

THE LANGUAGE OF PSYCHIATRY: A TIME FOR A CHANGE?*

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INTRODUCTION

The topic today resumes a great deal of my work. I shall be proposing that, for reasons that soon will become apparent, the language of description in psychiatry is in need of updating and modification. One important reason for this change is that such language is no longer meeting epistemological needs it was created to deal with in the first place.

NEUROBIOLOGICAL RESEARCH AND THE PSYCHOPATHOLOGICAL LANGUAGE

The hypotheses I should like to propose today are as follows: firstly, in each historical period there should be an optimal match between the level of description in psychopathology and the resolution power of neurobiological research. A version of the latter started during the 19th century, and to respond to its needs alienists were asked to construct a language of description. In the event, this language perfectly matched the level of description then utilized in microscopic work and in gross neuroanatomy. A hundred years later, we find that neurobiology is developing at great speed and yet the language of description of abnormal behaviour remains the same as it was a hundred years ago. I submit that this mismatch is making science suffer; and that this means that clinical psychiatry and indeed patient care may suffer.

Secondly, current level of description in psychopathology tends to be dichotomic or, at most, scalar, i.e. a hallucination is present or absent or anxiety is mild, moderate or severe. Once again, that level of description belongs to a period when the power of resolution in brain research was limited and subject to marked variance and hence had low reliability. A hundred years later we find that molecular biology includes quantitative and dimensional analysis that are well beyond the quantitative range of conventional psychopathological description. Likewise, during the last hundred years there has been a shift in models of brain localization. 'Localization', last century, was

defined as the state in which a particular round function related to a circumscribed region of the brain in a static manner. We have now developed more dynamic views, which go beyond geometrical, or three-dimensional localization to include 'chronogenic' or 'time-related' localization models where circuits and functional systems unfold both in space and in time. Old behavioural descriptions, which did not include accounts of the natural history of signs and symptoms, can no longer match these new views of brain representation.

DIAGNOSTIC MODELS AND SYMPTOMS

Diagnostic methods in psychiatry have also changed. Once again, in the 19th century alienists conceived of insanity as an 'all-or-none' state i.e. either the patient had the illness or not. In the past twenty years, such categorical notion has been replaced by 'continuum' concepts such as 'caseness', embracing probabilistic statements about the existence of disease which are controlled by context and objective. The question 'is this a case of dementia' is qualified by the question 'a case for what?'. Is it to be used in epidemiological or neuro-biological research or are we planning treatment? The dimensional and probabilistic basis of 'caseness' has caused serious disturbance in traditional psychopathology.

Complementing the caseness concept, new quantificatory approaches have been imported into clinical practice such as 'signal detection theory', according to which it is possible to explain clinical diagnosis as a subtle interaction between observation and decision making. That is, what the patient presents to the clinician (behavioural forms), is not sufficient for deciding on whether such sign or symptom is the case. Decisional mechanisms enter into operation which have to do more with previous knowledge, expectation and context than with the actual sense-data provided by observation. The application of these powerful mathematical theory requires that signs and symptoms in psychiatry be described in a more sophisticated way than they are now.

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CO-MORBIDITY AND SYMPTOMS

Psychiatric co-morbidity has become fashionable. In the USA, for example, so-called 'co-morbidity clinics' are beginning to appear. Basically, these are places specializing in cases who seem to have two 'DSM-III R diseases' at the same time; e.g. affective disorder or schizophrenia and drug addiction. If the notion of comorbidity happens to be correct, then traditional description of signs and symptoms will no longer hold as cases will soon appear where two diseases may be borrowing symptoms from one another. Subtyping of symptoms will be required to cope with this; and subtyping will demand a better knowledge of the internal structure of symptoms. This cannot be implemented in current psychopathology as the meta-linguistic tools required for such analysis do not yet exist.

