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ABSTRACT

A new theoretical framework for understanding and working with the dementing illnesses of old age is presented, and explicated with particular reference to senile dementia of the Alzheimer type. For several reasons the commonly accepted model, which assumes a simple linear causal relationship between neuropathology and dementia, is inadequate. A more comprehensive model, grounded in a monistic view of the mind-brain relationship, must take into account not only the psychological states that correspond to particular brain states, but also both developmental and pathological aspects of brain structure. Using this we can describe the dementing process, ‘normal’ psychological functioning in old age, pseudodementia and ‘benign senescent forgetfulness’. Further, the scope for some degree of ‘rementia’ is explored.

Introduction

It is now becoming clear that virtually all the losses and difficulties of later life are socially constructed: that is, they are a consequence not of the ageing process in itself, but also of the norms and collective arrangements that are taken for granted as applying to old age. There is one form of distress that is faced with particular dread, especially now that life expectancy has been prolonged. It is that of ‘going senile’: in other words, developing one of those dementing illnesses whose course seems to be little more than an inevitable path to degrading incapacity and, if life continues for that long, eventually to a near-vegetable existence. This dread has a clear basis in reality; for although the figures on prevalence are notoriously inconsistent (due, among other things, to the use of diverse diagnostic criteria), it seems likely that among all those aged over 65 in the industrialised societies some 7% will develop a dementia, and among those over 80 the figure may be as high as 20%. Empirically, it is now generally agreed that there are more and less helpful ways of ‘managing’ a dementia; or, to be more accurate, ‘managing’ the person who is dementing. But the overall conceptualisation of these conditions is far from adequate to their complexity, or to the needs of the sufferers themselves.
In much of the literature on dementia, especially that which adopts a medical-science, or ‘technical’ frame, data are commonly presented in such a way as to imply that there is no need to take the psychological level seriously, other than as a source of clinical indicators. It seems to be assumed that once the pathology present in the brain has been clearly described, one can find there a sufficient explanation of the dementing condition. The commonly assumed causal frame, then, which constitutes the ‘standard paradigm’ for aetiology, is linear:

\[ X \rightarrow \text{neuropathic change} \rightarrow \text{dementia}. \]

In the case of the dementias associated with multi-infarction, the nature of \( X \) seems to be fairly well understood; successive minor failures of blood supply cause death to the nerve cells and the accompanying tissues. In the case of senile dementia of the Alzheimer type (SDAT) \( X \) remains mysterious, and there is controversy over whether it represents one or several causal agents. In all dementias the fundamental problem is usually held to be a failure of cognition, associated especially with the short-term memory. Disturbances of mood and behaviour are seen as secondary, either as accompaniments or consequences of the cognitive deficits. The ‘standard paradigm’ originates in medical science research and is applied in a great deal of clinical psychiatry. Among many nurses and other caregivers, however, I suspect that often it is accepted only superficially, and with a good deal of ‘doublethink’.

As I have tried to show elsewhere the ‘standard paradigm’ faces a twofold difficulty, and this is particularly severe in the case of SDAT, with which we are principally concerned in this paper. Empirically, the correlations between the degree of dementia (as measured by behavioural and psychometric indices) and the extent and type of neuropathic change found post mortem are far less strong than seems commonly to be assumed, leaving some 80% of the variance unexplained in moderate or severe dementia; or, putting matters another way, there is a considerable overlap between the observed condition of the brains of mentally well-presented and of demented elderly people. Also in the scanning of the brain using computed tomography (CT), it is now widely accepted that measures of atrophy do not correspond closely to cognitive dysfunction, nor do they lead to sound prognoses in relation to dementia. CT has great value for the purpose of screening out other possible causes of impairment, such as infarction or brain tumours, but is of only very limited use in the positive diagnosis of SDAT. One research group has even gone as far as to suggest that besides the familiar ‘benign senescent forgetfulness’ we should now recognize a new term: ‘benign senescent atrophy’. This points to those cases where CT provides clear evidence of deterioration in brain tissue, but there is no evidence of dementia at a clinical level.

