

Conceptual Issues

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Born as a by-product of the nineteenth-century movement to organize society on a scientific basis, psychiatry was charged with the construction and enactment of normative views of madness. (Literature on social and clinical history of psychiatry is large; for scholarly papers on both, see the journal *History of Psychiatry* (started 1990).) Under the protection of medicine and the economic practices following the Industrial Revolution, psychiatrists developed representations of mental disease together with the professional and institutional apparatus to enjoin them.¹ Since its construction, the modalities of psychiatry have been many. For example, there is the group called biological psychiatry,² organic psychiatry, neuropsychiatry and behavioural neurology, which seem to share the foundational claims (FCs) that: (1) mental disorder is a disorder of the brain; (2) reasons are not good enough as causes of mental disorder; and (3) biological psychiatry and its congeners have the patrimony of scientific truth. These four congeners are man-made and there is nothing in nature to suggest real differences between them; indeed, any differences that might be suggested are bound to be historical in origin. To avoid confusion, the term 'biological psychiatry' will be used in this chapter as a proxy for the other three congeners. Since the seventeenth century, versions of what might be called biological psychiatry have come in and out of fashion;³ the reasons for these cycles are unclear.⁴

FOUNDATIONAL CLAIMS AND THE 'TECHNOLOGY ALIBI'

FCs are unproven and unprovable propositions used to start off the narrative of science. Chosen by 'experts', FCs escape audit and

¹ A representation is an image, model, view, concept or other definitional form that helps someone to think of something. Control includes social practices, such as therapy, prophylaxis, support, management, rehabilitation, policing, punishment, incarceration and the death penalty. A social practice is a rule-governed form of social behaviour that runs on habit, is passed from generation to generation, and contributes to the stability of society; the stability of the concept has been questioned (Turner, 1994).

² At this stage, a clarification needs to be made. In this chapter, we distinguish clearly between (1) biological psychiatry as a methodology and research *doctrine* and (2) the *clinical* practice of those who call themselves 'biological psychiatrists'. This is because it is highly likely that they will, like non-biological psychiatrists, talk, understand, support, counsel, and resort to whatever psychological therapies are needed to help their patients. This chapter is entirely about the limitations affecting the *doctrine* and *methodology* of biological psychiatry. Indeed, given its narrowness, we believe that it would be very difficult, even for rigid biological psychiatrists, to guide their clinical practice by doctrine alone. On the other hand, it would be wrong to invoke the flexibility and variety of approaches that biological psychiatrists have to use in their clinical practice as *evidence* that the *doctrine* itself is flexible and comprehensive, for it is not. For earlier work from our group on the methodology of biological psychiatry, see Berrios, 1995; Berrios and Denning, 1990.

are unchallengeable. The view that mental disorders are 'caused' by changes in the physical conformation of the brain is a typical foundational claim; that 'reasons' cannot constitute efficient causes for the generation of mental disorder is another.

Since the nineteenth century, and every time it has failed to deliver, biological psychiatry has used the excuse that ongoing technologies are not sensitive enough to identify the organic causes of all mental disorders and reassured the expecting public that future techniques will do so: we call this the 'technology alibi'.

Foundational Claim 1: Mental Disorders as Brain Disorders

Meanings for this FC range from the broad assertion that all psychological activity must have brain representation to the narrow assertion that specific brain lesion *l* is a necessary and sufficient cause for presence of specific mental disorder *m* (more on this later). Similar claims were made during the middle of the seventeenth century, e.g. by Thomas Willis in 1685 (see Vinchon and Vie, 1928).⁵

The claims that the body (brain) houses the human soul (or mind) and that changes (lesions) in the conformation or constitution of the body (brain) cause mental disorder (i.e. FC 1) are historically independent, and their relationship is asymmetrical; indeed, the

³ Neither cross-culturally nor trans-historically can biological psychiatry be considered as a *unitary* activity: according to social and economic need, different types and practices predominate in different contexts. For example, biological psychiatry during the second half of the nineteenth century was characterized by very specific definitions of disease, internecine disputes concerning the professionalization of neurologists and alienists, an increasing use of the light microscope, and early studies of brain chemistry. Differences were, on occasions, exaggerated by international rivalries and jingoism; for example, this is the case during and after both the Franco-Prussian War, and the Great War, when even writers like Chaslin and Kraepelin made silly remarks about 'enemy' countries. (On the history of biological psychiatry, see Berrios and Marková, 2002a.)

⁴ An interesting issue here concerns the relationship between the historical versions of biological psychiatry. Are they manifestations of an ongoing idea existing *sub specie aeternitatis*? Is their similarity skin deep and not worth bothering about? When Thomas Willis stated that mental illness is a 'disorder of the brain', was he being a precursor of similar current claims?

⁵ These claims are intelligible to us only because they were made within the new epistemological frame (dualism) created by Descartes. Similar-sounding claims made in earlier periods (e.g. by Galen) are much harder to understand. In pre-Cartesian times, explaining the *existence* of a form of behaviour (e.g. madness) in terms of a *link* to the soma (whether heart, hypochondrium or brain) made little sense because there was no differentiation between anatomy and function in the way that we understand it today. In this sense, it is anachronistic to believe that the history of biological psychiatry starts with the Greeks. Indeed, current concepts, meanings and categories in psychiatry were constructed during the nineteenth century (see Berrios, 1996).

belief that brain disease causes mind disease is not logically dependent on the belief that the mind resides in the brain (because the former could be plausibly held without the second being true).⁶

The claim that the body (brain) must house the human soul (or mind) follows from the materialist assumption that at the time the world came into being, there was only matter (Lange, 1957). Thinkers of this persuasion need to explain, however, where ‘mind phenomena’ come from, particularly if a highly configured model of the latter is entertained. Materialist thinkers make use of two types of matter: *Plain* matter is constituted by homogeneous units of analysis (atoms, fields, strings) and cannot explain mind configurations; these then are assumed to come from outside (cultural context). *Baroque* matter may be constituted by atoms of different shapes and sizes moving at different rates, or may include dynamic and hidden qualities (Cappelletti, 1979). For example, Cabanis (1802) (although overtly a materialist) held a baroque view of matter and hence was able to account for complex aspects of the mind without resorting to any external agencies.

Foundational Claim 2: Causes and Reasons

Causes and reasons are forms of accounting for the existence and/or origin of objects, tokens, episodes, processes and behaviours. Although etymologically different, they have overlapping semantic fields (i.e. are used as synonyms) and similar epistemic power (capacity to explain actions). Causes are used in the natural sciences; reasons are used in the social sciences (which, in the past, included psychology). What about psychiatry?⁷

Causes

What we have said above suggests that to ask for the cause of a mental disorder is already to opt for a particular model of explanation (Berrios, 2000a), as equally cogent would be to ask for a reason, based on the view that people may do things, including ‘talking and behaving crazy’, for reasons better known to themselves. Reason in this sense needs not be construed in material or mechanical terms but may remain expressed in the language of folk psychology. Even more radically, it could be said that mental disorders are not *effects* at all, that they do not follow either causes or reasons, and that they are just ‘givens’, i.e. they have been there from the beginning of time. In a world keen on looking for the causes of all things, this latter view may sound strange, but it is not unintelligible and it simply reflects the ontology of preformationism, a view of biology predominant in the eighteenth century.

It could be argued that reasons are just a subtype of cause. This claim muddles the issue further. It is true that the Latin *causa* meant reason, motive, inducement, but it also meant occasion and opportunity. It translated the Greek *aitia* and *aition* (stems of the medical term aetiology), which meant origin and ground but also the occasion of something bad. In practical terms, cause has been used

⁶ For example, at the beginning of the nineteenth century there was a debate as to whether the mind or soul was located in the cerebrospinal fluid or the cortex (Hagner, 1992). Likewise, the view was popular in the same century that because the soul (mind) was a marker of the divine, it could become neither divided or diseased. Its putative location on the cortex also caused difficulties in regard to its functional (or phrenological) parcelling (e.g. Jackson; see p. 8). The issue of whether organs other than the brain might contribute to the formatting of the mind and its diseases was discussed up to the nineteenth century; prime candidates remained the heart (Willis, 1685), hypochondrium (Whytt, 1768) and stomach (Broussais, 1828).

⁷ Biological psychiatry has not yet taken on board the importance of the distinction between reasons and causes. This may be due to the fact that explaining human action or symptoms by means of reasons does not fit into the rather crude mechanistic and deterministic frame of neurobiology.

mostly as a *relational* concept, i.e. as one without which another thing (effect) cannot be. Based on his own model of the Universe, Aristotle (1991; p. 1600) defined it thus:

We call a cause (1) that from which (as immanent material) a thing comes into being, e.g. the bronze of the statue and the silver of the saucer, and the classes which include these. (2) The form or pattern, i.e. the formula of the essence, and the classes which include this (e.g. the ratio 2 : 1 and numbers in general are causes of the octave) and the parts of the formula. (3) That from which the change or the freedom from change first begins, e.g. the man who has deliberated is a cause, and the father a cause of the child, and in general the maker a cause of the thing made and the change-producing of the change. (4) The end, i.e. that for the sake of which a thing is, e.g. health is the cause of walking. For why does one walk? We say ‘in order that one may be healthy’, and in speaking thus we think we have given the cause. The same is true of all the means that intervene before the end, when something else has put the process in motion (as e.g. thinning or purging or drugs or instruments intervene before health is reached); for all these are for the sake of the end, though they differ from one another in that some are instruments and others are actions . . .

Of the four Aristotelian types of cause (material, formal, efficient and final), only the efficient one has survived in our day.⁸ In the first half of the seventeenth century, Galileo interpreted efficient as resulting from general physical laws. During the second half of that century, Newton redefined cause in terms of a physical metaphor (‘collision between two objects, such as billiard balls’) (Doney, 1968). This made the concept of cause quantifiable, predicting and reversible but excluded human agency and rendered it soulless.⁹ In spite of Hume’s opposition to the Newtonian model of cause, the nineteenth century accepted the Newtonian view and soon enough this was incorporated into medicine and alienism.

Since the time of Georget (1820), biological psychiatry has hung on to the thin hope that the demonstration of a brain lesion in a patient with mental illness may be sufficient to generate a cause–effect inference that can be applied to all. During the nineteenth century, the existence of such a link was liberally assumed; for example, post-mortem lesions were considered, *simpliciter*, to be the cause of a disease, irrespective of the nature and age of the lesion and of the period in the patient’s life when the disease occurred; that is, the need for contemporaneity was not acknowledged. In current biological psychiatry, this principle of guilty by association is still present. It is unclear what type of contemporaneity is required by the ascertainment of reasons (e.g. discursive accounts of behaviour) and of causes (e.g. statistical correlation) (for the latter, see p. 18).

Reasons

Although in common parlance it is accepted widely that reasons provide adequate accounts for a range of human behaviours, the semantic engine that makes these explanations possible is unclear. Cicero used *ratio* to translate the Greek for account,

⁸ From Greek times, the concept of cause has been used to refer to both aspects of human agency (as per Aristotle) and the belief that effects result from the action of universal laws (as per Plato) (Berrios, 2000a).

⁹ This came back to haunt the studies of man during the nineteenth and twentieth centuries when the human sciences tried to ape the natural sciences and abandoned hermeneutics. Without a machinery to deal with meaning, reasons, subjectivity, and the understanding of the individual, the human sciences (and psychiatry with them) became vacuous. The Freudian movement attempted unsuccessfully to provide nineteenth-century psychiatry with a semantic model.

calculations, etc., and soon enough, it also translated the Greek for cognition, thinking, discourse, understanding, etc. This combination of meanings has plagued the term ever since. In most European vernacular languages, reason refers to the *illative* rather than the creative or imaginative aspects of the intellect. Since ancient times, reasons have been used to justify actions (*post facto*) or explain their origin. To say that I did *x* because of *y* remains a good way of accounting for my behaviour as long as: (1) there is a cultural or semantic frame within which *y* mostly leads to *x*; and (2) there is no reason to believe that I am cheating.

To some, however, accounts of behaviour by means of reasons are considered as after the event and hence do not constitute proper causal accounts. Two factors (*inter alia*) can explain this disenfranchisement: (1) causality is currently understood in terms of general laws (not idiosyncratic explanations); and (2) the neurobiology of reasons remains obscure, and hence there is no material or organic handle with which to convert reasons into real physical causes. There is also the question of whether reasons (as it is mostly believed in medicine, jurisprudence and moral theory) entail intentionality¹⁰ and conscious awareness; in other words, whether in order for person P to claim that he had reason *x* for behaviour *y*, he must have been aware *pre facto* of *x* and then taken a decision that *y* is the best course of action. This view is considered by others as narrow; for example, according to psychoanalysis, it would be sensical to claim that John gives so many presents to Mary because he is in love with her although he is not aware of it (and also sensical for John to say likewise *post facto* as an explanation for his generous behaviour).

The application of the language of reasons to psychopathology makes things complex. However, a claim by a clinician that a patient has a reason to report 'I am hearing voices when there is no one around' should not be dismissed *tout court*: at a superficial level, it may mean that the patient is believed to be 'entertaining an image' (i.e. experiencing something in terms of the old, passive recipient model), or it may mean that the clinician believes that the patient is actually participating (consciously or unconsciously) in the construction of his experience and that this participation is far more important to the fact that he is having such an experience than whatever signal might be flashing in his brain. This claim by the clinician would be based on a model of symptom formation in which formatting codes are essential to the constitution of the experience itself and its report. Let us say that a patient experiences an inchoate bundle of sensations (elsewhere called 'primordial soup' by us) (Berrios *et al.*, 1995). In order to experience it, handle it cognitively, remember it, and talk about it, he/she needs to configure the primordial soup. He/she does this by means of formats (personal, familiar, cultural) and in negotiation with the clinician (all these factors are extrinsic to the primordial soup). Once this process has been completed, the formatted product will be reported as a voice, a strange belief, sadness, anxiety, a physical sensation, etc. It follows that the fact that the primordial soup is always the result of a brain signal (related to a lesion or dysfunction) is not sufficient (and on occasions not necessary) to explain the utterance. This is because the utterance is about not the primordial soup as such (or it is, but in the remotest of ways) but a final product that is the result of a formatting cascade integrated by many reasons and decisions. In other words, the conceptual distance between brain signal, primordial soup and (reported) mental symptom is so wide that to say that the brain signal is the cause of the mental symptom (for it might correlate with it) has little explanatory meaning.

¹⁰ Intentionality has at least two meanings. According to the psychological one, as per Brentano (1973), all mental acts, *ex definitione*, have a content and are addressed at something. According to the causal (narrower meaning), intention to initiate (or inhibit) action is a requisite component of the very definition of behaviour (some may want to include here unconscious behaviour).

This claim about the depth of the formatting process must not be confused with the conventional view (which most biological psychiatrists would be happy to accept) that the form of the symptom is determined by the brain site from which it originates (e.g. symptoms related to the occipital cortex are visual, or those related to Broca's area are linguistic). The model of symptom formation explicated above states that not only the content but also the form of the mental symptom is determined by the formatting process. (There is no space in this chapter to discuss the confused and confusing distinction between form and content in relation to mental symptoms.) This has interesting consequences, for if the formatting process can override the influence of the site of origin, then correlations between blood flow (as a proxy for site) and mental symptoms have little meaning.

