing is gradually diminished when the disorder worsens. The fact that the disorder can be caused by lesions at almost any location is intriguing. The immediate cause of the symptoms *could* be a structural or functional disturbance of a network that is physically distributed over almost the whole brain. A more probable explanation, though, is that the underlying network itself is more strictly localized but that its proper functioning depends on its receiving *normal input* from a host of other brain areas. Such an hypothesis becomes even more probable if one considers that AED affects high-level, non-domain-specific cognitive functions such as the capacity to uphold continuous attention on any current topic. To serve such capacities, the network must of course receive convergent input from many domains. Being built to coordinate such input, it is no wonder if its performance becomes degraded when part of the input falls outside the normal range.

## Notes and references

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- <sup>1</sup> Lindqvist, G. & Norlén, G. (1966). "Korsakoff's syndrome after operation on ruptured aneurysm of the anterior communicating artery." *Acta Psychiatrica Scandinavica* 42, 24-34.
- <sup>2</sup> Lindqvist, G. & Malmgren, H. (1990). *Organisk Psykiatri. (Organic Psychiatry)*. In Swedish) Stockholm: Almqvist & Wiksell.
- <sup>3</sup> Lindqvist, G. & Malmgren, H. (1993). *Classification and Diagnosis in Organic Psychiatry. Acta Psychiatrica Scandinavica* 88, Suppl. 373. Copenhagen: Munksgaard.
- <sup>4</sup> American Psychiatric Association (1980). *Diagnostic and Statistical Manual of Mental Disorders, 3rd Edn. (DSM-III)*. Washington DC: American Psychiatric Association. – American Psychiatric Association (2000). *Diagnostic and Statistical Manual of Mental Disorders, 4th Edn. Text Revision (DSM-IV-TR)*. Washington DC: American Psychiatric Association.
- <sup>5</sup> There is not room to discuss alternative concepts in detail here but the DSM-III term "Organic Personality Syndrome" should be mentioned. Regrettably, the definition of this concept includes *emotional lability* which means that many patients with AE disorder (see page 4) must have been given the diagnosis Organic Personality Syndrome (along with patients with EMD).
- <sup>6</sup> For a review of this concept see for example Mariani, E., Monasterob, R. & Mecocci, P. (2007). "Mild Cognitive Impairment: A Systematic Review." *Journal of Alzheimer's Disease* 12, 23–35.
- <sup>7</sup> For details about the AED concept, including the relation between it and some past and recent constructs, see also Malmgren, H. (1998). "Without a proper definition, you do not see the phenomenon. The history of a missing diagnosis." *Philosophical Communications, Green Series* 60, 1998. Gothenburg: University. Internet: <http://www.phil.gu.se/posters/ae.html>
- <sup>8</sup> Korsakoff tried to treat some of his patients with a kind of electrotherapy and noted that one of his amnesic patients always went into "a bad mood" when he saw the electrical apparatus – in spite of not remembering having seen it before. He also noted that several patients developed emotional reactions to the people at the ward although they never learned to recognize them. Korsakoff, S. (1996). "Medico-Psychological Study of a Memory Disorder." *Consciousness and Cognition*, 5, 2-22. This paper is a translation of Korsakoff, S. (1889). "Étude médico-psychologique sur une forme de maladies de la mémoire." *Revue Philosophique* 28, 501-30. Compare also the following comment by one of the translators: "Neuropsychological findings have revolutionized the study of memory in the last decade and a half. Virtually all of the findings considered important about anterograde amnesia were reported by Korsakoff, and some that he reported still have not had the impact they should on current thinking." Banks, W. (1996). "Korsakoff and amnesia." *Consciousness and Cognition*, 5, 22-26.
- <sup>9</sup> For a recent illustrative case report see Saito, K., Kimura, K., Minematsu, K., Shiraishi, A. & Nakajima, M. (2003). "Transient global amnesia associated with an acute infarction in the retrosplenium of the corpus callosum." *Journal of the Neurological Sciences* 210, 95–97
- <sup>10</sup> Stuss, D. & Alexander, M. (2007), "Is there a dysexecutive syndrome?". *Philosophical Transactions of the Royal Society of London, B series*, 362, 901–915. The authors of this paper betray a generally high methodological awareness but seem not to have considered that evidence from exclusively *non-frontal* lesions is needed if one wants to tie a syndrome aetiologically to (subregions of) the frontal lobes. Most of the results that they report can be given an alternative theoretical account in terms of mixtures of EM and AE disorders.
- <sup>11</sup> This may be the reason why temporal lobectomy for otherwise intractable epilepsy sometimes improves "executive functioning". Cf. Martin, R., Sawrie, S., Edwards, R., Roth, D., Faught, E., Kuzniecky, R., Morawetz, R. & Gilliam, F. (2000). "Investigation of Executive Function Change Following Anterior Temporal Lobectomy: Selective Normalization of Verbal Fluency". *Neuropsychology* 14, 501-508. (Thanks to Kristina Malmgren for pointing out this.)