There is a second point, less widely acknowledged. The view of the relation between brain and mind that seems to be widely held by psychiatrists in the field is one of strong, or type-type identity. This postulates that whenever brain state \( Q \) exists, so also does psychological state \( P \), and that whenever psychological state \( P \) exists, so also does brain state \( Q \). This view is exceedingly difficult, if not impossible, to justify with logical rigour, and it seems wiser to assume a less stringent relationship such as
that of supervenience; accepting the radical differences between the psychological and natural scientific types of language.5

The conceptual confusion to which I am referring is revealed in another way in the literature on SDAT. The term dementia, as commonly used in geriatric psychiatry, oscillates uncomfortably between neurological and psychological referents. Dementias are often classified on the basis of lesions found post mortem in relatively small samples; and hybrid terms creep into usage, such as ‘hippocampal dementia’.6 For the two reasons given above, this simply will not do. On the one hand it is necessary to describe and if possible explain the lesions, using concepts belonging to the appropriate natural-scientific disciplines. On the other hand, it is necessary to retain dementia as a psychological term, covering a range of conditions that can only partially be distinguished at a clinical level – all of which involve progressive impairment of such faculties as memory, attention, planning, judgement and emotional response, together with the disappearance of selfhood; dementia means, literally, a ‘loss of mind’. The collapsing of the neurological and psychological frames into one could only be justified if two conditions obtained: type-type identity between brain and mind, and very high empirical correlations between dementia and neuropathology. There are some indications of a recognition of this in recent research; and one of the tendencies of the last few years has been to move away to some degree from neuropathology to clinical phenomenology, both in identifying SDAT and in understanding its progress.7

The relation between brain, mind and dementia, however, remains obscure. This paper is an attempt at a basic clarification. It is grounded in the practicalities of work with SDAT sufferers, but is also (I hope) consistent with the findings of medical-scientific research.

Further problems for the ‘standard paradigm’

There are, as we have seen, two very serious obstacles in the way of accepting the ‘standard paradigm’ as applied to any dementia: the one logical, and the other empirical. But in addition, there are several types of phenomenon that are extremely difficult to accommodate. Four are particularly relevant to our discussion.

(i) Pseudodementia. As is well known in the clinical field, it is possible for a person to present a range of symptoms that look extremely like those of Alzheimer’s disease, and yet for these to be reversible.8 The commonest cases are those associated with depression, where the lifting of mood is also accompanied by a more-or-less full return of normal psychology and behaviour. Furthermore, dementia-like symptoms are often found with conditions such as uraemia, chronic constipation and pneumonia; with the retention of anaesthetics in the nervous tissue after an operation; and with the accumulation of drugs in the body of a person whose rate of metabolism is rather slow. The crucial point is that here – as indeed with some cases of severe anxiety or depression in earlier life – there is clear evidence of cognitive impairment without, apparently, irreversible damage to brain tissue.

(ii) Apparent precipitation. From our own studies9 it seems that in a substantial proportion of cases (perhaps around 30%) a dementing illness of the Alzheimer type sets in fairly quickly after one or more major life crises. Our evidence for this is
derived almost entirely from retrospective accounts given by spouses, daughters and sons, and of course it is open to criticism on the grounds that it involves their own ‘search after meaning’, and hence a good deal of distortion. Even if such evidence is not to be taken literally, however, it certainly should be taken seriously. We have found the onset of Alzheimer’s disease to be associated in the minds of relatives with a variety of crises, such as bereavement, physical illness, severe family conflict, uprooting, assault and loss of major roles. Our evidence is tentative, because it derives from work that is still in its very early stages; but there are some clear pointers in the same direction in the literature; for example Amster and Krauss.10

(iii) Catastrophic decline. This phenomenon relates closely to the second, but is distinguished from it in that it refers to a dramatic downturn after a dementing illness was already clearly in evidence. The most common examples are those following admission into an institution run on a rigid regime, poorly resourced, and with a staff whose own morale is low. Sad to say, there are still plenty of such places, reflecting the workhouse of another era. After entering such an institution, a person who was still in the ‘borderlands of dementia’ might become globally impaired, with complete loss of recognition of relatives and friends, within so short a period as 3–6 months. Possibly this phenomenon has not been sufficiently researched in a systematic way, although it is well known to those who have been involved for some time in dementia care. A tentative explanation has been offered by Barnes, Sack and Shore,11 in their theory of a ‘cycle of dementia’: successive disconfirmations and disempowerments occur social-psychologically, to a person who is already, but not disastrously impaired in cognition.