To recapitulate: (1) brain site and lesion can provide only a jejune and nonspecific causality; (2) the fact that a proxy marker of a brain state (e.g. change in blood flow) correlates with a mental symptom cannot be considered as meaningful; (3) the formatting (i.e. construction) of the mental symptom (i.e. the cause of its form and content) has been governed by reasons; (4) personal factors seem essential to the formation of mental symptoms; and (5) what makes tokens of symptoms (say, hallucinations as reported by different patients) the same symptom is not that all those hallucinations originate from the same location in the brain but that the patients in question have formatted a variety of primordial soups (probably originating in different parts of the brain) by means of the same formatting routines.¹¹

The Technology Alibi

As far as we know, the first person to use the technology alibi was Georget (1820) to explain the failure of biological psychiatry to find any sustainable and replicable connections between brain and madness. Then, as now, it was only a 'matter of months' before the pathology and genetics of dementia praecox were sorted out; then, as now, the expectation was kept alive by the release of premature data; and then, as now, there was never the scientific honesty of publishing a denial saying that what had been claimed earlier was nonsense.

As popular now as it was a century ago, the technology alibi is not a hypothesis but the tail end of a foundational syllogism: the mind is represented in the brain → the mind is tantamount to a set of behaviours → hence, all behaviours are represented in the brain → abnormal behaviours are still behaviours → hence, all abnormal behaviours have brain representation → hence, the fact that no brain representation can yet be found for mental disorders must be due to faulty techniques. It is important to realize, however, that finding something in the brain that might be related to the disorder under investigation would not prove the truth of the foundational claim placed at the beginning of the syllogism (i.e. of the antecedent). To believe that it does is to commit the fallacy of affirmation of the consequent, because reasons other than the foundational claim can account for the same finding. Indeed, the same fallacy is committed by biological psychiatrists who believe that rejecting the null hypothesis actually proves that their own explanation is correct.

General Conceptual Issues in Biological Psychiatry: First Pass

The relationship between neuronal activity and mental symptoms can be examined from three perspectives, namely (1) the nature

¹¹ Biological psychiatry has dealt with this by creating a mosaic model of specialized brain sites according to which the form of mental symptoms is determined by its putative site of origin. Evidence for this is surprisingly lacking as current methodology seems tautological.

of the mental symptoms or syndromes, (2) the nature of the neurological dysfunction/lesion, and (3) the empirical determination of the relationship between mental and neurological dysfunction. The division here is simply for ease of analysis and is not meant to imply any actual conceptual separation between the different aspects of the psychiatric/neurological relationship. Thus, attempting to clarify the issues around the empirical determination of the mental/neurological relationship is crucially dependent on some understanding of the conceptual problems, assumptions and constraints affecting the nature of the variables on either side of the relationship. Here, we are concentrating primarily on the mental symptom/syndrome side for it is here that the main conceptual problems and lack of clarity arise.

Before addressing some of the specific points in relation to the mental/neurological relationship, and that will be the main focus of this chapter, it is necessary to raise, at least briefly, a few general issues. Exploration of the above relationship, i.e. between psychiatry and neurology, is dependent on defining the conceptual framework within which such work takes place. In turn, such a conceptual framework is underpinned by not only different sorts and levels of concepts but also assumptions relating to the degree to which such concepts can be understood and held valid. Here, three levels or perspectives are raised in order to highlight the difficulties that need to be acknowledged and the assumptions that must be made in attempting such work.

The Mind–Body Problem

The first, and perhaps most obvious, issue relates to the mind–body problem. In other words, the issue here revolves around the conceptualization of mind and body as distinct categories of substances (various forms of dualism) or conceptualization of them as belonging to a single substance (various forms of monism). Depending on the sort of view one has, different sorts of conceptions will be held in relation to ‘psychiatric’ and ‘neurological’ events and the relationship between them.

Taking a position vis-à-vis the mind–body problem is relevant to the biological psychiatrist from both the conceptual and historical perspectives. Historically, it makes him/her aware of the fact that in regard to FCs 1 and 2, there are fundamental differences in meaning between pre- and post-Cartesian writers, and that for current thinkers it is almost impossible to get into the mindset that must have been natural to pre-Cartesian thinkers. Indeed, half-seriously it could be said that the solution to the mind–body problem is to return to a pre-Cartesian time when man was conceived as a substantial unity. Conceptually, the biological psychiatrist must try to become aware of the views he/she really holds in regards to the mind–body problem. Saying ‘I am an empirical researcher and hence these philosophical issues are irrelevant to me’ is not an option, for it is the case that the very language of neurobiology is soaked in foundational claims. Furthermore, a significant statistical correlation between a mental event (e.g. hallucination) and a brain event (e.g. change in blood flow as per neuroimaging) cannot be understood unless it is given at some level some sort of metaphysical interpretation, i.e. unless some sort of ontological allocation is given to each—and this means taking a position in regards to Cartesian dualism. (On the conceptual basis of correlation, see p. 16.)

Mental Events and Brain Events

Second, we must consider the relationship between ‘normal’ mental events and brain events. This refers back to the old question of localization of mental states to brain states. The issue is not one of whether mental states are realized in brain states—most would agree that mental states are necessarily determined by brain

neuronal activity. The issue concerns the level of *specificity* that can be assumed. In other words, questions have to be asked whether, firstly, mental states can be localized to specific brain systems (and, if so, on what basis), and secondly, at what level or specificity can (a) mental states (moods, thoughts, fears, urges, etc.) and (b) brain states (molar/molecular, etc.) thus be considered.

For example, if it is argued that mental states such as moods, fears and intentions can, in theory, be localized to specific brain states (e.g. particular brain sites or neuronal systems), then by extension it will make sense to look for dysfunction in such areas or systems in relation to abnormal mental states, such as pathological fears, abnormal moods, etc.¹² It needs to be understood, however, that the above claim is, itself, dependent on assumptions. Foremost amongst these is the assumption that our conventional taxonomy of mental states actually corresponds to the brain/neuronal taxonomy. In other words, is it the case that the sorts of mental states or functions as constructed, identified and differentiated by man actually reflect the sorts of divisions operating through neuronal systems? Is there evidence that thoughts, perceptions, intentions, moods, etc. as individuated by language are individuated in a similar fashion by the brain? Evidence would seem to be stronger in relation to some mental functions (e.g. perception, memory) than others (e.g. moods, worries, intentions).

Similarly, considering the level or degree of specificity assumed in relation to both mental states and brain states demands questions that remain to be answered. For example, in regards to mental states, can worries be differentiated from thoughts, memories from daydreams, etc.? And what level of neuronal activity can be expected then to ‘account’ for preoccupations as opposed to fleeting thoughts, or beliefs as opposed to judgements, etc.? Again, this carries implications for pathological states, for example trying to localize delusions, hallucinations, etc. Furthermore, the level of specificity can go further. What about the content of thoughts, moods, beliefs, etc.? Will fear of dying be localized elsewhere or represented differently than fear of a spider, for example?

Role of Language

Third, from a slightly different perspective is consideration of the role of language in the psychiatry/neurology relationship. Language is used to describe both mental states and brain states, but the issue here is that the language used in relation to each concept has a different epistemic value. In other words, the sort of information captured in relation to mental states (moods, fears, preoccupations, etc.) is different from the sort of information captured in relation to brain states (temporal lobe lesion, cerebral atrophy, demyelination plaque, etc.). Crudely, language of the mind is not equivalent to language of the brain, and this carries implications for the hermeneutics of data (see p. 10).

THE HISTORY OF BIOLOGICAL PSYCHIATRY

The conceptual problems of biological psychiatry can only be understood from a historical perspective; unfortunately, the definitional blurredness of biological psychiatry gets in the way. In other words, is its history like that of a *real object* (horse, orchid, chair, Rosetta stone), a *purview* (aesthetics, ethics), a *disease* (dementia,

¹² The issue of whether emotions can be related to brain sites depends on definition. The passions, an older name for the phenomenon, were conceived of as manifestations of the animal part of man; this view was temporalized by Darwinian evolutionism. Because of their relationship to old brain structures and omnipresence in the brain, emotions are likely to modulate perception, movement plans, memory and cognition. This was also Descartes’ view; hence, the idea that he left emotions out of the picture by ‘error’ is both historically and conceptually nonsense.

anxiety disorder); a *social practice* (priesthood, cobblery, medicine, palmistry), an *ideology* (realism, idealism), or a *construct* (French Revolution, guilt, prostitution)? For the purpose of this chapter, biological psychiatry will be defined as a package of changing ideas and social practices seeking to explain and manage¹³ deviant human behaviours in terms of a neurobiological discourse. This enables the historian to ask why biological psychiatry has been successful, what social needs it meets, its best selling point, and how it reconstitutes itself.¹⁴

Before the Nineteenth Century

A version of the foundational claim (central to biological psychiatry) that mental disorders have a somatic origin can be found in the work of Thomas Hobbes (1588–1679) for whom the causes of madness were ‘motions of the blood and animal spirits as they variously expand and contract; the causes of these motions are phantasms concerning good and evil excited in the mind by objects’ (Hobbes, 1991). As efficient cause, Hobbes (1968) considered both external images of evil and physical causes:

... and to have stronger, and more vehement Passions for any thing, than is ordinarily seen in others, is that which men call MADNESSE. Whereof there be almost as many kinds, as of the Passions themselves. Sometimes the extraordinary and extravagant Passion, proceedeth from the evill constitution of the organs of the body, or harme done them ...

As Gert (1996; p. 165) has noticed, this dual aetiological view helped Hobbes to remain consistent with his materialistic stance: ‘Hobbes can be taken to say that sometimes a defective bodily part produces unusual motions, and sometimes the unusual motions of the passion injure the bodily part.’

Historians of medicine consider Thomas Willis (1621–75) to be the main English sponsor of the iatrochemical theory of disease: ‘All diseases [are] perversions of natural fermentations in which the sulphurous and spirituous particles in the sanguineous mass were set in too great a motion, and the blood therefore became overheated’ (Frank, 1980; p. 167) Brain diseases were explained in the same way (italics in original; Wills, 1685; p. 462):

... that we may deliver the *formal nature* and *causes of melancholy*, we may opine, that the liquor distilled from the blood into the brain (which filling and irrigating all the pores and passages of the brain, and its nervous appendix is both the vehicle and vinculum of the animal spirits) has degenerated from its mild, benign, and *subtle* nature, into an *acuetous* and *corrosive* disposition ...

Willis also believed that these changes could be shown if careful post-mortem studies of the brain were to be carried out (Conry, 1982). By the middle of the eighteenth century, G.B. Morgagni (1682–1771) found little of interest in such work:

¹³ Explaining and managing are forms of accounting that can be felicitous, unsuccessful, etc. but of which cannot be said that they are ‘true’ or ‘false’ because there is no way to ascertain it.

¹⁴ The process of self-reconstitution can only be understood if the history of terms, concepts and behaviours is considered independently. For example, the fact that the word melancholia has lasted for more than 2000 years has led some to believe that the current condition called melancholia is, in fact, the same as that known by the same word 2000 years ago. This is an anachronism based on the ontological view of disease. The same error has led some to express surprise about the fact that schizophrenia was not reported before the eighteenth century. The explanation regarding melancholia is that all that survived was the word as an empty shell. In the case of schizophrenia, the condition was not reported simply because it did not exist: it was only constructed during the late nineteenth century (see Berrios, 2000b).

If you join these six dissections of mine [of patients with a variety of mental disorders] with that which I described to you in the first letter (h) and compared them all with those you have in the *Sepulchretum*,¹⁵ or other books, you will immediately perceive, that among those things which others have observed, *some of them have been never found by me* ...

(our italics; Morgagni, 1769; p. 156). Indeed, Morgagni makes the point that similar forms of madness may show lesions in different parts of the brain; this caused some embarrassment to the ‘specificity’ claim hinted at earlier on by Willis.

Pierre Cabanis (1757–1808) will be known forever as the man who said that the ‘brain secretes thought as the liver does bile’. (Taken out of context, this quotation was used by anti-materialist writers to discredit Cabanis (Chazaud, 1993).) Closer reading, however, shows the complexity of his thought. Cabanis (1802) started with a baroque definition of matter, which, as Moravia (1981; p. xxiii) remarked, ‘comprised a full array of forces and properties from whose physico-chemical combination even the most complex living organisms may emerge’. Cabanis’ matter could thus give rise to a complex mind (and complex mental disorders) (see p. 3). ‘Body’ meant brain, heart, stomach, genitalia, etc., and for Cabanis all these organs generated complex patterns of feelings and meanings; it would be anachronistic, however, to consider this view as an anticipation of James and Lange. Biological psychiatrists interested in somatizations and unexplained medical symptoms could study Cabanis with advantage as his ideas are at the basis of the concept of *cœnæsthesia* and ‘*cœnæsthopathy*’ (Berrios, 2001b).

The Nineteenth Century and After

Georget and Bayle

Based on Bichat’s vitalism,¹⁶ E. Georget (1795–1828) proposed an organic aetiology of mental disorder (and introduced the technology *alibi*). His views seemed confirmed by A.J. Bayle (1799–1858), who ‘showed’ that dementia, other forms of mental disorder, and general paralysis might be related. This has been hailed as the *fons et origo* of biological psychiatry, but it is historically and conceptually wrong (Berrios, 1985). Quételet (1990; p. 161) is correct in claiming:

General paralysis thus defined might have done no more than figure in psychiatry nosography (though difficult to situate in the nosography of Pinel and Esquirol) had it not fitted in so well with the arguments of those who believed that the aetiology of madness was necessarily organic ... For the first time, then, something had been discovered in the brains of the insane! Moreover, these anatomopathological lesions were not only to make general paralysis the model of organic mental disease, they were also to swing the psychogenetic conception of madness.

Griesinger

The quotation that ‘all mental diseases are diseases of the brain’ has been attributed to Wilhelm Griesinger (1817–68). A physician (the concept of psychiatrist did not exist at the time) interested in mental disorders, his contribution to ‘psychiatry’ is limited to

¹⁵ *Sepulchretum Sive Anatomica Practica* (Bonetus, 1679) included almost 3000 post-mortem reports; Morgagni set out to check many of its findings.

¹⁶ Vitalism is a doctrine according to which the phenomenon of life results from a principle different from the physical and chemical principles that sustain matter in general. Bichat reacted against metaphysical vitalism, i.e. the idea that the vital principle was part of nature. For Bichat, such a principle can be understood only if it is incorporated into each tissue as sensitivity and contractibility (see Haigh, 1984; Pickstone, 1976).

a textbook, a few papers, and the foundation of the *Archiv für Psychiatrie und Nervenkrankheiten*. His views were grounded on materialist biologism, the new physiology, vitalism and romanticism (Arens, 1996). When Griesinger wrote the first edition of his *Textbook of Mental Pathology and Therapeutics*, he was 28, had little psychiatric experience, and had to borrow most of his clinical illustrations from French and German sources (Griesinger, 1861). Like Jaspers' *General Psychopathology*, published seven decades later, the book is important not so much because of its clinical content but because of its philosophical maturity and imaginative proposals. Griesinger's view that official clinical categories are (and always will be) arbitrary but that their 'elementary' units of analysis (mental symptoms) are ontologically sturdy and stable has provided the basis for descriptive psychopathology (Berrios, 1996).