TEMPORAL DIMENSION OF PSYCHO-PATHOLOGY

There is also a need for more work in relation to a putative interaction between 'time' as a variable and the evolution of disease. The issue here can be exemplified by describing, say, an investigation into the aetiology of depression in multiple sclerosis. The conventional paradigm is collect a cross-sectional patient sample, assess it by means of depression scales and neuroimaging, and establish statistical correlations, say, between number of plaques and depression global scores. Then, it might be found that such correlation is higher with plaque number in the frontal than in the temporal lobe; and it might be concluded that frontal lobe involvement is the basis for depression in MS. The fallacy here is that because the study does not take into account 'time' as a relevant variable, no effort is made to 'date' or 'time-tag' plaques. Without this crucial correcting variable, the correlation is meaningless as it is between two sets of variables operating in different temporal scales or realms: depression scores originated from one point in time, plaques are telescoped relics from a succession of events. This methodological flaw that Dr Quemada and I described in 1990 in the *British Journal of Psychiatry* has led to the suggestion that each neurological disease will require a different model for psychopathological research according to whether it evolves in discrete episodes or linear fashion, whether it is progressive or not, etc. Once again, to deal with these methodological difficulties, the description of signs and symptoms needs to be modified.

CLOSURE OF CURRENT SET OF SYMPTOMS

Finally, a sheer insufficiency in the current set of psychiatric symptoms is becoming obvious. Have you ever counted how many symptoms you actually use in your everyday psychiatric practice? We may have 70 or 80 if the small print of all textbooks and historical papers is studied, but in practice no more than 15-20 are used. How come? Are these sufficient to cope with all the mental disorders listed in ICD-10? Why is it that this 19th century lists remain the same, and few if any new symptoms have been added?

The language of signs and symptoms was invented during the 19th century by clinicians just like ourselves, who were good enough at observing abnormal behaviours in their own patients. There is some evidence that both the clinical profile of patients, and the psychological theories governing their description, have changed markedly during the last century. Why is it that the list of symptoms has remained the same? I submit here that there has been a tendency to premature 'closure', that is, the mandarins of our discipline have decided that descriptions are now complete and that all research efforts must be directed to nosological studies. Such closure has caused the feeling that psycho-pathological research is old-fashioned. But, is it the case that we already have all the symptoms that we will ever get in mental illness? Where is the evidence for this? You may say that evidence is provided by the fact that the daily examination of patients does not seem to yield any new symptoms.

This surmise is incorrect for we tend to use special 'diagnostic grids' to examine patients and many odd behaviours for which we do not have a name we either force into a known symptom or leave out altogether. This is because the 'closure effect' determines what has meaning and what has not in diagnostic terms. For example, very few of you will measure the size of the big toes in schizophrenia because, you will say, there is no evidence that it correlates with presence or severity of schizophrenia. First of all, I know of no paper which has shown such correlation to be bogus; secondly, it could well be that the gene that controls the size of the big toe is linked to that of schizophrenia and hence it could be considered as a good marker. What I am trying to say is that, perhaps, it is time that we kept an open mind as to the total list of psychiatric symptoms and signs; that is, that we felt able to add to the list. I hasten to add, however, that I am not advocating for a minute to abandon what we have achieved. Glossaries such as DSM-III, PSE, AMDP are essential for communication and to sustain high levels of reliability. What we cannot assume, however, is that the validity of our descriptions has also improved. I believe that we have now done enough for reliability and that we ought to start working towards validity.

One solution to the 'closure' problem, which might not upset glossary makers, is to allow a two-tier system in psychopathology; i.e. parallel to the conventional high reliability descriptions, a freer system of description, often of low reliability but potentially high validity, should be created so that ordinary clinicians might exercise, once again, the lost right of describing new symptoms. Of the many glossaries I know, only AMDP, the German-French glossary is the only one that offers spaces for new symptoms.

PROPOSED CHANGES

MULTI-DIMENSIONAL REPRESENTATION OF SYMPTOMS

Today, I should like to propose some changes in the structure and content of descriptive psychopathology. Firstly, and at least for research purposes, we should abandon the dichotomous, unidimensional view of symptoms and replace it by a multidimensional model according to which symptoms are envisaged as composites constituted by 'units of analysis' recognizable as components or dimensions. Often, the latter themselves may be micro-composites and require to be broken up into further components. The natural boundaries of these units of analysis can be recognized by the fact that their isolation does not lead to great loss of information. Symptoms, however, have structures, i.e. the units of analysis are coupled either horizontally or vertically in hierarchies, and can be mathematically modelled as 'vectors' or 'matrices'.