(iv) Moderate or transitory ‘rementia’. It is time that a term such as this found its way into the discourse surrounding Alzheimer’s disease, for it signifies a phenomenon that is crucial for clear understanding of the nature of this condition. In medical science it is now recognized that treatment with certain drugs, such as THA (tetrahydroaminoacridine), may bring about some degree of (possibly temporary) recovery from dementia in some patients, possibly about one third in a typical sample.12 Parallel phenomena of recovery without drugs are well-known to caregivers, and there are occasional reports in detailed case studies, such as that of Roach.13 Under certain circumstances sufferers from Alzheimer’s disease who had, apparently, gone far down the path of behavioural and cognitive impairment, can regain some of their lost faculties. The conditions for this seem to include a very high ratio of caregivers to sufferers, close personal attention, and a general climate in which care can be expressed physically, together with free expression of the emotions. The best-known indicator of rementia is the re-gaining of urinary and faecal continence, but it is possible also to observe moderate recoveries of memory, social skill, and ability to complete simple tasks, together with a general reduction of signs of anxiety. Even more puzzling than the modest improvements to which I have been referring, are those occasions on which sufferers in the early stages of SDAT are able to exhibit short-lived and fragile restoration of near normal function. It is as if the dense overcast layer surrounding the psyche parted for a few moments, revealing sunshine and blue sky beyond. The family care-giver reports ‘Suddenly it was as if I was with the old, familiar [John, Joan . . .], and I remembered how it used to be with us – I had almost forgotten.’ In some cases this phenomenon seems to be evoked by particular persons.
'My daughter came to visit, and for half an hour he was almost like he used to be. After she had gone, he was as confused as ever.' Also in some cases it is evoked by the opportunity to carry out long-practised, and highly skilled tasks.

It is difficult, although not impossible, to explain phenomena such as these in terms of the ‘standard paradigm’, with its linear causality.

\[ X \rightarrow \text{neuropathic change} \rightarrow \text{dementia.} \]

The pseudodementias have to be presented as a fundamentally different set of conditions from true SDAT, originating solely in neurochemistry, although some of the symptoms of the latter are closely ‘mimicked’ in the former. The apparent precipitation of SDAT is explained in terms of a theory of ‘unmasking’: that is, the person already ‘had’ the dementia, although it was not apparent until after the crucial life events. Here the arbitrary sliding of the term dementia between neurological and psychological frames of reference is particularly apparent. Catastrophic decline has to be accounted for in roughly the same way (the dementia was in fact already far worse than had been realised); for unlike the case of multiple infarction, it is implausible to suggest that massive Alzheimer-type degeneration of the grey matter can occur over a space of 3–6 months. Rementia has to be reframed in terms of a precarious distinction between remediable and non-remediable symptoms, or on the assumption that the original diagnosis was incorrect. ‘Benign senescent atrophy’ remains uninterpreted, but it is possible to postulate a shrinkage of grey matter, without the actual death of nerve cells.

It may well be the case, then, that the ‘standard paradigm’ can now only be upheld through the rather dubious procedure of ‘saving the appearances’; or, to use Popper’s term, through a succession of ad hoc modifications. There is a different, and possibly more fruitful way of making sense of the wide range of phenomena associated with SDAT, which brings the psychological and neurological aspects more closely together. The central hypothesis is not remarkable, although its implications are profound. It is that in all cases of SDAT we are dealing with a combination of structural damage and functional change in brain tissue, and that the pathology found in the brains of SDAT sufferers after death is not primarily causal, but epiphenomenal or consequential; however, the presence of neuropathic change itself sets limits to brain function, and increasingly so as the condition progresses. If so, SDAT is very far from being the straightforward ‘organic mental illness’ (in the old-fashioned sense) that it is usually claimed to be.