Put back into its context, and corrected by what the words meant in the 1840s, the claim that 'all mental disorders are disorders of the brain' loses its perspicuity. Griesinger had reacted against German materialism (Gregory, 1977) and was keen on the new physiology of Wunderlich, Roser and Müller; his concept of lesion, therefore, was not anatomical but physiological. It was in the new (and ambiguous) space of the new physiology (Schiller, 1968) where Griesinger located mental disorders. On account of this, and due to the crude ontology to which twenty-first-century biological psychiatry has returned, it is not easy to translate Griesinger's concepts into current categories; for example, although he called mental symptoms 'organic' or 'biological' in nature, he did not expect them (necessarily) to leave an imprint on the brain (Berrios, 1984). His concept of physiological lesion led in the fullness of time to that of psychological lesion.

Meynert

Influenced by Darwinian evolution and the new physiology, Meynert (1833–92) sought to correlate development, function and form. Associationism, localizationism, top-to-bottom inhibition, regional cerebrovascular variations, and the nutritional status of neurons were the pillars upon which he based his speculations on the nature and localization of mental illness. Influenced by the representational ideas of Herbart, he believed that in the network of projection and association fibres, there inhabited a functional ego. The first volume of his *Psychiatrie* (Meynert, 1885), the only one published, is a textbook of neuroanatomy with minor references to psychological concepts (Marx, 1970). Physiological and pathological states (such as obsessions, hallucinations, mania and melancholia) Meynert explained by changes in cerebral blood supply. Nutritional changes at cellular level, resulting from haemodynamic changes or from congenital defect he considered as the ultimate causal mechanism.

The contribution of Meynert to the aetiology of mental illness is not easy to assess (Lévy-Friesacher, 1983 and Pappenheim, 1975). He has been portrayed as a 'brain mythologizer', but this accusation neglects the fact that on occasions he seemed to be speaking metaphorically (he was also a poet), even in matters medical. He believed that brain activity depended on brain nutrition; the latter was controlled by vasomotor activity, thus, circulatory disturbance could cause mental disorder.

Wernicke

A disciple of Meynert, Karl Wernicke (1848–1905) is considered to be one of the most important psychiatrists of the late nineteenth century (Lanczik, 1988). From the perspective of biological psychiatry, three are his important contributions: (1) a model to encompass all brain-related diseases (whether so-called psychiatric or neurological); (2) the development of a pathophysiological model to mediate

between the brain and behaviour, a model that had, until then, been absent from psychiatry; and (3) the introduction of the first neuropsychological approach to mental symptoms.

Central to Wernicke's model was Meynert's idea that the brain of man was endowed with a system of projection and (transcortical) association fibres, and that the latter was the organ of consciousness and high intellectual functions (Wernicke, 1906). Focal lesions to the projection system caused neurological disease; damage of the association system generated mental illness. Using modern jargon, Wernicke was more a connectionist (Stein and Ludik, 1998) than a localizationist.

Neglected Models of Biological Psychiatry

It is an interesting question as to why men like Hughlings Jackson, Von Monakow, Goldstein and Guiraud (*inter alia*) have not had more impact on biological psychiatry. Their ideas are paid lip service but somehow have not been taken up.¹⁷ On account of lack of space, only the ideas of Jackson and Von Monakow will be mentioned here.

Jackson

J.H. Jackson (1834–1911) (Critchley and Critchley, 1998; López Piñero, 1973) spent most of his clinical life giving opinions on the nature of mental disorder. In 1894, he published the 'The factors of insanities'; a paper of relevance to biological psychiatry (Jackson, 1894).¹⁸ Jackson proposed a hierarchical model of the nervous system, with the upper layers inhibiting the bottom ones. Evolution made brain function more complex but less stable; dissolution (Smith, 1982a and 1982b) did the opposite.¹⁹ He defined insanity as a departure from ordinary mentation, which included physiological states such as sleep, chronic brain diseases, and temporary toxic states.²⁰

Jackson proposed that insanity originated from the action of four factors. Factor 1 concerned the depth of dissolution; factor

¹⁷ In general, the history of medicine (and psychiatry) remains a chronology of socially successful views that are presented as truthful. This type of history does not seek to understand and explain but to congratulate. It is of the 'whom to worship' variety. It assumes that diseases are like plants waiting for a discoverer. The alternative view is to see all clinical categories as constructs, and discoverers as those sponsoring views that became a social or financial success. The advantage of this view is that it also lends attention to the losers for their work, which in fact, may be far more important from the point of view of coherence, rationality, humanity and predictability than that of the victors.

¹⁸ Why Jackson has had so little influence on British (and later) Anglo-Saxon psychiatry (compared with neurology) is an interesting and understudied question (Berrios, 1977; Dewhurst, 1982). Even the unsuccessful classification of the symptoms of schizophrenia into positive and negative has been based not on Jackson but Reynolds, although its sponsors believed it to be Jacksonian (Berrios, 1985). This was not a trivial error: the Jacksonian model demands an interaction between positive and negative symptoms that has never been shown in schizophrenia (Berrios, 1992).

¹⁹ Jackson's conceptual model was not meant to be mapped on to the real central nervous system, and he said so. However, efforts have been made to find analogical links (e.g. Kennard and Swash, 1989; Dubrovsky, 1993).

²⁰ He seems to have advocated a continuity (rather than a categorical) view of mental disease, which in practice stretches from physiological states to all gradations of insanity. In this regard, he introduced the concept of mental diplopia, by which he meant definitions of the same behaviour that led to clashes and contradictions, such as using insanity for proper madness and also for a mild degree of drunkenness. Jackson wondered why post-epileptic coma (which he called acute temporary dementia) was not considered as an example of insanity by alienists while post-epileptic mania was. Jackson's concept of insanity or dissolution is only meaningful when compared against a standard, and he used the state of the same person before the disease.

2 the person who has undergone dissolution;²¹ factor 3 the rate of dissolution, i.e. how slow or fast the removal of control was; and factor 4 personal variables, providing lists of putative elements and themes around which positive mental symptoms may condescend. Factors 2 and 4 feed meaning (i.e. information about individuals) into Jackson's model (Berrios, 2001a). Interaction of the four factors gives rise to positive and negative symptoms (Berrios, 1985). The model cannot explain the generation of different types of mental symptoms (e.g. hallucinations, delusions, etc.).

Because of his dualism à outrance,²² Jackson's stance vis-à-vis FC 1 is equivocal. It is true that he believed that all insanities were related to the brain, but his concept of insanity is so different that it is difficult to link the two.²³ Put together, his views do not tally well. Given his dualism, his concept of mind should have been ontologically independent enough to provide a platform for mental disorders; but it does not. Although not a religious man (Critchley and Critchley, 1998), at some level Jackson seems to have harboured the (nineteenth-century) belief that the mind (or soul) was indivisible and intangible and beyond the reach of disease.

Von Monakow

Constantin von Monakow (1853–1930) authored three books, founded three journals, and mentored great biological psychiatrists (Mourgue, 1931). Together with R. Mourgue, a French psychiatrist and historian of science, he also wrote one of the most important (and neglected) books on biological psychiatry in the twentieth century (von Monakow and Mourgue, 1928). Central to this work is the notion of *Horme*, i.e. the tendency of all living beings to develop all their genetic potential. In each individual, the *Horme* is governed by *Syneidesis*, i.e. a principle that regulates and balances all instincts in the interest of the given individual. These principles govern both function and structure, hence von Monakow and Mourgue develop a neuropsychiatric model of the type that Guiraud (1950; p. 165) called 'dynamomorphological'.

Based on the assumption that biological psychiatry was a subfield of biology, the authors imported into it the notion of chronogenetic localization. This concept required that the variable time was built as a parameter into all neuropsychiatric phenomena. Functions (e.g. movement) are processes that, like music, unfold in time and according to a specific kinetic melody. Hence, it would

²¹ Twenty years earlier, Jackson (1874) had described factor 2 as 'the kind of *brain* in which reduction occurs'. This rephrasing reflects maturity of thinking and a mellowing attitude towards the mentally ill. Jackson believed that variables pertaining to the individual and genetics influenced the form of the insanity but says little else. Personal factors such as age, intelligence and education showed best in states of minor dissolution; 'genetic' factors concerned not the inheritance of specific mental disorders but a tendency to 'give out' to dissolution.

²² Jackson's views on mind, matter, their relationship, and FC 1 are difficult to map in a coherent way. He was so much of a dualist that he borrowed Clifford's concept of concomitance (Berrios, 2000c) to explain how mind and matter might interact. Jackson (see Taylor, 1931) wrote: 'The doctrine I hold is: first that states of consciousness (or, synonymously, states of mind) are utterly different from nervous states; secondly, that the two things occur together—that for every mental state there is a correlative nervous state; third, that, although the two things occur in parallelism, there is no interference of one with the other. This may be called the doctrine of Concomitance.'

²³ For Jackson, insanity was on a continuum with other states, such as drunkenness or dreaming (hence, he called them states of 'temporal insanity'); only its negative symptoms actually reflected pathology (i.e. lesions or dissolution of the higher, human, inhibiting layers of the brain); positive symptoms were the expression of evolution, of *normal* activity produced by the release of *normal* tissue.

be a mistake to attempt to localize processes (i.e. brain functions) in terms of specific brain sites (i.e. space alone). Now, since most mental symptoms are considered to result from disordered brain function, it follows that it would be equally erroneous to try to localize symptoms on specific brain addresses. Influenced by Jackson, von Monakow and Mourgue believed that chronogenetic localization was a late acquisition in evolutionary time, and hence regarded it as a complex but unstable mechanism.

One of the implications of the concept of chronogenetic localization is that both cross-sectional studies and conventional longitudinal studies (as collections of cross-sectional snapshots) are inadequate for the capture of neuropsychiatric symptoms. The latter, von Monakow and Mourgue insisted, have to be observed as they unfold in time according to their own kinetic melody; for example, a hallucination is understood fully only when an entire token or hallucinatory episode, which may last minutes or hours, has been studied. In addition to its conventional cross-sectional features, such an episode includes real longitudinal information, such as, for example, modulations in intensity and changes in imagery, and accompanying emotions can make sense only when integrated along a time dimension. From an aetiological viewpoint, knowledge of these longitudinal variables may in fact provide more information on the brain localization of the symptom than traditional static snapshots.

THE DEFICITS OF BIOLOGICAL PSYCHIATRY

The empirical credibility of biological psychiatry is threatened by not only its weak foundational claims and equivocal history but also the suspicious validity of its data (capture), of its descriptive and analytical methods (data processing and interpreting), and of the rhetoric of its data reporting. There is no space in this chapter to discuss all these problems in depth; to render the points clear, however, one or two examples will be discussed in each section.

Data Capture in Biological Psychiatry

In the context of biological psychiatry, and the psychiatric-neurological relationship in particular, data fall into two groups, namely psychiatric data (psychopathology—symptoms, signs, behaviours) and neurological data (neurology symptoms, signs, neuroimaging lesions, etc.). The focus in this section will be on psychiatric data, but even on superficial examination, it becomes apparent that there is a mismatch between the way in which the 'mind' or psychiatric data are captured and the way in which the 'brain' or neurological data are captured. With respect to the latter, it is evident that ever-more sophisticated tools are being developed and finer discrimination being sought in terms of visualizing brain structure (e.g. computerized tomography (CT) scanning, magnetic resonance imaging (MRI), etc.) and brain processing (e.g. single positron emission tomography (SPECT), positron emission tomography (PET), functional resonance imaging (fMRI), etc.). Pathology is described at molecular, neurotransmitter, amino acid, etc. levels. In contrast, the level of capture of 'psychiatric' data remains much the same as it was in the nineteenth century when such descriptions were developed (Berrios, 1996). In fact, in many ways descriptions of psychopathology have become narrower and less rich than they were then, constrained perhaps by the classification systems of current times. Thus, the problems with respect to psychiatric data capture in the context of biological psychiatry can be divided into two main areas: general issues pertaining to data capture in psychiatry as a whole, and specific issues relating to data capture in relation to biological psychiatry.

General Problems

Some of the general problems facing data capture in biological psychiatry need to be mentioned. First of all, what sorts of data are being captured? Are psychiatric data different from other sorts of data? What kind of differences are we dealing with? How might this affect the informational value we can obtain from such data? What are the implications for use of such data as variables in correlational studies, etc.?

To answer such questions, we need first of all to define the particular psychiatric data. Broadly, these can be divided into four types: (1) subjective complaints of patients (e.g. feeling depressed, anxious, hearing voices, etc.), (2) signs and behaviours elicited by clinicians (e.g. thought disorder, psychomotor retardation, disinhibition, etc.), (3) psychiatric diagnoses (Diagnostic and statistical manual (DSM) IV categories) (APA, 1994), and (4) scores on various rating scales (e.g. Beck Depression Inventory (BDI), Hospital Anxiety and Depression Scale (HADS), Hamilton Depression (HAM-D), etc.).

Subjective Complaints as Data

The Subjective complaints made by patients (given spontaneously or elicited by questioning) form part of the data captured by clinicians and used to determine the presence of a particular psychiatric symptom, syndrome and/or disorder. It is, however, the nature of such symptoms as 'data' that raises the problems: problems that relate to the sort and quality of information that can be inferred from such data. In other words, questions have to be asked concerning the validity and reliability of subjective complaints as data. Such questions must relate to not only how validity and reliability might be determined but also the epistemological justification for the validity and reliability of the data.

By definition, subjective complaints consist of individual interpretations of some sort of perceived dysfunction or change—whether this is experienced bodily or mentally. Where, then, and what are the origins and constituents of such complaints, and how do they develop into symptoms? These are the crucial questions, since understanding the validity and epistemic value behind subjective symptoms as 'data' depends on the answers. If it is assumed that, irrespective of aetiology (brain lesion, neurological dysfunction, external event, etc.), there is a change in the internal state of an individual, then how does that particular change become transformed and translated into subjective complaints? And can it really be assumed that the 'same' (and this particular assumption carries many problems that cannot be addressed here) internal state will result in its equivalent interpretation and equivalent description by different individuals? In other words, can it be assumed that the data obtained from individual reports of internal states are of similar type to the data obtained from reports of, for example, objects in the public domain, such as sightings of trees, or, more relevantly, fracture on an X-ray, pulse rate, lesion location on CT scan or hot spot on PET? One problem here is that there is not the same system of verification in regard to internal states as there is in relation to external events. How can we know that the internal state as described by an individual is actually that particular experienced state that is matched by the description (i.e. does the description match the experience accurately—assumption of validity)?²⁴ Secondly, even if the match is assumed to be accurate,

²⁴ Aspects of this debate (particularly a criticism of the epistemic value of data obtained by introspection) were first rehearsed during the second half of the nineteenth century in the nascent field of 'scientific' psychology. It was then argued by experimentalists that the quality and reliability of information obtained in the laboratory was superior to that obtained by introspection (Boring, 1953). Those who took this seriously started to train subjects into being good and attentive reporters. This created a 'professional' class of experimental, which no doubt biased findings further.

how do we know that that particular experience is the same as the one described by someone else (i.e. assumption of reliability)? Furthermore, there is no good model of symptom formation that might help us answer such things (for a précis of our model, see p. 4). Nevertheless, it is difficult to conceive of the process of interpreting a particular internal state and subsequently articulating this in a particular way, as a uniform one, unaffected by individual and sociocultural factors at any stage. Far more plausible would be the possibility that changes in internal states are going to be interpreted in different ways by individuals. For example, it is conceivable that a particular unpleasant internal state change might be read or interpreted as depressed mood in one individual (or at a particular time) while the 'same' or similar internal state might be interpreted by another individual (or at another time) as anxiety or irritability or pain or tiredness, and so on. The factors that might be important in determining a specific interpretation (e.g. past individual experience, cultural biases, contextual issues, language limitations, contribution from the eliciting clinician, etc.) are not the issue here. The point, however, is that it is more than likely that making sense of changes in internal states, and interpreting such changes, is going to be affected by many factors. This means that it is equally unlikely that there will then be a direct relationship between whatever the neurobiological 'signal' manifesting the internal change is and its outward manifestation as a subjective complaint. In other words, there will be a marked degree of formatting going on (noise from various sources), which will change (distort) the original signal to variable extents.