The second proposal is that, as mentioned above, everyday clinicians should be actively involved in the description of new symptoms. The third proposal is that more emphasis should be placed on 'signs' that are instrument-dependent, or what I have called elsewhere 'micro-symptoms' or 'molecular' symptoms. The fourth and final proposal concerns a model that explains how the biological signal is transformed in the depth of the patient's mind into a recognizable and reportable 'symptom'.

'NOISE' IN ACQUISITION OF SYMPTOMS

The medical approach we ought to have in psychiatry, and the belief that mental illness has brain representation, lead to the assumption that symptoms are transformed biological signals. This process of transformation starts when the subject experiences a subjective 'feeling' caused by the distressed brain site. This feeling may be distinct or fuzzy but the patient is expected to recognize it and name it on his own. At this juncture, it is important to remember that the patient is also dysfunctional in other ways, i.e. his consciousness, cognition or attention may be disturbed in gross or subtle ways. Nonetheless, he is supposed to be an objective 'reader' of his experience, to be able to transcribe into concepts or categories whatever is going on in his consciousness, and finally report it in speech. The transformation does not stop there. Once in the cognitive purview of the psychiatrist, the information is further processed according to new 'grids', and eventually announced as a 'hallucination', 'guilt' or whatever. Thus, symptoms can be conceived of as biological signals experienced as feelings (the raw material) which are, at least, processed twice: firstly, in the consciousness of a subject who is often ill and confused; and secondly in the consciousness of another subject who, hopefully, is normal but who may have his own cognitive agenda.

According to one's epistemological perspective, one can see the same process as one which is very unreliable and

hopelessly contaminated by noise or as a sturdy mechanism, originally set by evolution and gradually made stable by the cultural parameters in which it takes place. Whether one or the other interpretation applies, one must recognize that so far conventional 'symptoms' have served us very well, and are what psychiatric diseases nowadays are all about. They result from a cognitive negotiation between patient and doctor and hence, even unwittingly, include a host of relevant information.

The problem is at the level of neurobiological research. Symptoms, as we have seen, are bits of information (brain signals) enveloped in 'noise'. The central aim of symptom describing, however, is identifying the brain source of the signal, and descriptive psychopathology can be described as a set of descriptors to decode signal from noise. My feeling is that our decoding capabilities are not yet adequate. Signals are generated by diseased brain addresses which can be conceived of as neuroreceptor populations, circuits or systems. On occasions, the best way to get at the signals would be to map abnormal behaviour in terms of micro-symptoms. Unfortunately, the latter are still in their infancy. The crucial thing to remember, however, is that descriptive psychopathology is a cognitive instrument which has been designed to increase the gain of the signal and minimize the noise.

THE GRAMMAR OF PSYCHOPATHOLOGY

In order to improve the decoding capability of descriptive psychopathology we must have a clear idea of its 'grammar', and develop ways to streamline and improve it. Once again, the problem is more research-related than clinical as we all know that great clinicians can achieve major diagnostic feats with the old language as it is. They can sit in front of the patient for hours and obtain a detailed picture of what's going on 'inside' his head. Unfortunately, not all psychiatrists are like that. Some of us don't have that capacity and hence require further cognitive aids. We need to be taught how to do it and this means that the grammar of psychopathology needs to be made explicit.