The relationship between mind and brain

It is obvious now that there can be no ‘purely organic’ mental disorders, clearly demarcated from those which are functional, and this is acknowledged in the approach adopted in DSM III.14 But what is still far from clear is how the psychological and material (neurophysiological–neuropathological) levels are to be brought together into a single framework. Very few people today find ontological dualism of the Descartes kind – the postulation of two fundamentally different substances, matter and mind – convincing. A basic commitment to monism, however, does
not entail that one can dispense with the psychological level, as some psychiatry seems implicitly to claim. An approach is required that does justice to the validity both of mental descriptions, which we clearly need for our life as social beings, and those other descriptions cast within the frameworks of the various natural sciences.

We may begin in the following way. For every psychological (ψ) event (e.g. having a desire) or state (e.g. feeling elated) in an individual, there is a corresponding biochemical-electrical brain state (b). Or, as some philosophers would say, the one is identical to the other, but the descriptions employ different frameworks: the one intensional and the other extensional (within which law-like generalizations may be stated).

In short, then, \( \psi \equiv b \),

b might loosely be termed a ‘functional brain state’. It exists within, or is instantiated by a brain in such-and-such a structural condition.

The microstructure of the brain arises, as is now clear from neurophysiological and related sciences, in part from the various interneuronal connections that exist and in part from those enduring chemical changes which are associated with long-term memory. There is, no doubt, a genetically given basis to all this, on which a structural development is brought about through a whole range of learning experiences. In the case of experimental animals, principally rats, the evidence for structural change accompanying learning is very strong.\(^{15}\) With humans the subject-matter of cognitive psychology is finding the logical counterpart of certain structural patterns in brain tissue; and it also makes good sense to say that selfhood in all its complexities (and, perhaps, fragmentations) corresponds to a structural configuration in the brain. In this sense brain structure, then, is continually changing. We may well suppose that when a person undergoes intensive psychotherapy and acquires a different sense of self, or takes a degree in middle life, moving from concrete to formal-operational thought in some field, new interneuronal connections are made, and hence a new pattern of brain structure has emerged. For obvious reasons, it is extremely difficult to ascertain the nature of these changes in detail in human beings; but if monism is correct it follows that all relatively enduring psychological developments must have their counterpart in brain structure. In other words, in the brain enduring function gradually becomes structure. Function might be regarded as ‘fast process of short duration’, and structure ‘slow process of long duration’.\(^{16}\) Unfortunately, however, that does not exhaust the story of brain structure, because we have also to reckon with the fact that the tissue is subject to lesions of various kinds, including those which are associated with the two most common forms of senile dementia: Alzheimer-type degeneration, and multiple infarction. Overall, then, brain structure involves two main aspects. These might be termed ‘developmental’ (B\(^d\)) and ‘pathological’ (B\(^p\)). We may express varying degrees of neuropathology as B\(^p_1\), B\(^p_2\), etc. If our concerns were primarily ‘technical’, this would need to be expressed much more precisely, and with reference to type and location.

Thus the full situation, whether in mental health or mental illness, and whether the brain is in good or bad repair, may be represented as follows:
ψ ≡ b,
\[ \text{B}^d, \text{B}p \]

The horizontal line here signifies ‘carried by’, or ‘permitted by’, a brain in such-and-such a structural condition.

It would seem to follow that a formulation of this kind should be reckoned with in all psychiatry, whether of those who are, in traditional terms, functionally or organically ill. However, it is reasonable to suppose that some mental disorders (such as a mild reactive depression or a short psychotic episode) involve changes primarily at the level of brain function rather than structure, and so might be represented as

\[ \psi' \equiv b' \]
\[ \text{B}^d \]

(since Bp here is an irrelevant term).

For most people, most of the time, the structural brain state (\( \text{B}^d, \text{B}p \)) does not determine in a positive way what is the case functionally. So long as the tissue is fairly intact, the brain state simply sets certain limits, allowing a very wide (but not infinite) range of possibilities. Presumably, however, if the brain is damaged through an external event, or if a neuropathological process of some severity sets in, the range of functional possibilities becomes more restricted. The limiting condition, of course, is actual brain death, where the functional brain state as indicated either in the \( \psi \) or \( b \) frames is zero.