Further complications arise from the fact that subjective complaints themselves cannot be considered a homogeneous class of data. Such symptoms vary in form (moods, fears, perceptions, etc.) and content (anxiety, irritability, elation, hopelessness, grandiosity, voices, images, etc.), and the heterogeneity is likely to be reflected also in the ways in which such symptoms develop and their overall structures (Marková and Berrios, 1995a). In other words, the formatting by other factors is likely to be different in relation to different subjective symptoms. Complaints of depressed mood, for example, may involve types of interpreting factors other than complaints of hearing voices, which one could envisage as perhaps more directly representing the original signal. And when it comes to more complex symptoms or judgements made by patients, e.g. insight into their mental experiences or illness, then the sort of distortion produced by the very many different factors contributing to the construction of such a meta-concept is likely to be much greater (Marková and Berrios, 1995b).

So, what will all this mean for the validity and reliability of subjective complaints as data for biological psychiatry? Given that at present there is little evidence to indicate that a particular description of an internal state actually reflects in a direct way a specific signal or dysfunction, then we have little to justify the validity of such data. Evidence for validity can only start to be gathered when the factors important in distorting the original signal can be identified and teased out in some way. Similarly, reliability will also depend, in part, on the factors determining validity. In addition, however, the issue of whether a particular signal/dysfunction is going to be experienced (leaving aside the issue of interpretation) in the same way in different individuals is open to question. Or perhaps the question here might more usefully relate to there being sufficient similarity in internal state experiences for the state to warrant similar descriptions (again leaving aside the issue of interpretation). What is evident, however, even on this superficial analysis, is that epistemological justification for subjective data in terms of their validity and reliability remains poor. That is not to say, of course, that such data are not useful in themselves. Individuals do have to communicate about not only external objects and events but also internal states, and in general, degrees of understanding can be achieved between individuals (though clearly to various extents), which would argue for some

level of concordance. Thus, subjective symptoms as data used, for example, in clinical management, in terms of making some sort of sense of what and why a patient might be experiencing a particular state is likely to be of practical importance. The essential issue here, however, is not to confuse the practical validity with the epistemic validity of such data. This is of crucial importance in the context of biological psychiatry and any research exploring the psychiatric-neurological relationship, particularly in terms of inferences drawn from correlational research (see Section 3.2), where the data on either side of the correlation may not be epistemologically comparable.

Objective Signs and Behaviours as Data

Psychiatric data are also collected as signs and behaviours identified by the clinician during clinical assessment. Thus, based on a patient's presentation, behaviour, speech and responses, clinicians will determine the presence of, for example, disinhibition, psychomotor retardation, thought disorder, blunted affect, etc. These types of data are thus dependent on the clinician's interpretation of speech and behaviours manifested in the patients. The validity of such data in turn depends on (1) whether the clinician is actually identifying changes that are, in fact, present in the patient, and (2) whether such changes are correctly described and named by the clinician. (In addition, of course, there is the issue of whether the signs and behaviours that are identified by the clinician are in fact the relevant ones out of many possibilities.) Reliability, on the other hand, relates to the extent to which clinicians will agree on the nature of the data presented.

Once again, the questions here, then, are how can validity and reliability be determined, and what are their epistemological justifications? Examining the types of data in this case, it is evident that the data elicited must be dependent not only on whatever dysfunction is manifest in the patient but also on the clinician's interpretation of the patient's presentation. In turn, this will again depend on a number of factors, including clinical experience, knowledge, individual biases, context of subjective complaints, etc. For example, whether a clinician identifies a sign such as affective blunting might depend not only on the explicitness with which it was present but also on the clinician's past experience in seeing this feature, his knowledge and consequent readiness to identify this, his own concurrent mental state, e.g. level of concentration, and whether the patient was denying, for example, depression and anxiety. It can be argued, however, that given the more direct elicitation of such data, in terms of observation of particular outward manifestations, there is more objective determination of the phenomena and hence less likelihood of distortion by the individual factors related to the clinicians.²⁵ Thus, validity and reliability may, in this sense, be greater in relation to these types of psychiatric data than in relation to the subjective complaints as data. This may well be the case, but the issue remains that interpretation of signs and behaviours must still occur (although perhaps to a lesser extent) so that the potential for distortion is still there and there is so far little known about the factors important in the development of such 'noise'. Similarly, the issue of heterogeneity is again a factor to consider in the capture of these data. It is likely that some signs and behaviours may be more direct manifestations of

dysfunction than others and thus less prone to distortion by clinician factors.

Once more, then, the question must be asked: what does all this mean in relation to the epistemic justification of signs and behaviours as data? Such data are clearly once again very different from the sort of data collected in, for example physical science, electrocardiogram (ECG) readings, measurements of height and weight, etc. The latter are, of course, subject to interpretation as well as are any external perceptions. However, the degree of interpretation involved in the elicitation of signs and behaviours, fluctuating in character, themselves dependent on often unclear or abstract theories, must be of a different and greater extent. Identifying changes in patients and naming them correctly (whatever that may mean) is dependent on more than actual dysfunction or lesion. This, together with the fact that little is known about the specific relevance of the identified in relation to the non-identified signs and behaviours, indicates again that epistemological validity is weak.

Psychiatric Diagnoses as Data

Again, in the context of biological psychiatry and the search to explain or clarify the psychiatric-neurological relationship, psychiatric diagnoses are frequently used as data against which brain abnormalities or dysfunction are assessed. In general, issues surrounding validity and reliability of such data have tended to be addressed by the use of lists of diagnostic criteria and diagnostic categories from established classification systems such as ICD-10 (WHO, 1992) or DSM-IV (APA, 1994). The question arising here, though, is what does this actually mean in relation to the use of diagnostic categories as data in biological psychiatry? Reliability here refers to the extent to which clinicians will agree on a specific diagnostic category as applying to a particular patient. Thus, reliability will depend on clinicians agreeing that a certain clinical state fulfils a number of set criteria. And, insofar as levels of agreement are reached, then the classification systems can be said to be relatively successful. But the issue is that whilst this refers to the general facility with which clinical descriptions can be applied, it does not refer to the nature of the data themselves. In other words, it is again the validity of these data that needs to be questioned. The validity of psychiatric diagnoses refers to the extent to which the diagnostic categories actually reflect specific diseases or conditions. Looking more closely at this brings the epistemological problems to the surface. Making a diagnosis will depend, in theory at least (and arguments have been forward against this assumption; see Berrios and Chen, 1993), on identifying and eliciting the primary psychiatric data, namely history, symptoms, signs and behaviours. These will then need to be checked against the list of diagnostic criteria to determine the best fit and hence the most probable diagnosis. It is apparent, therefore, that the informational value of diagnoses (assuming that these are second-order data made on the basis of primary data [signs, symptoms, etc.]) will be affected, in the first place, by the same factors discussed above in relation to subjective complaints and signs and behaviours. In other words, the epistemic value of such data cannot be greater than that underlying the original constituent data. In addition, there is another problem here as far as validity is concerned. This concerns the actual grouping of the primary data into the diagnostic categories and the question of how far such groupings represent true disease process. And, given the so far lack of knowledge concerning aetiology of psychiatric disorders, it has to be accepted that the current groupings are determined by more arbitrary factors, with face validity based on consensus of opinions, but not by information concerning disease states themselves. Furthermore, epistemic justification of diagnostic categories as data will also be affected by heterogeneity in diagnostic categories. For example, criteria set out for the diagnosis of bipolar affective disorder are likely to have an epistemic basis of

²⁵ Aspects of this issue relate to the old nineteenth-century debate, inspired by the first positivist philosophy, on whether signs were more informative, stable or objective than symptoms. By the end of the century, the success of neurology over alienism seemed to confirm that this was the case (Berrios and Porter, 1995). As far as these authors know, and even within the semiological revolution that took place during this period, there has never been an adequate debate on the epistemic value of signs and symptoms and on the fact that they name continuous rather than discontinuous phenomena (Berrios, 2001c).

a different sort to the criteria set out for diagnosis of, for example, neurasthenia or dissociative disorder.

It is important to emphasize again the point applying to all these types of psychiatric data, i.e. that the issue of epistemic validity should not be confused with their practical utility. It is the former that is weak, and the limitations need acknowledging in research.

The use of specified diagnostic categories, by virtue of their specified criteria, may give rise to assumptions about their validity as data, which may be unjustified since these categories mask the actual lack of epistemic validity underpinning primary psychiatric data.

Data from Rating Scales as Data

To measure is to map in numbers features of objects or processes so that operations on the numbers might obtain patterns of relationships assumed to exist in the objects themselves.²⁶ As a praxis, measuring is likely to have started very early in the history of mankind, particularly in the fields of bartering, commerce, building and engineering, and astronomy. Cubit, foot, fathom, grain, etc. attest to the fact that *ab initio* parts of the human body and/or ordinary objects were used as measures. Since very early, standardization was required to prevent disagreements and cheating; the former process has not yet been completed.

The stage at which measuring changed from being a praxis to becoming an epistemological tool, i.e. a process considered as important in the acquisition of knowledge about the world, is difficult to say. Neither Plato nor Aristotle seems to have felt that measuring added to the rational analysis of reality. By the high Renaissance, however, a theoretical shift towards quantification is clearly noticeable (it is applied to painting and drawing, map making, and even the deployment of soldiers in the battlefield) culminating with the epistemological redefinition of the new seventeenth-century sciences as 'quantitative' (Crosby, 1997). This was a reflection of the belief that nature itself had a numerical structure, and hence that knowledge of such codes would make man a master of reality. Thus spoke Galileo: 'Philosophy is written in this grand book, the universe, which stands continually open to our gaze, but the book cannot be understood unless one first learns to comprehend the language and read the letters in which it is composed. It is written in the language of mathematics ...' Newton shared this view.

By the late eighteenth century, the rational, discursive analysis of quality and individuality had been abandoned by the natural sciences and had to be taken over by a small group of inchoate disciplines; a century later, these became the great group of 'sciences of the spirit', which, in the hands of Dilthey and others, developed their own epistemological structure, set 'understanding' (Verstehen) as their objective, 'Erlebnis' as their subject matter, 'hermeneutics' as their tool, and shunned quantification altogether. For a while, psychology was considered to be part of the *Geisteswissenschaften* (e.g. by Dilthey), which may explain why efforts to introduce measurement into psychology met resistance at first. In the event, Weber, Fechner, Donders and Ebbinghaus encouraged the unilateral independence of psychology, redefined concepts, and set the process of its 'naturalization' going (Boring, 1942; Boring, 1950; Boring, 1961).²⁷

²⁶ These may range from comparative orderings (larger than, equal to, etc.) to complex pattern-recognition techniques said to be able to extricate from data sheets meaningful numerical configurations. In either case, the assumption is that numbers are mapping configurations that exist in nature.

²⁷ That is, the view that because the mind is a product of natural evolution, then its study should also fall within the purview of the natural sciences. The narrower view that meaning and individuality are irrelevant to scientific psychology is based on additional assumptions adopted happily during

In the shape of rating scales, graphic representations and statistical analysis, measurement did reach psychopathology and psychiatry after the Second World War. Rating scales and graphic representations had been in use in psychology since the turn of the century, and statistical analysis had become available since before the Great War but was not used in medicine or psychiatry. Although momentous, these events have been neglected by historians.²⁸ All we know is that rating scales arrived furtively, sometime during the late 1950s, hidden in the Trojan horse of the incipient drug industry of the day. Early scales (such as the Hamilton Depression Scale) were designed exclusively for the evaluation of drug trials (Berrios and Bulbena, 1990). After some resistance, rating scales become popular for they seem the ideal response to the *cri de coeur* for objectivity and reliability so characteristic of biological psychiatry, and because they yield the scores required by statistical manipulation.

Given the hybrid nature of psychiatry (a discipline whose subject matter requires both the natural and human sciences), it is extraordinary that at the time (and since) it occurred to no one that the introduction of quantification needed to be justified epistemologically.²⁹ Of the three topics listed above, only rating scales will be treated in this section. We will mention statistical analysis later. There is no space to deal with the numerical, aesthetic, rhetoric and heuristic aspects of graph representations.

Rating Scales

Under the aegis of the first mental tests—such as the Ebbinghaus word lists to evaluate memory (Ebbinghaus, 1964), questionnaires appeared towards the end of the nineteenth century purporting to capture other phenomena such as hallucinations (Parish, 1897). (Lest it is concluded that this is the beginning of measurement in psychiatry, the point must be made that Parish and others were amateur gentlemen wanting to test beliefs in the parapsychological and psychical fields.) Encouraged by eugenics, educational policies and the war effort, 'proper' tests were first developed to measure intelligence and personality. The development of psychometry and factor analysis was, in turn, spurred on by the need to standardize and process such instruments.

When rating scales arrived in psychiatry in the 1950s, the view that mental symptoms were immeasurable was still common, and not only amongst those of a psychodynamic persuasion.³⁰ However, the need to measure outcome in drug trials encouraged the construction of rating instruments based on items believed to be susceptible to rapid change (in days or weeks), their incomplete item coverage making them unsuitable for diagnosis (e.g. the

the behaviourist era. Current 'cognitivism', particularly the one based on information technology, has not been able to regain any of the old semantic and symbolic approach to human behaviour.

²⁸ The arrival of quantification in psychiatry needs analysis. The historian is interested to know about the social conjuncture that allowed its adoption, particularly the groups that benefited from it.

²⁹ The issue here is not whether measurement should be adopted by psychiatry. The issues are: (1) what types of information or forms of meaning included in the psychiatric discourse are susceptible to measurement? and (2) how much of the semantics of qualia is lost after it is translated into the items of a rating scale? These questions cannot be answered by empirical research.

³⁰ Personal communication by the late Professor Max Hamilton. The immeasurability hypothesis must be taken seriously. It is based on the view that mental symptoms are semantic events, and that mensuration of features external to meaning or imposed upon their structure (such as dimensions, intensity and localization) missed out on the only element relevant to the understanding and management of mental symptoms. A claim of immeasurability can also be grounded on technical argument, such as the unreliability and low epistemic value of proxying (see p. 16).

Hamilton Depression Scale) (Berrios and Bulbena, 1990; Berrios and Marková, 2002b).