We must ask how is it that great psychiatrist process information and eventually come out with a diagnostic conclusion which is right and useful to the patient? Is it intuition? I don't think so. I think these skills are learned in the same way as language skills are learned by the child. Implicit rules are passed on from generation to generation, all children will learn but some will be better than others. The difference is that the rules of descriptive psychopathology should be far simpler than those of language in general. The problem is that so far the great emphasis has been not on the grammar of description but on that of 'nosology' i.e. on diagnosing diseases. This has caused amongst many researchers the wrong belief that psychopathology is "transparent". If you read DSM III-R, you will find about 400 pages on diagnostic-criteria for diseases and very few on symptoms and signs. I believe, however, that

the recognition of symptoms is hard, and that psychopathology is incredibly opaque. Furthermore, I believe that the crucial moment of diagnosis is not really the putting of signs and symptoms together in some cluster or other but in the recognition of symptoms and signs. However old-fashioned this claim may sound to you today, I firmly believe that therein lies the future of research in psychiatry and that it is only a matter of time before American psychiatrists change their mind. Once they have done so, psychopathological research will become respectable and everyone will follow.

EVOLUTION OF THE LANGUAGE OF PSYCHOPATHOLOGY

In order to accept the view here expressed that psychopathology is in state of evolution and that we clinicians have the responsibility of contributing to this, we must first get rid of the idea that it is a cognitive system which is now complete and created for ever. There is no better way to do so than looking at its historical origins.

Descriptive psychopathology was totally constructed during the 19th century and it is not true, as it is often stated, that Karl Jaspers invented it during the early 20th century. There is no time today to rehearse for you the evidence for these claims but you can find it in various publications of mine. The crucial point to remember is that when one language is invented during a particular period, it automatically is made to enshrine contemporary principles and assumptions. Descriptive psychopathology is no exception to this. But this also means that those historical figures who were involved in the construction process were able to build into their contribution their own personal biases and conceptual imprints. For example, it is not difficult to recognize the contribution made to the concept of Hallucination by Esquirol, to delusion by Baillarger and Griesinger, to obsession by Morel, to cognitive impairment by Kraft-Ebbing or to confusion by Chaslin. Nor is it hard to see how the great debate between Kraepelin and Wernicke created a conceptual cleavage in the language of psychiatry that reverberates even in our own day. Thus, there are tensions in the psychopathological language, there are pressures pushing in various directions that make it a dynamic and changeable entity. In this sense, the life and work of individuals matters. For example, there is good historical evidence that, had Wernicke lived longer (he died in his 50s in a road accident) we would be living now not in the Kraepelinian but in a Wernickian world for he was far more imaginative and charismatic than Kraepelin. We would not live in a two-psychoses but in a many-psychoses world. As you know, it is only in the last 10 years that there has been a revival of the Wernicke-Kleist-Leonhard line. I suspect that in the next few years if a proper European psychiatry develops we might see once again the revival of the Wernickian views.

From a historical viewpoint, as I mentioned above, there was no language of psychopathology before 1800. It is very clear from reviewing casenotes, however, that by the end of the 19th Century everybody was using a psychopathological language. Where did this language come from? What happened was that by the beginning of the 19th century there was the convergence of three major forces: once was the availability of convenient psychological theories, the other the development of an anatomo-clinical model of disease, and the third the descriptive need of the medics just incorporated into the mental hospitals. This meant that the great monolithic concepts used until then to refer to mental illness were soon fragmented and re-analysed. Thus insanity, delirium, mania, and melancholia (whose meanings were then very different from what they are now) were teased out into the new symptoms. A psychological theory called association-ism assisted this process. It proposed that the mind was a collection of ideas glued together. This Newtonian view of the mind was extremely important as it suggested that the totalistic notion of insanity could be analyzed out into units or components. If you study casebooks in old British asylums between 1810 and 1850, you'll see how psychiatrists gradually abandoned the old monolithic notions and began to describe the new symptoms.

Indeed, the interesting situation developed that by 1850 there probably were more symptoms than we have nowadays. There were, therefore, symptoms which are no more such as 'aural haematoma', which consisted in a bluish swelling in the lobe of the ear shown by many patients. Indeed, during this period the scientific literature on this symptom is large and the predominant hypothesis was that it resulted from a susceptibility in the insane patient to develop blood clots which correlated with severity; i.e. the more severe the psychoses the more marked the discolouration. It was only after great deal of research and debate that it was concluded that it was a false symptom and that it was caused by the keepers or early nurses holding patients by their ears to lead them from one place to the other!! But the correlation still held, i.e. the more crazy you were the more likely it was that you will have an "aural haematoma".