The most puzzling thing (as ontological dualists might agree) in explaining the relationship between mind and brain, is the nature and direction of causation. Speaking loosely, it is obvious that events at the mental level can bring about changes at the neurophysiological level; and vice versa. A clear example comes from the threshold of the mental hospital. Two persons are experiencing severe anxiety, complete with a range of physiological symptoms, such as palpitation, sweating, restlessness, etc. One spends an hour with a therapist, and emerges in a much calmer state. Undoubtedly biochemical changes have occurred; and yet the external cause was an interaction that was purely symbolic. The second person is given an injection of a tranquillising drug, and left to rest a while; and the results are rather similar to those in the first case. In the terms of already used in this paper, the proper way to represent these events is:

\[ \psi \equiv b_{\text{time 1}} \]
\[ \text{causal agent.} \]
\[ \psi \equiv b_{\text{time 2}} \]

Clearly it is a shorthand to say

\[ \psi_{\text{time 1}} \rightarrow b_{\text{time 2}} \]

for the first example, or
for the second, postulating purely mental causes in the former, and purely chemical
causes in the latter. If we choose to use the mental language, we can talk in a loose way
about mental causes, although the language is not such as to lead to the postulation of
causal laws. If we choose to use a natural-scientific language (from biochemistry,
neurophysiology, etc.), again we can speak of causes, and we might indeed find some
causal laws. However, it would be mistaken to suggest that, at any one point in time, a
mental event or state causes a neurophysiological event or state; we are simply dealing
with alternative descriptions of the same reality.

**Application to the psychiatry of old age**

Let us return now to the four components which, I have suggested, are necessary for
contemplating the mind-brain relationship for the purpose of psychiatry. We can
characterise what may be the case in ‘normal’ old-age psychology, in benign senescent
forgetfulness, in pseudodementia, and in senile dementia of the Alzheimer type.

First, then, let $\psi_a$ signify normal psychological functioning in old age. ‘Normal’
here is used in a fairly weak sense, implying that a person is able to go about the
business of life, can perform the necessary cognitive tasks, can relate well enough with
others, has an intact sense of self, etc.; there may also be certain interests, preoccupa-
tions and concerns that relate specifically to the predicament of old age. Accompany-
ing $\psi_a$ there is also a set of functional brain states typical of later life: $b_a$. Possibly the
neurotransmitter balance is different, from that of earlier stages, or the overall level of
activity is less, but the brain function is not impaired in any serious way. Structurally,
there has been only a relatively small amount of degeneration of grey matter ($P_1$) such
as is well within the range for well-preserved old people, and which brain function
can, so to speak, tolerate. The situation, then, may be represented as shown below:

$$\psi_a \equiv b_a$$
$$B^4, Bp_1.$$

We come now to the case of ‘benign senescent forgetfulness’, where the self is clearly
intact and mood shows no abnormality, but there are unusually large deficits of mem-
ory, especially short-term. This can be distinguished from the above in that now, it
seems, there are structural impairments in the brain tissue ($P_2$); corresponding to
‘benign senescent atrophy’:

$$\psi_a \equiv b_a$$
$$B^2, Bp_2.$$

The case of pseudodementia in old age stands in a marked contrast to this,
because here, undoubtedly, there are brain-functional changes that entail cognitive
deficiencies, and that special kind of ‘loss of self’ that represents the psychosis peculiar
to old age: in short, $\psi'_a \equiv b'_a.$
However, since pseudodementia is (by definition) reversible, it is not accompanied by appreciable structural damage to the grey matter. The condition, then, may be represented as shown below:

\[
\psi_a' \equiv b'_a
\]

\[
B_d, B_p_1
\]