The numerical and semantic meaning of the resulting global score has also caused confusion amongst biological psychiatrists. It does not take long to realize that such a score masquerades as a continuous number. In fact, it is just a totted summary of qualitative (ordinal scale) decisions taken at item level. In the case of the Hamilton Depression Scale, for example, not all items are captured by scales of the same size. The reasons why some researchers believe that a continuous number can issue out of the arithmetic addition of qualitative decisions is one of the mysteries of psychiatric psychometry. There are, however, hundreds of papers where the Hamilton global score has been entered into statistical analyses that are known to demand numerical and parametric features of the figures involved which the global scores cannot hope to meet.³¹ The curious thing here is that biological psychiatrists, in general demanding of reliability and objectivity, are happy to accept global scores as numerical proxies of that kind. In fact, the best approach is to understand global scores as summary narratives, as forms of mapping a phenomenon by means of the capture of its features (items).

Something must be said on the noble and mysterious concept of 'item', the 'unit of analysis' of psychiatric scales. Items are claimed to stand as semantic and mathematical proxies for attributes, dimensions or expressions of a phenomenon under measurement (see *The semantic mediator of rating scales* below). The content of items ranges from questions meant to capture the presence of the phenomenon (the question 'hallucinations present/absent' is an instrument with one item; indeed, it generates the global score 1/0, which is ordinarily entered into statistical calculations), to scalar anchor points (nominal, ordinal, interval, ratio and, very rarely, absolute forms of measurement), to visual analogues. All these contents are made to yield a number. Through its content, each item is also made to fulfil a proxy function, and the success of any scale depends on the quality of the proxying. So-called standardization methods (including those addressed at evaluating reliability, validity and internal coherence such as Alpha Cronbach) do not, indeed cannot, evaluate the proxying function of each items (on the complex problem of proxying, see Section 3.2.1). In a way, the relationship of a rated psychiatric instrument to its items is (on the model of DSM-IV) like that of a made diagnosis to the mental symptoms or criteria it is based on. Much fuss has been made about the reliability of psychiatric instruments and of DSM-IV diagnostic categories, but all this work is vacuous without having a clear idea of the proxying function of individual items and the way in which mental symptoms themselves (criteria) are actually recognized.

The Semantic Mediator of Rating Scales

Whether depression, thought disorder, apathy, anxiety, alexithymia or whatever, rating instruments do not measure *directly* the target clinical state, trait or condition. They measure it indirectly, i.e. they are always mediated by a construct. A construct is a notion (such as the concept of virtue or disease) or image (painting) purporting to map or represent something else. Schizophrenia, obsessive disorder, Cotard syndrome, autism, theory of mind, etc. are not objects like stones, horses or stars; they are all 'constructs'.³² They are semantic grids that format opaque and formless behavioural

phenomena and allow their capture. On occasions, the phenomenon may be linguistic or semantic and hence devoid of neurobiology (the co-varying 'something' that is captured by neuroimaging is not necessarily the phenomenon but resources used to express the phenomenon, e.g. linguistic activity). This should not worry us, however, for as far as the patient is concerned, the phenomenon is still a bother, irrespective of whether it has representation in his/her own brain.

Constructs are thus semantic structures that select, circumscribe, impose coherence upon, interpret and create a narrative about mental symptoms. Rating scales hang off these constructs and proxy for them. Hence they have no direct contact with the phenomenon underneath, whether neurobiological or not. Constructs are used to: (1) set the boundaries and interpret the raw phenomenon in question; (2) break it up into features, attributes, dimensions and latent traits (in which latter case it will need to include probes or handles to get at them), thereby creating a 'featural pool'; (3) translate the featural pool into a language that can be understood by the average person; (4) select from the pool attributes according to criteria (pathognomonic, representative, convenient, fashionable, interesting, susceptible to change, susceptible to measurement, etc.); and (5) convert the attributes into items by tethering them to specific response categories.

At each step, distortions and biases of a hermeneutic or semantic nature are introduced into the representation that cannot be eliminated by the statistical evaluation of the scale. Calculating a reliability index (e.g. alpha Cronbach) can only ascertain whether the attributes selected cohere or relate to each other and converge on to a unitary construct; they cannot help to decide on the relationship between the construct and the behaviour. The little there is left of semantic information after a scale has been constructed is to be found in the instructions and in the history of the scale. And yet the latter two are rarely consulted and pondered over, for there is the magical belief that once treated statistically, rating instruments become autonomous entities that do not need to have a past. This is wrong, for all scales belong in cultural and clinical niches and hence wear off, loose reliability through time or mismeasure when applied to phenomena or contexts not specified by their instructions or semantic history.³³ Researchers in biological psychiatry do not seem to bother much about such semantic information; this explains why, for example, they continue using the Hamilton scale (which is, in fact, losing sharpness as the surface symptomatology of depression changes) to measure 'depression' in frontal lobe stroke (and other neurological conditions) in spite of the fact that Max Hamilton instructed specifically that patients suffering from such disorders fell outside the semantic purview of the scale (Berrios and Bulbena, 1990).

The Informational Capacity of Rating Scales

Rating scales are believed to be objective. The denotation of this term is valid and reliable; its connotation is scientific, good, above board, not subjective, sellable, better than other approaches, etc. Hence, rating scales are considered to be a form of assessment superior to the happy chat or the clinical interview. Evidence for such a notion is surprisingly limited, but it survives as a

by medication). This fear may in turn result from the (wrong) belief that constructs are not part of nature but artefacts of language or fictions. This, in fact, is not the case.

³³ This is a well-known aspect of psychometry. For example, the original Wechsler scale was found to have lost calibration because it was standardized in pre-war samples that are now believed to have been less 'bright' than post-war samples. This principle has not yet been applied to psychiatric rating scales in spite of the fact that there is evidence that the expression of mental disorders has changed on account of both neurobiological and social reasons.

³¹ Except in the case of rating instruments based on absolute measurement, global scores cannot be considered as absolute, i.e. as numbers starting from true 0. For example, a 0 score on the Beck Depressive Inventory cannot be interpreted as absence of depression; nor can someone scoring 40 be said to be twice as depressed as someone scoring 20.

³² Resistance to the idea that the categories of psychiatry are constructs may issue out of fear that such a view will undermine their ontology (and consequently threaten their localizability, neuroimaging and management

counterpart to the (wrong) view that interviews are subjective. Rating scales have benefited much from comparing them with rulers and other measuring instruments. In fact, the conceptual distance between a stick and the ruler is much shorter than that between a mental symptom and its scale. By conceptual distance, we mean here the number of semantic interventions required to convert a phenomenon into an item. The more interventions, the more the loss and/or distortion of information. As long as scales are taken to be short-hand, semantic devices, or summary narratives, such distortions matter little. They cause needless fatalities, however, when rating instruments are considered to be mirrors of nature and powerful generators of knowledge, and their global scores to be true measurements (and entered into correlations).

In general, there is nothing magic about meters or scales or the other devices created by man to allocate numbers to objects and qualities. All measurements (including the simplest ones) are mediated by concepts and are dependent on aids taken from anatomical (cubit, inch, fathom) or common objects (grain, stones) that have since become abstracted and standardized. Measurements in earlier days were comparative and relational. Reliability (that users meant more or less the same by each unit of measurement) was in those early days more important than accuracy (the concept of validity is not important in a world of comparisons).

Specific Problems

So far, we have concentrated on problems associated with psychiatric data capture in general. Emphasis has been placed on the issue that psychiatric data are of a different sort to other types of data that are used in research and other situations (e.g. height measurements, pulse rate, number of plaques on MRI, etc.). Whilst some differences in validity and reliability can be identified between sub-groups of psychiatric data, as a group psychiatric data are marked by weak epistemological support and hence poor epistemic validity. This carries implications for correlational research in biological psychiatry where such data are treated as hard variables (see p. 18).

In addition to conceptual problems applying to psychiatric data capture in general, there are issues specific to psychiatric data capture in biological psychiatry that need to be considered. The specificity of these issues is, to some extent, defined by the presumed neuropsychiatric relationship. This relationship is generally taken to encompass several possible perspectives. Limiting things here to a situation where a patient has a neurological condition and associated psychiatric problems, then these perspectives can include views that: (1) the neurological lesion might be causing directly the psychiatric manifestations (directly in the sense of specific brain sites or neuronal systems damaged); (2) the neurological lesion might be causing indirectly the psychiatric problems in that the latter may reflect the response of the patient to the consequences of the neurological condition/disability; (3) the neurological lesion might be both the direct and indirect cause of the psychiatric problems; and (4) the neurological lesion and psychiatric problems might be occurring independently and hence coincidentally. These perspectives, whether or not stated explicitly, seem to underpin a number of the conceptual and empirical problems facing data capture. The problems are various, and again it is not possible to cover all in this chapter.

Behavioural Phenocopies

One such problem concerns the nature of psychiatric data in the context of neurological disease and relates to the question of whether such data can be considered similar or different to those manifested in conventional psychiatric disorders. This problem is raised in a number of ways. For example, in the case of organic depression (e.g. depression in the context of stroke), questions remain

as to whether this might be, in fact, the same sort of condition (i.e. same sort of structure, symptom pattern, behaviour, response to treatment, etc.) as depressive disorder occurring without a neurological condition. ICD-10 classifies organic affective disorders in a separate diagnostic category from affective disorders, but uses 'presumed organic aetiology' as the only discriminator between the two. Some evidence suggests that phenomenologically there may be differences between the two depressions, and that some types of organic depression may be behavioural phenocopies in that although appearing similar, they have different symptom profiles.

For example, a cluster of affective complaints can mimic depression, only to disintegrate after a few days of observation. It could, of course, be the case that the depression has resolved rapidly and spontaneously, but often enough what the observer has noticed is a behavioural phenocopy of depression (Berrios and Samuel, 1987). Such clusters carry implications in relation to the neuropsychiatric relationship perspective in that they may also be mediated by the neurological dysfunction either specifically or as a general response. Another example raising the issue of similarities and differences between mental states in the context of biological psychiatry is that of dementia and so-called 'pseudodementia' (or whatever other name one wants to use to name the behavioural phenocopies of dementia). Thus, dementia-like syndromes can be manifested, in terms of both symptoms and the disease, by other functional and neurological disorders (Berrios and Marková, 2001).

The question, then, is how does this problem of similarities and differences affect the psychiatric data or rather the epistemic validity of such data in this context? Again, in order to tease out some of the possible factors involved, various possibilities need to be considered. First, the psychiatric data in the context of neurological disease may, in fact, be of the same nature and structure as those manifested in conventional psychiatric disorders. In other words, the same sort of psychiatric symptoms, signs, behaviours and diagnoses would occur, and the specific neurological lesion would not be adding anything different or new to the possible psychiatric picture. In this case, the epistemic validity of the data is limited by the same factors discussed earlier in relation to psychiatric data in general.

Second, some of the psychiatric data in the context of neurological disease may appear and actually be different in nature and in structure, from those found in conventional psychiatric disorder. Patients might express different sorts of complaints, talk about strange experiences, and exhibit odd signs and behaviours, all of which might not necessarily fit current lists of conventional symptoms and diagnoses. In the context of the neuropsychiatric relationship, such differences may arise directly as a result of the neurological lesion (relating to the site and/or neuronal system affected) or be mediated by any ensuing brain damage (e.g. dysphasia interfering with abilities to articulate experiences, personality changes colouring behavioural manifestations, etc.). The problems in this situation, as far as data capture are concerned, would relate to the identification, the capture and the naming or classification of such data. For example, where 'new' and odd experiences are complained about, i.e. those not fitting into conventional categories, then it might be more difficult to identify these as data; often, such complaints are then ignored (or forced into the nearest category available). Similarly, were such experiences to be completely new to the patient, it might be particularly difficult to articulate, for they may not have the language to do the experience justice. Again, this may result in ignoring the experience or fitting it into a known category of description, thus causing difficulties in capturing the data. Both such difficulties carry implications for the 'correct' naming of data.

Third, some of the psychiatric data in the context of neurological disease may only appear to be similar to those found in conventional psychiatric disorder. This is the issue raised in relation to mimics and behavioural phenocopies. The question then follows: what does 'similar' mean? The analysis of similarity is complex, but here

we shall use a simple definition: ‘two symptoms are similar when they have many but not all properties in common.’ A property, in turn, is an ‘attribute or quality belonging to a thing or person: in earlier use sometimes, an essential, special, or distinctive quality, a peculiarity; in later use often, a quality or characteristic in general (without reference to its essentialness or distinctiveness)’ (Oxford University Press, 1992). Properties can be essential and accidental. For example, ‘being related to vision’ is an essential property of the current definition of a visual hallucination, and ‘forgetting’ is an essential feature of a memory deficit. On the other hand, properties that can change without affecting the class of symptoms involved (e.g. content) are called ‘accidental’. Both types of properties can generate similarity. Following previous definitions (Berrios and Marková, 2001), the term ‘mimic’ is used here to refer to a clinical state that shows partial similarity to another. In the case of dementia, it may consist of one predominant symptom (e.g. disorientation). The term ‘behavioural phenocopy’, on the other hand, is used to refer to a mental state that resembles more completely the full clinical picture of a particular condition. For example, organic depression could be considered to be a behavioural phenocopy of depressive episode, and depressive pseudodementia might be said to be a behavioural phenocopy of dementia (Berrios and Hodges, 2000). In addition, behavioural phenocopies are met with in clinical practice that remain unnamed and hence unrecognized because they are fleeting, clinically uninteresting, or cause little subjective distress; all three, however, are still temporary coincidences of symptoms (each originating from a different mechanism) that create the impression of being something else.

The question of whether all similarities engender mimics remains to be answered. Nor is it clear whether essential or accidental properties are more likely to give the impression of similarity. For example, in a patient with psychomotor retardation and memory deficits, the presence of delusions with a content of poverty (an accidental property) will be more likely to create a copy of depression than a delusion with another content. On the other hand, in a patient with confusion and memory deficits, the presence of visual hallucinations (an essential property), regardless of content, may create a copy of Lewy body disease (Perry *et al.*, 1996).

Similarity can be predicated of both symptoms and diseases. In regards to the latter, patients are said to have a similar disease x , regardless of whether they share all the same symptoms pertaining to x . For example, if x is constituted by symptoms x_1 to x_{10} , in an extreme case one patient could suffer x_1 to x_5 and another suffer x_6 to x_{10} and they would still be considered as having the same disease in spite of their phenomenology being different. In practice, however, not all symptoms x_s have the same weight (e.g. some may be considered as pathognomonic, *sine qua non*) and hence decisions taken concerning similarity depend on more complex judgements relating to patterns and selection of symptoms presented. Alternatively, both patients might have x_1 to x_5 and would thus show very similar presentations of the disease.

The issue of similarity, or mimics and behavioural phenocopies, is clearly important, but what are the implications for psychiatric data capture? One way to answer this and to examine the epistemic problems around the psychiatric data capture in this context is to separate out where such similarity might occur. In other words, the question first is at what level might the similarity be generated? Following the previous line of analysis, broadly three levels can be distinguished here: (1) the level at which the actual dysfunction is being *experienced* (ontological level); (2) the level at which the internal changes are being *interpreted* and/or manifested; and (3) the level at which the psychiatric symptoms and signs (data) are being *captured* by the clinician and hence identified and named. The level at which similarity might be generated carries different implications for the epistemic value of the resultant psychiatric data, and it is useful to briefly examine each of these in turn.