Another example is that of verbigeration, a symptom described by Kahlbaum in 1874 in his little monograph on catatonia (where he reported 29 cases of which only very few we would nowadays call catatonia). It referred to a way of talking which was fast and incomprehensible. At the time much attention was given to verbigeration as a 'disorder of language' in psychiatry and was considered by many as the central feature of catatonia, in spite of the fact that no more than 8 cases out of the 29 in his series had it, and no more than 10 could be said to suffer from catatonia! What happened to verbigeration? I don't know, but the symptom has disappeared.

PHENOMENOLOGY AND PSYCHOPATHOLOGY

I am now going to say a few words about the extent to which the philosophical movement called 'phenomenology' has influenced psychopathology. Basically, the issue is as follows: we are told everyday that we have the great luck that Jaspers taught us that it was possible to produce 'neutral descriptions' in psychopathology, to generate descriptions of behaviour which are theory-free. There are a number of problems with this story. Apart from the issue that perhaps it is never possible to produce descriptions of behaviour which are theory-free, there is the question of whether phenomenology was the source of that approach; i.e. that Jaspers did in fact borrow such a belief from phenomenology. My own research (and that of others such as C. Walker in Leicester) has shown that there is no evidence that Husserl influenced Jaspers' thought. In fact, when Jaspers sent his material to Husserl, Husserl did not pay much attention to it nor believed that Jaspers was developing his own ideas.

Husserl was onto something else. Neither his phenomenology was relevant to psychopathology nor did phenomenology ever put forward the view that theory-free descriptions of the world, let alone behaviour, were possible. The word phenomenology is used in the UK in many ways and none is relevant to psychiatry, including the meaning that relates it to the description of signs and symptoms. The point is that saying that a description is phenomenological does not add anything to it except confusion.

Indeed, during the early 20th century the most important figures in psychopathology were Jules Seglas and Philip Chaslin. The former wrote in 1906 a superb account of 'semiology', i.e. the science of signs and symptoms in psychiatry. The latter, who was a great mathematician apart from being a great clinician (just like Max Hamilton was to be 40 years later) wrote in 1912 (before the first edition of Jaspers book) a magnificent book on all the symptoms of psychiatry where he put together all knowledge in this field. In 1914, he also wrote a classical paper entitled 'Is psychopathology a well-made language?' With great foresight he discussed therein issues pertaining to distortions in the description of symptoms and wondered

whether alienists often added more noise than the patients themselves. He also said that qualitative descriptions of symptoms may need to be improved upon by quantification. He was, of course, a great mathematician and he could see that symptoms were susceptible to dimensional analysis.

CONCLUSIONS

What I have said to you today can be considered as a footnote to Chaslin's great paper. The complete footnote should say that there is good reason to believe that the clinical sciences in psychiatry will only progress further if a very careful matching is made between the level of clinical description and the level of analysis in neurobiology, which is now moving fast. The language of description in current usage may be adequate in clinical practice but is of little use in neurobiological research. This is because it belongs to a period where neurobiology was constructed in terms of gross anatomy and limited microscopy. We must overhaul the language of psychopathology. This means to study its grammar and syntax, and to create a meta-language that can deal with the structure of symptoms. The latter will have to be seen as dimensional composites, probably organized in hierarchies, which can be modelled mathematically. This will allow the researcher to describe behaviour at a level of resolution that matches the requirements of current molecular biology and PET technology.

Why would we want to do this? Just as an intellectual or aesthetic trip? I suppose partly so because it is pleasant and fulfilling to do research. But that should not be the main reason. We tend to forget that the reason there are departments of psychiatry and there are psychiatrists and researchers is because there are patients. If patients disappeared, none of us will have a reason to exist qua professionals in mental health. Therefore, the reason for occasionally thinking in the rather abstract manner in which I have done in front of you today is not necessarily intellectual satisfaction but the need to improve upon what we are doing, because in doing so we shall be serving our patients better.

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