Now we come to the crucial case, senile dementia of the Alzheimer type. Here there seems to be not only immediate functional impairment \((\psi'_a \equiv b'_a)\), but also some degree of neuropathic change. In the early stages of the illness the structural state of the brain still allows a range of functions – as is evidenced by the wide variability in cognition and behaviour over a short period in a single SDAT patient. The condition may be represented as shown below:

\[
\psi_a' \equiv b'_a
\]

\[
B_d, B_p_2
\]

Finally, in very severe dementia, where a person is seriously impaired in almost all function, having also lost virtually all of his or her everyday sense of selfhood, and in that sense has moved into a kind of deep psychosis \((\psi''_a)\), we have:

\[
\psi''_a \equiv b''_a
\]

\[
B_d, B_p_3
\]

When the illness has progressed to this degree, and only then, is it possible to suppose that something approximating to the widely accepted standard causal paradigm is in operation.

A scheme such as the above is only a first approximation, and no doubt needs a good deal of refinement. But its immediate usefulness is that it allows us to rationalise the field in a new way, one that has many implications for research and care-giving. Seeing Alzheimer’s disease not as a direct consequence of \(B_p\), but in the more complex terms of \(\psi, b, B_d\) and, \(B_p\), potentially explains one of the most notorious features of the condition – the wide range of presenting symptoms. Simply, there can be great variation in \(\psi \equiv b\), and in \(B_d\), while the brain has a common form of pathology: the degenerative changes associated with the neurofibrillary tangle, and so on. Further, we can make sense of an observation that is almost the converse: similar symptom patterns are sometimes observed in dementias that are found, post mortem, to be associated with different underlying pathologies of the grey matter, or with no pathology beyond the norm. Here there are, presumably, similarities in \(\psi \equiv b\), and in \(B_d\), strong enough to override variations in \(B_p\).

Earlier in this paper I mentioned four types of phenomenon that are difficult to explain in terms of the ‘standard paradigm’, and thus far only one – pseudodementia – has been dealt with in terms of my more elaborate scheme. Apparent precipitation can be accepted as a true (although of course partial) explanation in aetiological terms. For what we are dealing with here is, presumably, a fairly sudden deterioration in \(\psi \equiv b\),
the consequence of a registration, whether consciously or ‘organismically’, of difficult and stressful life events. The hypothesis would then be that there was, initially, a reaction of a mild pseudodementia type; but at that stage it was taken as sufficiently close to a normal reaction for it not to arouse great attention or concern. In other words, the early stages of SDAT might be explicable purely in neurochemical terms, with varying degrees of neuropathic change occurring slowly, possibly both before and after the critical events. There is some direct empirical support for this view, from studies that show neurotransmitter deficits in SDAT sufferers. However, the correlations with clinical indices (as also with neuropathology) are rather low. At this stage no clear conclusions can be drawn. 

Catastrophic decline might also be explained along similar lines. For it seems extremely unlikely, from what is known about the general progress of SDAT, that major degenerative changes of the Alzheimer type could occur in the grey matter within a mere few months. It is much more reasonable to suppose that such a rapid advance in a dementing condition occurs because of a deterioration at the psychological (and concomitantly, brain-functional) level; the dementing person feels betrayed or abandoned, perhaps; and an iterative ‘cycle of dementia’, involving progressive changes in neurochemistry, rapidly ensues. Transitory or partial ‘remenia’, too, does not have to be explained away. With those whose brain structure has not deteriorated too far, some function can be restored; there may be certain situations which are registered psychologically as highly comforting or pleasurable, which stimulate almost immediately a change both in the balance and degree of neurotransmitter activity. We do not know whether cells on the threshold of degenerative change can be returned to health, but there is no sound reason in biology for assuming that functional change is always the consequence of prior cellular pathology.

Conclusion

The scheme that I have presented in this paper is extremely bland, and it needs to be developed a good deal before its explanatory potential can be evaluated. Even at this stage, however, it does suggest a new way of ordering the data, both neurological and clinical, relating to SDAT, and it points to where there may be major lacunae in research. Also, and of more immediate relevance, it implies a way of ‘being with’ those who suffer from SDAT, a way which affirms their personhood even when their competences are very seriously impaired. In this closing section, then, I should like to draw attention to three implications from what I have been proposing.