Experiential Origin

Similarity may be generated at the level at which a particular dysfunction is experienced. This would mean that different sorts of lesions or dysfunctions would result in similar internal experiences. For example, similar feelings of low mood, fatigue, amotivation, etc. may be produced in response to a brain tumour, stroke, multiple sclerosis, dementia or major depression. Whether it is individual symptoms or whole clinical syndromes that are experienced as similar matters little at this point. The issue is, however, that similar experiences may be produced as a result of quite different brain insults. To make better sense of this, the notion of similarity here demands deeper analysis. The latter, however, is complex and well beyond the remit of this chapter, since we are dealing here with an ontological issue, itself dependent on the exploration of underlying problematical concepts such as identity and localization. Here, the only point to highlight is that at the level of symptom origin, internal changes might be experienced as similar and hence give rise to similar clinical pictures. This issue relating to the specificity or non-specificity of mental symptoms in relation to different brain insults is not new. During the late nineteenth century, there was a debate as to whether the phenomenology of the organic mental disorders (also called exogenous psychoses) always carried information or a mark that betrayed its underlying aetiology. For example, did the symptomatology of exogenous psychoses produced by hypothyroidism, brain tumours, toxins, meningitis and brain injury differ from each other in any way? Again, there is little space here to expand on this, which was but a remnant of the old medieval theory of ‘signatures’. Kraepelin (1899) believed that, in principle, such subtle marks were embedded in the phenomenology of most exogenous psychoses but that not enough research had yet been done to identify them in all cases. By the early twentieth century, however, and mainly under the influence of evolutionary theory, views began to change. In the event, Bonhoeffer’s proposal was accepted that the brain had only a limited number of stereotyped responses to insult and injury (confusion, delirium, stupor, cognitive disorganization, etc.), which were triggered by whatever aetiology. Subsequent questions asked by Bonhoeffer (1910), Redlich (1912) and others referred to whether some aetiologies preferentially chose particular stereotyped responses.

What, though, are the implications for the epistemic validity of the psychiatric data, in the context of biological psychiatry (the neuropsychiatric relationship), if similarity were to be generated at this level? If very similar internal states are being generated, irrespective of the specific cause, then any determinable clinical differences will depend not on the original signalling but on the nonspecific (in relation to the brain or other trigger) individual interpretation (formatting) of the internal change. Such interpretation would, however, represent the ‘noise’ in the system rather than reflect specifically the original signal. Thus, mimics or behavioural phenocopies arising at this level may have similar core structures but different subjective colouring and different aetiologies.

Epistemic justification of such psychiatric data in this situation will therefore be weakened on two main grounds. Firstly, the factors important in the interpretation of internal states, as discussed in the general section, remain to be established. Secondly, and specifically concerning the similarities issue, as far as the relationship to the neurological lesion/dysfunction is concerned, then the informational value of the psychiatric data will be limited primarily by the level at which original signals can cause different internal changes. In turn, this depends on understanding at a deeper (and as yet unknown) level the mechanisms underlying brain changes and their mental manifestations. In practical terms, clearly similarity occurring at this level would mean that attempts to develop finer clinical or phenomenological discriminators would not help to differentiate between ‘true’ psychiatric

states (e.g. depression, psychosis, etc.) and different mimics or behavioural phenocopies.

Interpretative Origin

Similarity, postulated to arise at the level of interpretation of an internal change, carries different implications for the nature of psychiatric data. Here, the idea is that different brain events or lesions produce different internal experiences, but similarity is generated through a commonality of interpretation. In turn, this implies that there may be limits to the way in which internal states are interpreted and named by individuals. Such limits are different from those mentioned above, since they are imposed not by the signals themselves but by the capacity of an individual to describe and discriminate between different internal states. For example, it could be envisaged that different states of sadness, emptiness, gloominess, tension, irritability, etc. could all be interpreted as feelings of depression associated perhaps with loss of interest and enjoyment. In turn, these could lead clinicians to infer the presence of depressive disorders. Nevertheless, it may be that these differences and other qualitative differences in internal states may, in fact, be real differences, in the sense that they represent different (and significant) internal changes. Similarities are thus invoked on account of the way in which such changes are interpreted and articulated. In terms of epistemic justification for the validity of psychiatric data in this situation, there are, therefore, the same general problems as mentioned before in that the factors important in determining such interpretation and articulation will need to be understood. Firstly, it may be that differences may exist between individuals in terms of sensitivity to changes in internal states. Some individuals may be able to discriminate between more subtle differences than others. Secondly, even if all could discriminate to the same level, then limits to interpretation may be imposed by language itself and the linguistic categories available to the individual. Again, it is only by determining and clarifying such factors that the validity of the psychiatric data and the neuropsychiatric relationship itself can be improved.

Observational Origin

Similarity can also be generated at the level at which psychiatric data are being elicited and captured by the clinician. In other words, there may be differences in clinical symptoms, signs and behaviours that are simply not being determined by the clinician. For example, patients may be manifesting and/or expressing symptoms and behaviours that are suggestive of a depressive disorder and hence interpreted as such by the clinician, whereas in fact the clinical states are similar only superficially. Epistemic validity of psychiatric data here is dependent not only on the previously discussed problems of validity and reliability in general but on the specific factors determining similarity. The latter were raised earlier in the discussion concerning the notion of properties both essential and accidental and will not be explored further. The main point, however, is that, as elsewhere, such factors remain largely unresolved and hence again contribute to the weakness of the validity of the psychiatric data. Practical implications follow from this since, in theory, developing finer phenomenological discriminators would help to distinguish between mimics and behavioural phenocopies arising at this level. Thus, in the example above, applying finer discriminators to the patient with a behavioural phenocopy of depression might help distinguish those clinical states likely to benefit from antidepressant medication from those needing other forms of management.

In summary, psychiatric symptoms, signs and behaviours are data of a different sort to those captured in the physical/biological sciences and, as such, present specific problems in terms of their epistemic validity that carry important implications for both

the clinical and research aspects of neuropsychiatry. Given the increasing technical sophistication of techniques designed to capture infinitesimal neurological lesions or brain dysfunction, the aim in this section has been to focus on the other aspect of neuropsychiatric data collection, namely psychiatric data. It is evident that the latter, in comparison, remain relatively neglected as objects of research, and only some issues relating to their epistemic justification as data have been raised here. Nevertheless, in the context of biological psychiatry, and the neuropsychiatric relationship in particular, further work on such issues relating to psychiatric data will be crucial.

Data Processing in Biological Psychiatry

The section on data capture dealt with threats to the validity of the information on which biological psychiatry bases its conclusions. This section deals with problems affecting data processing. The availability of canned statistical packages has changed the way in which biological psychiatrists conceive (or rather do not conceive) of the meaning and conceptual basis of statistical analysis. The old art of preparatory thinking (calculations in the pre-computer age were labour intensive³⁴) has been replaced by a high-speed, magical processing that in milliseconds flashes on the screen more data than the operator can ever handle. Far from releasing time for the understanding of the concepts and limitations of statistical data processing, computers have just created a mirage of objectivity. The conceptual complexities of biological psychiatry require this bewitchment to be broken. The best research starts at the level of concepts.

The slowness of progress in biological psychiatry research can be attributed to difficult subject matter, bad or inappropriate technology, lack of ideas, and excessive reliance on the epistemic power of statistical analysis. This section will focus on the latter.

The best way to understand issues concerning epistemic power (here, 'epistemic' means capacity to gain, construct, generate knowledge) is through an example: is there any epistemic difference between the claim by a biological psychiatrist that hallucinatory voice x correlates with a hot spot z in Broca's area and the claim by a physical anthropologist that in humans, there is a correlation between size and weight? Unpacking this question should illuminate the (epistemic) problems confronted by biological psychiatry. Before doing so, three issues need to be discussed: representativeness and proxying (as in proxy variables); correlations (as in statistical correlation); and generalizability (as in whether statistical techniques can be used in fields other than those in which they were originally created).

Representativeness and Proxying

It is a common assumption that the world is a complete and stable system populated by fixed objects, facts and events all regulated by the same laws of nature, and that only experimental science can identify and harness these facts for the good of humanity. The furniture of the world is organized in sets sharing similar essential and accidental features (natural kinds) that can be represented (without residuum) and measured by man-chosen variables. Members of a natural kind are so similar (except for some accidental features) that to know them all it is enough to measure some. It is, however, important that the sample selected

³⁴ For example, when done by hand calculator, each factor analysis might take up to one hour. This encouraged reflection on its appropriateness to the project in hand, on the variables to be included, on the type of rotation, etc. It is unclear whether similar thinking goes on in the computer age.

is representative of the group, i.e. within respective value ranges, sample and universe should share essential features.

In real life, things are not as cosy as this. There is little evidence that the world is a stable system, or that its furniture is fixed, or that the laws of nature apply in all instances. The same probabilistic notions that encouraged the development of statistics also influenced twentieth-century physics, and natural laws (and later biology) are now understood as statements of probability (Gigerenzer *et al.*, 1989). To biological psychiatrists working in the no man's land between the organic and the psychological, these new views are of importance; hence, their main worry is not sample representativeness (as there are conventional techniques to resolve such) but what we call here 'object representativeness'.³⁵ This refers to the nature of the relationship between the object and its qualities, and can be divided into ontological and conceptual representativeness and proxying. It should be remembered that representativeness (of whatever type) becomes an issue only when phenomena are studied scientifically with the intention of drawing generalizable conclusions.

Ontological Representativeness

This refers to the nature of the relationship between the object of inquiry and its features (essential and accidental). Whether a diamond, a dog, a virtue or a mental disorder, features must contribute to the ontology (existence) of the objects they characterize. The issue of whether an object is just the collection of its features or a substance from which all features hang is not relevant to ontological representativeness. For when it comes to diamonds or dogs (and exemplars of other natural kinds), essential features must still contribute to the objects' ontology, ideally without leaving a residuum (unless there are unreported features); the point being that the object's total ontology would be diminished if one feature were to be taken away. It remains unknown whether features are aliquots, i.e. contribute the same amount of ontology.

Things become interesting when the same question is asked of objects such as a virtue (justice, prudence, temperance, etc.) or a mental disorder (say, schizophrenia) that are not natural kinds but constructs, i.e. their ontology is dependent fully on a bet (prediction) made by a person in times past (say, Kraepelin or Bleuler) that features x_1 to x_n (which was all that was available to them and is available now) define an object s as a firm candidate for being a natural kind. In this regard, pathognomonic would name features that contribute the most to the ontology of their object. Given the way mental symptoms and disorders have been constructed historically, it is unlikely that even the pathognomonic features themselves are ontologically or informationally homogeneous (i.e. aliquots).³⁶

³⁵ It is important to distinguish between sample, design (ecological) and object representativeness. *Sample representativeness* refers to the assumption that the objects selected for study (the sample) show the same distribution of qualities as the parent universe. Generalizability is warranted by the degree of sameness. *Design* or *ecological representativeness* concerns the view that sampling theory must also be applied to the objects or, more generally, the stimulus or input or environmental conditions of experiments. Because subjects live in a probabilistic world, their responses to tests and stimuli will be tentative in the sense that they never have enough information or training to respond in the deterministic way that the theory dominant in a particular area will predict. In consequence, responses will also be probabilistic and often biased by cultural learning. Design representativeness relates to ecological validity. *Object representativeness* concerns the issue discussed in this section, namely how well the variable represents the object under study.

³⁶ We have studied symptom heterogeneity elsewhere, but for the needs of this chapter suffice it to say that mental symptoms are constructs exhibiting different inner structure, semantic load, and relationship to the brain signal

But then who decides for how long can the bet be allowed to go on? Should that be a scientific, social or economic decision? At what stage should it be said that the people who placed it have lost in that all they produced was an empty construct, a mirage? This does not mean to say that the complaints of the many people who have been told they suffer from schizophrenia are the less real or devastating. It means that those who put together the earlier cluster of features wrongly selected, ontologically privileged, interpreted and explained some of the complaints (features) uttered by those patients. In doing, so they created a malformed concept,³⁷ a concept that nature, after a century of persistent interrogation, knows nothing about.³⁸

It could be argued that it is nonsense to claim that schizophrenia is like a diamond or a dog or a virtue in that the very definition of disease entails that it is always expressed through someone's body and/or mind. The problem of ontological representativeness becomes more complicated in this case, for now we have object x and its x_n features (i.e. schizophrenia as an abstract disease) and object y and y_n features and object z and z_n features (y and z are, in fact, two people suffering from schizophrenia). The question here is whether the way in which the disease expresses itself through the brain resources of Messrs Y and Z will distort the distribution or pattern of ontological representativeness suggested by the abstract definition. The answer is that it should, for in each individual, personalized formatting activity will take place that would cause major distortions in the expected patterns, particularly in relation to symptoms that are (1) idiosyncratic re-elaboration and reformatting of primary experiences (which makes them escape the control of whatever lesion underlies the disease), and (2) symptoms that are atavic behaviours written into the brain by evolution and hence nonspecific, and (if we are to believe Hughlings Jackson) are regularly 'released' by the lesion to the point that some believe that they are caused by it.³⁹ It can be concluded that disease features relate to bearers in different ways (have different ontological representativeness), and hence the choice of a feature or features to establish correlations with other variables poses interesting problems. These cannot be handled statistically, for the values for ontological representativeness are unknown.

that putatively originates them. These dissimilarities result from the fact that they are constructed by different mechanisms that provide them with their specific form and content. Structurally, there is very little similarity between a hallucination, delusion, sadness, fear, etc. The real difference is determined not by content or brain site but by the formatting imposed upon the symptom at the moment of its formation (Marková and Berrios, 1995a).

³⁷ Conceptual malformation results from semantic underspecification, poor biological anchoring, negative definition, conceptual parasitism, over-contextualization, porous boundaries, anomalous linguistic practice, etc. Malformed concepts can be identified, understood and corrected only by historical analysis (not empirical research). There is nothing terrible about psychiatry creating malformed concepts. It is in the nature of the descriptive and explanatory enterprise that this happens. What is bad is that biological psychiatry is not aware of malformed concepts and hence has no mechanism to deal with them.

³⁸ The problem is that since no real neurobiological marker has ever been discovered, samples of schizophrenic cases are selected in terms of conventional feature arrays, which seriously distorts the interrogation of nature exercise, e.g. if pancreatic weight were to be an important marker, how could that be discovered if that information is not collected in the first place? For a full discussion of these issues in relation to schizophrenia, see Berrios, 1995; Berrios, 2000b.

³⁹ If frequent (say, like auditory hallucinations), dormant features may be taken to be pathognomonic in spite of the fact that they are related to the disease neither structurally nor aetiologically. The fact that they often they show a high correlation with diagnosis is a good example of the unreliability of correlations.

Conceptual Representativeness

Given that we do not know about the ontological import of symptoms (features) in relation to specific bearers, choosing those with adequate representativeness (say, for the creation of a rating instrument or a diagnostic tick list) becomes difficult. Conceptual representativeness refers to the relationship between the chosen features and the expression of the disease (i.e. how a subset of features co-varies with a vectorial representation of the disease).⁴⁰

Now, a statistical solution (such as 'random' selection) will not do, for disease features are structurally and empirically different from one another.⁴¹ This is certainly the case intra-individually and some may want to argue that the same holds across individuals. For example, let us say that disease *d* as specified by DSM-IV consists of criteria c_1 to c_5 . Although criteria are often made to include more than one feature of the disease, we shall assume that there is a 1:1 correspondence between criterion and feature. As a rule, c_1 to c_5 are considered as different from each other in all respects (phenomenology and structure), but the manual does not specify their representational or epistemic validity. Previously, this notion was also conveyed by categories such as primary/secondary, pathognomonic/accidental, etc., but for some reason symptom hierarchies have gone out of fashion.

However, it is dangerous to assume that all criteria (features) have the same representational validity (*vis-à-vis* the disease) since little is known about the structure of the concept of mental disorder in general and of schizophrenia (or any other disorder) in particular. Indeed, it is even unclear whether it makes sense to claim that all mental disorders share a similar structure or, as in the case of mental symptoms, each group (psychoses, neuroses, personality disorders, etc.) will have its own internal structure, which would have different implications for the way in which features relate to each disease. At the moment, features are chosen in terms of criteria, such as ease to measure, availability, saliency, etc., but not in terms of their conceptual representativeness.

Proxying

Proxying is a form of conceptual representativeness. A proxy variable is defined as 'a variable⁴² used as an *indirect measure* of another variable when that second variable is difficult to measure or to directly observe. For example, the frequency of abuse of street drugs is difficult to measure but it can be studied through the proxy variable of hospitalizations for drug overdose' (Reber, 1995). Proxying is often used in biological psychiatry, and hence it is important to know how it works.

The practice of proxying is based on the assumptions that: (1) variables can represent other variables (where 'represent' seems to mean co-vary) and (2) sensible or plausible proxy variable selection is possible. The first claim can be understood as meaning that variable *p* (proxy) co-varies with variable *x* (proxied), or that *p* and *x* co-vary with variable *z* (although not necessarily with each

other). In order for proxying to be made to work, the biological psychiatrist needs a model that embraces *p*, *x* and *z* (otherwise the exercise become nonsensical). The assumption that sensible or plausible proxy variable selection is possible leads to the same conclusion, namely that selection can be justified only on the basis of a model. Only on the basis of the latter could finding a significant co-variance be reasonably taken to be a form of ascertainment.

Ex definitione, a proxy variable is further removed from the object of inquiry than the proxied one. This means that it is less under its ontological and epistemological control and that is more exposed to extraneous factors than the proxied variable. Because in most cases the distance between object and proxy variable is unknown, the latter is chosen on the basis not of science but of expediency. For example, the choice of the proxy variable 'hospitalizations for drug overdoses' to measure levels of street drug use or of using 'changes in blood flow' for changes in brain activity (and thereby subjectivity) may not sound extremely plausible, but it is all the biological psychiatrist has. However, in terms of verification (empirical ascertainableness), there is a difference between the two examples. Impractical as it may be, it should be possible to carry out a large epidemiological study to measure street drug usage and its co-variance with hospitalizations. However, no amount of empirical research will be able to ascertain the real nature of the co-variance between blood flow and subjective mental activity.⁴³ Unfortunately, there is no space in this chapter to develop our own model of proxying.

Correlation

One of the assumptions built into earlier metaphysical accounts of reality was that everything in the world was connected with everything else; hence the qualitative or philosophical concept of association (a mode of correlation) can be considered as ancient. The Newtonian and probabilistic revolutions introduced a new metaphor, to wit, that the book of nature is written in the code of mathematics, and that knowledge about objects and their laws and relationships has to be ascertained piecemeal.

The modern concept of correlation appeared in the biological thinking of the eighteenth century as a general explanation for observed (size) co-variations between organs in living beings. Such co-variance could be *physiological*, i.e. organs worked towards a common purpose (e.g. stomach, guts, liver, etc. in relation to digestion); *developmental*, i.e. organs grew in a synchronized fashion (e.g. secondary sexual organs) when serving a specific function; and *featural*, i.e. somatic characters developed in connection with each other. These three aspects of organ co-variance were conceptualized in terms of the old laws of association.⁴⁴ During the nineteenth century, featural co-variance became incorporated into evolutionary theory, for it was characters or traits (anatomical and physiological) that were believed to be the substratum for spontaneous variation and the target for selection.

Up to the 1870s (including the work of Darwin), the description of variation was qualitative, but Francis Galton (Darwin's cousin), Weldon, Edgeworth, Karl Pearson (Stigler, 1986) and others developed quantitative descriptions, which, in the event, became the

⁴⁰ Since biological psychiatrists believe that all mental disorders have a seat in the brain, then the presumption must be that such a feature will also correlate with a vectorial representation of a putative lesion.

⁴¹ All diseases are constructs locked in a particular space and time. The small sample on the basis of which the original description was made tends to be fixed as prototypical. Ideology, opportunity and luck determine the success of the construct: constructors are always guided by expectations and anticipatory concepts, are opportunistic, and must have a modicum of luck.

⁴² A variable is a mathematical symbol for a quantity that can take any value from a set of values called the range. A variable that can take any value between two given values is called continuous; otherwise it is discrete. A quantity that can take only one value is called a constant. This definition does not deal with the issue of how that quantity in question relates to its source, how it is measured, and how it is represented.

⁴³ The term *qualia* (singular = *quale*) has become standard amongst philosophers of mind to refer to properties of mental states and events that determine what it is like to have them, i.e. to subjective, experiential aspects. It is a synonym of phenomenal properties or qualitative features.

⁴⁴ According to associationism, certain principles of thought encourage human beings to assume a relationship between objects, e.g. when they are close together (contiguity), look the same (similarity) or are the opposite (contrast). This form of cognition and prediction is likely to have had its origin in the simple observation that members of the same genus have a family resemblance and march together. First formalized by Aristotle, the principles of association were to become psychological laws in Western thinking (Warren, 1921; Rapaport, 1974).

current concept of correlation. Pursuant to his overwhelming interest in eugenics, Galton wanted to show that 'good' features ran in 'good' (English) families and used for this purpose 'error theory' (originally developed to correct measurement errors in astronomy). He reintroduced the term 'correlation' to name a new idea, a numerical association (coefficient r) that was supposed to measure the strength of co-variances between objects or their properties.⁴⁵

What data are needed to calculate r , say, between objects or features a and b ? One measure of each will not do, for r needs profiles of variance, i.e. it needs to be based on a data series, i.e. a number of magnitudes, degrees of some attribute, or the like, viewed as capable of being enumerated in some sort of order. Series of pairs of data points can be obtained from either a group of people (cross-sectionally) or one individual (longitudinally). The former are single measures (from many individuals) of properties a and b at a given moment in time; the latter are repeated measures (from one individual) taken on a time series. The statistical treatment of both series may be very similar, but the conceptual treatment should be different as they are ontologically dissimilar. As far as the authors know, no mechanisms seem to exist to carry the ontological differences to the interpretation of r .

To make possible their inferences and generalizations, biological psychiatrists must make (often implicitly) a number of assumptions: (1) that if features a and b co-vary, they do so in the same way regardless of the source of the data; (2) that a and b change with time; (3) that the changes will be the same whether spontaneous or provoked by the researcher; (4) that when a and b change, they do so in a synchronized way such that the pattern of their relationship is preserved; (5) that r (as a quantity) holds a relationship of sorts with whatever is happening in the ontological substratum or natural phenomenon (this relationship has been called reflection, picture, mapping, etc.); and (6) that a very low r means that there is a weak or no relationship, and a very high r means that is a very strong relationship. All these are ontological assumptions for they pertain to the reality that the correlation is numerically meant to portray. At the moment, they remain implicit and often contradict each other. We hold that they must be connected with the scientific hypothesis that the researcher is trying to test.

In order to achieve this connection, an ontological model needs to be specified. Is the co-variance pattern the same when a and b : (1) hold a cause-effect relationship (the type of relationship that biological psychiatrists long for but that is, in fact, rarely available as the ontological basis for a correlation); (2) depend on a third factor (this type of ontological account is ambiguous as it can be interpreted as coincidental (trivial) or meaningful); (3) hold a pre-established harmony (this type of co-variance may give rise to a significant r but is empty of meaning); and (4) are truly coincidental?⁴⁶ Is the co-variance pattern the same if the changes in a and b are spontaneous or if they are provoked by the researcher?

⁴⁵ Two objects or features are said to co-vary when changes in one 'map' changes in the other, so that the overall pattern of their relationship remains unchanged. Co-variation may be linear or sigmoid. In the former case, both variables change more or less at the same rate along the range of measurement; in the latter, they may change at a different rate. Correlations may be positive (both magnitudes increment at the unison) or negative (increases in one correspond to decreases in the other). Co-variation is a dynamic and relative concept that must be interpreted. This is done in terms of an ontology model; without the latter, correlations are empty.

⁴⁶ Claims that a coincidence has taken place (i.e. a concurrence of events or circumstances having no apparent causal connection) are meaningful only if made within a specific domain. For example, the claim 'Within this particular theory of schizophrenia, a significant correlation between intensity of formal thought disorder and length of left toe must be considered as coincidental' is intelligible; the same claim made in general about schizophrenia becomes nonsensical.

Correlations are one of the most common forms of statistical analysis used by biological psychiatrists as they search for cause-effect relationships. Since all the number crunching is now undertaken by canned computer programs, many are no longer aware of the operations performed on the data series (or of the data themselves). This can be excused as division of labour between man and computer; less excusable, however, is ignorance of the issues discussed in this section, namely that correlations are patterns of thought based on forms of reality (ontological models) assumed to behave according to specific assumptions. These patterns must dovetail with the general model controlling the area of research in hand (whether depression, brain receptors, PET scan of verbal hallucinations, neuropathology of schizophrenia, etc.). In other words, this knowledge is essential to the biological psychiatrist both in the context of discovery (hypothesis making) and in the context of justification (relating 'evidence' to the logic of the model). Without this knowledge, the scientist is creating empty narratives, just like writing a bad novel guided by a recipe.

Generalizability

Does the fact that statistical techniques were developed to resolve problems generated by particular object arrays affect their applicability to the field of biological psychiatry constituted by different object arrays? For example, does the fact that R.A. Fisher⁴⁷ developed analysis of variance specifically to measure the effect of variable additivity on seed growth (*static* objects) mean that it should not be applied to *dynamic* arrays such as are obtained from the longitudinal measurement of human behaviour? Lest there is confusion, this problem is not about the normality of distribution of variables (and the remedial application of so-called parametric and nonparametric statistical techniques); it is about the relationship between the mapping capacities of mathematical models and the ontological features of an object array on the basis of which they were first developed. In the case of analysis of variance this means object array (seeds) → representations and concepts (choice of variables, definitions, measurements specific to seeds) → algorithms for analysis (analysis of variance). Has the original reality (seeds, static object) formatted the algorithm to the point that the latter cannot be used in object arrays other than the original or very similar one? The stock answer is that the whole point about algorithms is that they are formal and abstract and transferable and hence can be applied to any realm of reality.

To recapitulate, analysis of variance was developed to deal with static arrays of objects (seeds in bags) where simultaneity between variables data points (size, weight, etc.) was guaranteed. The question is whether analysis of variance can work equally well (here, 'work' means able to generate valid results rather than formally applied) when applied to data whose simultaneity cannot be guaranteed. Simultaneity cannot be guaranteed in much of the data collected in biological psychiatry. For example, patients may be tired, thus neuropsychological assessment needs to be undertaken on more than one occasion. (However, when data are entered in a two-dimensional matrix, the assumption is made that data are at least contemporaneous.) Surprisingly, there has been little work on the (temporal) definition of contemporaneity. Without good accounts (backed up by empirical data) of why correlations of variables measured a year apart are epistemically weaker than correlations of variables measured contemporaneously, it is difficult to carry out research in biological psychiatry.

Biological psychiatrists seem to take a common-sense view of this problem. In some situations, say PET scanning of

⁴⁷ When Fisher arrived at the agricultural station of Rothamsted, he found years of raw data waiting for analysis, mostly seed collections that had been cultivated in different environmental conditions (Box, 1978).

hallucinatory experiences, researchers are likely to want to define contemporaneity as simultaneity (i.e. require a time overlap between image capture and subjective experience). However, in many other studies, say analysis of association between plaque density and cognitive deficits in multiple sclerosis, researchers worry less about contemporaneity, i.e. about the event (memory impairment) occurring within a reasonable time of the development of certain other events (plaques) (Berrios and Quemada, 1990). Little is known about what 'reasonable' means in this context or about the way in which simultaneity and contemporaneity affect statistical significance, and the interpretation of results (on interpretation, see p. 20).

All this suggests that when applying statistical techniques to data in biological psychiatry, the researcher must take into account what could be called 'hermeneutics of analysis', namely the relationship between ecological time, semantics (pertaining to the data), and the way in which 'statistical significance' is interpreted. A biological psychiatrist genuinely keen to understand their subject matter should spend more time solving this crucial problem than worrying about formally meeting journal conventions.

Epistemic Value

Now to the question asked earlier as to whether there is any epistemic difference between the claim by a biological psychiatrist that hallucinatory voice x correlates with a hot spot z in Broca's area and the claim by a physical anthropologist that in humans there is a correlation between size and weight. The anthropologist's claim is redolent of the old eighteenth-century debate on correlations between physical features in biological entities; as we have seen above, the question first was given an ontological answer and later a probabilistic one. This happened because in the intervening century, probability and evolution theory had developed sufficiently to change the definitions of species and variability. The former concept reconstructed the idea of biological natural kind; the latter offered the substratum upon which evolution would operate its selection process. The epistemic value of the physical anthropologist's claim depends on three conditions: (1) compliance with representativeness and other sampling requirements; (2) theoretical warrant for their ontological claims; and (3) a factor x , namely a combination of acceptability by peer group, fashionableness and luck.⁴⁸ The anthropologist's claim seems to have met all three conditions.

Although it seems to have the same form as the anthropologist's, the claim by the biological psychiatrist differs in terms of epistemology and ontology. This means far more than the trite claim that all variables differ from all other variables. Therefore, its epistemic value depends on the resolution of these differences. The claim is very different for it reports a correlation between two *sui generis* concepts, hallucination and change in blood flow (deeply different from size and weight). Auditory hallucination is a construct based on the utterance by a subject that they are hearing voices when there is no one around. The issue is not whether the experience and phenomenon are real but that all the researcher has to go by is (1) an utterance and (2) two assumptions: that utterances always report images and that the quality of the reporting is not impaired by the presence of an acute psychotic state.

Although the two claims above are formally similar, only the hallucination/Broca's area claim is trapped in an unresolvable ambiguity for there is no way of telling whether the subject reports an auditory image or the belief that he is having one. From a

⁴⁸ In fashionable areas of research, e.g. neuroimaging, statistical rules may be violated on the excuse that it is early days, and that the mathematics will be sorted out eventually. Manuscripts violating statistical rules (e.g. with no correction for $>150,000$ t tests) may therefore be published and their results accepted by the throng; this constitutes factor X .

therapeutic perspective, this may not matter, as the same treatment is offered. It is, however, crucially important to neuroimaging studies purporting to correlate the utterance with a marker of brain activity. The discourse of science may or may not be about truth, but it certainly is about coherence and reduction of ambiguity, and the one reported above cannot be overlooked for it affects all neuroimaging studies of mental symptoms. In practice, this means that it drastically reduces their epistemic value.