First, studies of brain micro-structure have generally failed to take into account what may be of crucial importance in determining whether or not a person develops a dementing illness in later life. This is Bd, the developmental aspect. At present there is no non-invasive method available for examining Bd in human beings, although there is a vast literature pointing to its significance in experimental animals; here it is clear that experience is translated directly into interneuronal connections. However, what we can do in our own case is to deal with Bd indirectly, through that other descriptive frame, the psychological. The question then becomes whether certain kinds of psychological ‘strength’ might be sufficient to enable an individual to remain intact as a social and communicative being, despite the presence of pathological processes in the
brain. Because the correlations between dementia and pathology are rather low, this seems at least plausible. My own hypothesis is that one of the crucial factors is the extent to which the ‘experiential self’ has or has not been well-developed: that is, an integrated centre, grounded in feeling and emotion. For this can remain when the ‘adapted self’ (derived from role-performance and meeting others’ expectations) declines – as is very often the case for people in later life.

Second, there is a serious problem in relation to SDAT (but by no means unique to it) in that a case generally comes to the attention of health or social service professionals only when the person has gone a considerable way down the road of deterioration – often when his or her behaviour has become a nuisance or a threat of some kind. At this point the possibility of doing radical developmental work has long passed. According to the ‘standard paradigm’, the sufferer is midway in some ineluctable process, on which it is only possible to gaze with compassion and dismay, ‘managing’ it with kindness and skill. However, as with a range of illnesses, it seems highly likely that there is a period when the organism is in a metastable state, with the possibility either of passing back into health or forward into major pathology. Murphy and Brown studied this phenomenon, and noted that in their cases the onset of illness was often preceded by an affective disturbance such as depression. There is surely a lesson here for our understanding of SDAT. Perhaps a new priority in work with older people is to be sensitive to the possible presence of that metastable state, which would be related as much to disturbance of mood as to cognitive deficit. At this stage work on $\psi$ (hence b) is possible, and also perhaps on $B_d$, whatever are the tendencies in the area of $B_p$.

Third, the main emphasis in current psychological work related to SDAT is cognitive, reflecting to some extent a fashion that prevails in psychology as a whole. For example, much attention has been paid to charting the nature and development of deficits in information-processing; and in remedial work techniques such as Reality Orientation have been widely used, in the attempt to help a confused person retain his or her bearings in the everyday world. There is ground, however, for questioning this emphasis. We know from phenomena such as pseudodementia, and the small success in the area of rementia (whether through drugs or other means), that when the biochemical environment is modified, cognitive function may also be radically changed. When this is translated out of the b into the $\psi$ framework, it is tantamount to saying that to some extent cognition is dependent on the emotional ambience. Perhaps, then, more attention should be given to the nuances of the SDAT sufferer’s emotional state. Caregiving involves something far more skilled than attempting to adjust the dementing person to our (cognitive) reality; it involves the immensely subtle task of attuning ourselves to his or her (emotional) reality. Some of the drug trials give hopeful indications of what can be done through exogenous alteration of the brain’s biochemistry, though probably securing only temporary relief. However, if we accept the identity $\psi \equiv b$, an endogenous restoration is also perfectly conceivable. In the long run, it would be far more therapeutic to stimulate the body’s own neurochemicals, through means that are fundamentally psychological. At present we have only tiny and exceedingly transitory indications of how this might be effected. The task (as indeed with the great breakthroughs in technical innovation) is to explore all possible ways of turning such indications into clear and consistent signals. If there were success here, the
problem of SDAT would still be very far from being solved. But we might have found ways of keeping the sufferer in the world of persons, and mitigating a kind of psychological pain whose persistence and intensity we can scarcely envisage. The full solution can only come when we have dealt also with brain structure, in both its pathological and developmental aspects. Perhaps the fact that developmental change in later life has been generally neglected is itself a reflection of negative and empirically unjustified images of ‘normal’ old age. As these images change in society at large, so will the norms of caregiving; also, of course, of psychiatric practice.

Notes


18 Kitwood, T. in *Social Behaviour*, 1988, op. cit.