Data Interpreting and Reporting in Biological Psychiatry

How does biological psychiatry fare in relation to data interpretation? It is often believed that in modern research, methodological and statistical algorithms can even indicate what conclusions to draw. This may be so superficially; in practice, however, efforts are made by biological psychiatrists to select and interpret. How do they fare? Due to the nature of its research questions and variables, problems that are standard in other fields of inquiry become magnified in biological psychiatry.

Interpretation and Evidence

To interpret means to expound the meaning of (something abstruse or mysterious); to render (words, writings, an author, etc.) clear or explicit; to elucidate and to explain. The sort of data obtained in biological research are of the type that require careful interpretation in all the senses listed above. An interesting feature of interpretations is that they cannot be said to be true, exact or apodictic; they can only be fair, imaginative, beautiful, audacious, speculative or silly.

Evidence

In biological psychiatry, much is made of the claim that clinical narratives and decision making are now based on evidence. Members of the public may rightly wonder as to how medical and psychiatric decisions were made before the concept of evidence was invented in the Oxford of the 1980s. In fact, the concept of evidence (Latin, *evidens*: clear, distinct, plain, visible⁴⁹) is ancient, complex, kaleidoscopic and changeable, and there is no reason to believe that the current (blurred) meaning will last for long.

One problem with the historical analysis of evidence is that its origins go back to conceptual contexts and worlds that are no more. Another problem is that because it is an epistemological notion (one that talks about how we know the world), its study causes tautologies and self-reflexive contortions. The notion of evidence was first conceived in a world in which, in addition to sense perception, humans were believed to acquire knowledge through a variety of other mechanisms. Thus, between the fourth and second centuries BC, *enargeia* was used to mean 'clear, visible, datum of experience, manifest, in the mind's eye, evident, prominent, palpable, in bodily shape, brilliant, distinct, etc.' (Liddell and Scott, 1994). All these terms revolve around the claim that, whether through perception or directly coming into the mind, objects in the

⁴⁹ The impact of the term 'evidence', i.e. what moves people in the street when told there is evidence for x , relates to its metaphorical force — "clear, distinct, plain, visible"; to the fact that it relates to something that appears to the eye; to the suggestion that it points at something objective, tangible, visible, above board, public, pure, innocent and uncontaminated. Even when evidence is not made public, the point is to reassure people that it could. The personal vision of at least one witness (seeing is believing) is crucial here: 'Now Thomas (called Didymus), one of the Twelve, was not with the disciples when Jesus came. So the other disciples told him, "We have seen the Lord!" But he said to them, "Unless I see the nail marks in his hands and put my finger where the nails were, and put my hand into his side, I will not believe it"' (John 20:25).

world can be known with certainty, and that epistemological feature is called evidence. This also explains the ambiguous purview of the term, namely that it refers to the *feeling* of certainty itself (subjective evidence); to the *objects* that cause the feeling (objective evidence); and to its *inferential force* (epistemological and social authority) (more on this below). When at the end of the seventeenth century John Locke (1959) did away with all methods of knowledge other than experience via perception (fundamentally) and reflection (secondarily), the force of the mechanism of evidence became seriously undermined, particularly in regards to its direct access to the mind (bypassing perception). This attribute of non-perceptual directness survived for a time in the old notion of intuition and also in the later concept of apperception (Lange, 1900) but it died out with them. The legal usage remains fully extant, but it is no more than a generic name for objects or testimonies that can be used as a source of inferences.

Thus set asunder, the concept of evidence has had no safe port of call. Since the Cartesian challenge, perception has been considered as too fallible to provide the epistemological purchase needed by evidence; considering it as a derivative of certainty causes a tautological loop; basing it on the old concept of dictum by authority is not politically correct. The twentieth century cleverly linked evidence to science, and the tautological consequences of this link have been hidden from view: any narrative generated by science is now considered as sufficient epistemological purchase for evidence, and that is the way it has been defined. When someone asks what legitimates the scientific narratives themselves, the answer is that it is evident or that they are what evidence is about. Evidence can work only if it gets its force from somewhere. Originally it came from magic, and the notion then worked at its best; theologians took it over and the divine was a good replacement for magic. Nowadays, it is science. In all cases, it would seem that the concept of evidence fulfils more than a philosophical need, and that is why societies cling to it at all costs.

Evidence as a Social Practice

The claim that certain things are true can be understood in various ways, e.g. true now and ever (static), inching towards the truth (dynamic progressive). Whichever, it needs to be related to, or anchored in, the mind and behaviour of people. People have to be convinced that *a* is the case before they fully accept *a*. Evidence is the mechanism whereby this is achieved. In each historical period, those in control (whether religious, political or scientific leaders) have put together cognitive and emotional packages for the rest of us with the form, 'The world is like *w* and the reasons to believe it is so are contained in evidence *e*.'

Thus, from the perspective of social order, evidence acts as the long arm of the abstract notion of certainty. In this sense, it can be defined as a social practice or mechanism designed to induce in the majority of the collective social an unconditional acceptance (both cognitive and emotional⁵⁰) of certain world views (*Weltanschauungen*). The latter contain 'truths', whether religious, moral or scientific, that are convenient to social order. Evidence is offered as a proxy for truth since there is little choice for the social collective but to accept and internalize as certain a world view that has been chosen for them.⁵¹ Who decides on what counts

⁵⁰ The role of emotions in the acceptance of evidence has not been well studied. This may be because, both in science and the law, evidence is made to appear as the *summum bonum* of objectivity and hence as denuded of emotions. In this regard, it is good to remember Wittgenstein's claim: 'Every explanation is indeed a hypothesis. But someone who is disquieted ... will not find much help in a hypothetical explanation. The latter will not reassure' (Wittgenstein, 1989).

⁵¹ The force of *videre* issues out of the original metaphor, 'Seeing with your own eyes is believing'. The form of the negotiating gambit is therefore,

as evidence, and which world view is more convenient remains a problem. Earlier solutions revolved around authority and the creation of an elite of seers of evidence. However, the type of society that issued out of John Locke's 'democratic individualism' renders the above solutions incorrect, and new solutions had to be found that ideally were based on impersonal authority of statistics and database systems. These are sold as autonomous epistemological devices, untainted by human hand, and hence impartial and objective. By calling them 'the' evidence, these systems benefit from whatever is left of the force of a once magical term.

The Scaffolding of Evidence

Whether in theology, jurisprudence or epistemology, historical analysis shows that the concept of evidence has been shaped by specific metaphors and dichotomies. One distinction concerns the use of the term to refer to (1) facts or objects (e.g. knife, document, etc.) from which some other fact can be inferred, and (2) the testimony of persons as to the existence of facts. In the former case, there is a real object available to the collective; in the latter, there is a claim by a person who has seen or heard something. In the cultures of the West, the concept of evidence was first (fully) used by organized religion. For example, up to the medieval period, there was a debate in the Christian church as to whether the best grounds for believing in god was rationality (what nowadays would be called a cognitive understanding) or faith. Evidence played a role in the buttressing of rationality and was related to the contents of the Bible. Not always perspicuous, part of the latter often were given more than one interpretation. Who was to decide on the correct one became a problem, and one of the solutions was to create a cast of interpreters.⁵²

By challenging the legitimacy of these interpreters, Luther created not only the need to redefine interpretation (hermeneutics) and evidence but also to rethink the relationship between god and men (Lohse, 1987). Another challenge to the old person-based concept of evidence came from John Locke at the end of the seventeenth century, particularly in his conception of the individual as a *tabula rasa* and the democratizing aspects of epistemology (object will reflect identically in the camera obscura of all minds). As against this, the Platonic influence on both Galileo and Newton generated the great hermetic metaphor of the seventeenth century, namely that evidence is encoded in nature, that nature needs to be interrogated or read, and that the code is mathematics. The probabilistic revolution that started at the end of the eighteenth century led in due course to the mathematization and relativization of the concept of scientific truth.

The history of evidence as a relative concept is a consequence of these changes. Evidence-based medicine is part of this social process, whereby the old authoritarian structures have been hidden behind the statistical techniques (such as meta-analysis), which are presented as impersonal cognitive devices able to extract meaningful information (sufficient for decision making) from large masses of data. They have now become the highest tribunal, and the evidence they issue is presented as the truth. Things, however, are not what they seem, for figures need *interpreting*, and this is still done in the way it always was: in smoke-filled rooms by men

'You must believe in this because it is based on evidence' (i.e. on something that you see or you could see). Buttressed by evidence, certainty generates authority. The concept of evidence is therefore central to social order and links stable views of the world with social control.

⁵² The highest appeal station for the intermediaries was (and is) Rome. The doctrine of Papal Infallibility was confirmed by the First Vatican Council (1869–70). It was the role of the intermediaries that Luther was to challenge, and by doing so he not only redefined the concept of evidence within the confines of religion but created a new way in which man related to evidence and interpretation.

(and now some women) who decide in the end what is and what is not passed on to the throne.⁵³

Interpretation

Central to the cognitive and emotional organization of the world is the concept of interpretation (Grondin, 1994). First a noun naming contradictions and options in everyday life, the Greek term was to become a verb to refer to the action of choosing between options. It then entered Aristotle's epistemology as the name for the link between the sign and the mental image or concept (what the Greek philosopher called 'affections of the mind'). Lastly, since choices and decisions were negotiated in words, interpretation became a term that was used mainly in the realm of language. *Hermeneia* and derivatives were rendered into Latin as 'interpretatione'; soon enough, this voice became incorporated into most European vernaculars.

To start with, the practical acts of interpretation (*exegesis*) concerned sacred and legal books; hermeneutics followed as its theoretical discipline. Within Christian philosophy, the problems posed by biblical interpretation were seen as resulting from the distance that existed between man and God; hence a decision had to be made about whether the Bible needed to be interpreted literally, doctrinally, philologically or historically. Luther challenged the Roman Catholic view that interpretations had to be left to the experts. In encouraging a personal reading of the scriptures, and a direct dialogue with the divinity, he opened up the need for a new subjective relationship between man and God and the concept of inner self, as a private space for such encounter emerged from these momentous changes. During the late eighteenth century, J.G. Herder (Pènisson, 1992) introduced the view that problems with understanding could also be due to different cultural purviews. During this period, interpretation is applied to literary works on the assumption that good hermeneutics (which included a study of the worlds, the history of the ideas) should lead to the one final meaning. Although the reader still defers to the author, the view is also introduced that the former may end up knowing more about the work than the latter. Schleiermacher broadens this definition further by making the interpretative task one coextensive with comprehensive psychology, and Dilthey (at the other end of the nineteenth century) sees hermeneutics as the central task of the human sciences (Grondin, 1994). During the twentieth century, the attention of hermeneutics shifts towards the role of the author and in due course conflates it with that of the reader. The idea that texts have only one meaning is surrendered, and the view that hermeneutics is about texts alone is replaced by the view that it is about the study of human communication.

All these changes are of momentous importance to psychiatry and the derivative biological psychiatry. For a time, the natural sciences presented themselves as immune to hermeneutics on the argument that objectivity was about eliminating subjectivity and rendering knowledge impersonal. These claims are groundless and part of the selling rhetoric of science. Like all other discourses, the scientific discourse is also open to interpretation and hermeneutics, and deferring to statistical significance and the results of laboratory experiments is a cop-out. Whilst that stance can be defended in physics or chemistry, it cannot in relation to psychiatry and biological psychiatry. These are best defined as interstitial disciplines, as doings that inhabit the borderlands between the natural and social sciences. Rhetorical reductionisms and naturalizations of the mind do away with the essence of psychiatry, which remains

⁵³ It is not true to say, for example, that quangos like NICE (National Institute of Clinical Excellence, Great Britain) simply transmit to the throne the results of meta-analysis and literature searches. Whether overtly or covertly, they *interpret* evidence in terms of social, political and financial criteria. (NICE is a special health authority in charge of systematically appraising health interventions.)

the specific understanding of human beings with a disorganized psychology. If modelling these processes may be tough, that is not an excuse for giving up and skirting around them (Dupré, 1993, 2001).

Interpretation is essential and pervasive in biological psychiatry, and its practitioners interpret away regardless of whether they have been trained to do so. This makes interpretation haphazard and uninformed. Interpretation occurs at the level of the contact with the patient, the level of the organization of the information, and the level of science making (both context of discovery and justification). The old positivistic view of science jars particularly in the context of biological psychiatry. There is little doubt that even neurobiological research would benefit from a hermeneutic approach to biological psychiatry.

Data Reporting: the Leniency of Fashion

According to the needs of the market place, interpretations of the same biological psychiatry data may suffer a different fate. When in fashion (a sure indicator of large investment by the industry), certain topics or techniques are supported by the major academic funding bodies in the land, and the unsaid message is that they are nearer the truth than older or tired approaches. In addition to an understandable rush by the young, there is an unseemly run by even older researchers who pursue money rather than lifelong research programmes. These fits and instabilities seriously affect long-term team approaches in biological psychiatry, and cherished hypotheses are indecently abandoned as soon as the industry withdraws investment. The current surrender of university research to such oscillations is to be regretted. The publishing industry joins in by showing surprising leniency towards fashionable topics, which are given a much easier passage than 'unfashionable' ones. This practice causes distortions in the public definition of biological psychiatry, and it should be asked seriously whether the privileging of certain topics because they are considered as good investment should not be considered as a form of scientific fraud. Biological psychiatrists should be aware of the way in which the vagaries of the market can destroy any chance of pursuing a balanced and coherent line of research.

CONCLUSIONS

Biological psychiatry is one of the incarnations of psychiatry. It is based on the foundational claims that all mental disorders are disorders of the brain, and that they should be explained by causes and not reasons. In various guises, these claims have individually been made since the seventeenth century, and by the nineteenth century they were defended by organized groups. A particularly keen version of biological psychiatry is predominant at the moment, and its success is due less to anything intrinsic or scientific but to the fact that its definition of mental illness fits in well with the ongoing philosophy of globalism. It is clear that if it was to be decided some time in the future that psychological approaches to mental illness are after all cheaper and a good investment, and that biological therapies are causing too much litigation, etc., universities, the government and the industry without any compunction would pull the epistemological rug from under the feet of biological psychiatry. Let us hope that it does not happen and that (a much improved form of) biological psychiatry goes on for a long time.

Predominance and fashion, however, should not make biological psychiatry immune to criticism. Its *sui generis* and interstitial nature, in fact, creates specific problems that sooner or later biological psychiatry will have to resolve by returning to the drawing board. It seems abundantly clear that the naturalization lark is not working. There are serious problems at the level of data

capture, processing and interpreting, and all threaten its epistemic validity. The extraordinary complexity of mental disorder demands a humbler attitude on the part of everyone and the incorporation of a hermeneutic dimension to its study. The latter should not just be paid lip service, but should be integrated in whatever organic models are going to be pursued in the future. What biological psychiatry requires is a new model of symptom formation and a new language of description (Berrios, 1999). Thus equipped, biological psychiatry will be harder to research and practise, but it will also be far more likely to generate the kind of narrative that may capture the extraordinary phenomenon of mental disorder, and be more helpful to sufferers.

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