

Occasional review

“Depressive pseudodementia” or “Melancholic dementia”: a 19th century view

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SUMMARY Nineteenth century views on the interaction between dementia, depressive illness, general paralysis and brain localisation are discussed in the context of a book by A Mairet entitled: *Melancholic Dementia*. It is shown that by 1883 there was already awareness of the fact that severe affective disorder could lead to cognitive impairment. General paralysis was the commonest diagnosis put forward to account for patients with depression who went on to develop dementia. Patients so diagnosed, however, often recovered and clinical and statistical analysis of Mairet's case histories suggests that some were in fact suffering from depressive pseudodementia. Evidence is marshalled to show that during the 19th century there was wide disagreement concerning the clinical domain, course and even histopathology of general paralysis. This casts doubt on the traditional view that this condition served as “a paradigm” for other psychiatric diseases during this period. It is shown that by the turn of the century these difficulties led to a redefinition of the concept of dementia and to a marked narrowing of the clinical bounds of general paralysis.

A spate of recent publications attests to a growing interest in “Pseudodementia”.¹ Neither term, concept nor the issues they raise are, however, new.² Now, as it was during the 19th century, it is unclear whether the “dementia” of pseudodementia is just a bad behavioural copy, and hence only real in the eye of the untrained psychiatric beholder³ or represents, in fact, a genuine form of cognitive impairment.⁴ If the latter is the case then important questions emerge concerning the validity and reliability of the diagnosis of dementia.

Both dementia and pseudodementia received a fair amount of attention during the 19th century and themes such as reversibility, non-cognitive symptomatology and aetiology were often discussed. All three are well illustrated in *Démence Mélancolique*, an extraordinary monograph published by Albert Mairet in 1883.⁵

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Albert Mairet (1852-1935)

Mairet was Professor in Mental Illness in the University of Montpellier and Chief Psychiatrist to the Public Asylum of L'Herault. His doctoral dissertation on *Illusions and Visual Sensations as Causes of Illusions* (1876)⁶ was his first substantial work. Between then and 1928 Mairet published at least 14 monographs—mostly on psychiatric subjects and some co-authored, and over 20 articles. Special mention should be made of his books on heredity,^{7,8} on jealousy,⁹ on hysterical psychosis¹⁰ and on dementia praecox.¹¹ His originality and empirical approach produced work ahead of his time. Not surprisingly this failed to interest his contemporaries.

Mairet lived during a period of great books and great men. French psychiatry was mainly based in Paris where it was organised in schools and coteries and had reached full maturity.¹² The Great War ended this Golden Age. Guiraud, Delmas, Charpentier, Baruk, Courtois, Claude, Ey, Lacan and other French psychiatrists between the wars staged a recovery but the world of psychiatry had changed and new battles had to be fought. Men like Mairet (Magnan had died in 1916 and Chaslin in 1923) belonged to a different era. His death in 1935 was

not even recorded in the *Annales Medico Psychologiques*.

The Book

*De la démente mélancolique. Contribution à l'étude de la périencéphalite chronique localisée et à l'étude des localisations cérébrales d'ordre psychique*⁵ is 318 pages long and has two parts. The first contains 14 complete case reports; the second is subdivided into three sections dealing with symptom patterns, pathological findings and experimental neurosurgery. In this third section Mairé reported on the behavioural deficits shown by three dogs following unilateral experimental lesions to their temporal lobes. The second and third sections include another 44 case reports, many borrowed from the literature. References to Voisin, Ferrier, Munk, Magnan and Calmeil are made throughout.

Its historical relevance

The title of Mairé's book may deceive 20th century psychiatric historians but it cannot account for the scanty attention it elicited from its contemporaries. More likely explanations for this neglect are its inter-disciplinary nature, the novelty of its conception and the fact that it boldly brought together dementia, general paralysis, melancholia and brain localisation. The concept of dementia had, after major vicissitudes, achieved steady state by the 1880s. Reports of reversible dementia had been explained by postulating the existence of "vesanic" dementia, that is dementia caused by functional psychosis.^{13,14} The report by Mairé of cases of dementia which were reversible but showed brain changes challenged this compromise.

General Paralysis or Bayle's disease represented at the time, or many wanted it to represent, the highest expression of the "anatomoclinical view" in psychiatry.¹⁵ This useful myth has been kept alive by 20th century historians who wish to write off all psychiatry before Freud as prehistorical organicism.^{16,17} Mairé asked penetrating questions concerning the relationship between the specificity of the general paralysis lesion and psychopathology.

The diagnosis of melancholia had become, by the second half of the 19 century, an empty shell. Efforts to redefine this noble concept as a primary disorder of affectivity had not yet succeeded^{18,19} and the old ghosts of "affective monomania"²⁰ and "lypomania"²¹ had not yet been laid to rest. Cotard's view that the nihilistic statements of the melancholic represented a delusional disturbance reinforced the old view that melancholia was a disorder of intellect.²²⁻²⁴

Thus, historians who attribute the "origin" of the

modern (primarily affective) view of manic depressive illness to Baillarger or Falret²⁵ forget that as late as 1883 mania^{26,27} and melancholia²⁸⁻³⁰ were still considered by many as disorders of intellect. In the classical French tradition of Esquirol these two categories represented examples of total and partial forms of insanity, respectively.

It makes more historical sense to consider the contribution of Baillarger and Falret as more relevant to the burgeoning issues of the "combined insanities", that is to the view that two forms of insanity could be seen in the same individual.³¹ This new interest on the possibility that different psychoses could succeed each other in the same patient reflects a preference for longitudinal analysis instead of cross sectional phenomenology. This interest, however, was but the culmination of a process started earlier in the century and which led to the realisation that the temporal dimension was important to the definition of mental disease.^{32,33}

Hence it is anachronistic to regard Baillarger and Falret as the "discoverers" of a new form of psychosis for this word only acquired its current meaning in the work of Kraepelin. In fact doubts on the clinical discreteness of manic depressive insanity can even be detected in the latter's writing.¹⁹ As opposed to the predominant intellectualistic interpretation of depression Mairé made a determined attempt to define it as a primary disturbance of affect.

Brain localisation studies were popular enough during the 1880s but, in the main, were not concerned with affective functions.³⁴ For example, to the very end of the century Flechsig remained unconvinced of the possibility of localising the feelings.³⁵ Mairé, however, asked not only for the localisation of mood states but also of delusions and hallucinations. Similar work by Luys,³⁶ Burckhardt³⁷ and others on the localisation of complex psychiatric states was exceptional during this period.

Mairé's observation that melancholic patients were found post mortem to have changes in the temporal lobe led him to hypothesise that this area might be related to primary feelings of sadness and that the nihilistic delusions were in fact secondary developments made possible by the spread of the lesion to the cortex; it also led him to test his hypothesis experimentally. The search for the brain localisation of delusions is not exclusive to the 19th century as a recent publication shows.³⁸

It is a telling example of the fickleness of historical fame that an author who dealt so well with so many important issues did not make it to the Pantheon. It is perhaps fitting that his data be reanalysed on the occasion of the fiftieth anniversary of his death.

Table 1 Pseudodementia: Mairet's and a current series compared

	Mairet (1883)	Bulbena and Berrios (1985)	Statistics	Significance
No of patients	21	83		
Sex M/F	6/15	35/48	$\chi^2 = 0.790$	NS
Age	41.09 (SD \pm 10.3)	65.1 (SD \pm 12)	$t = 9.23$	$p < 0.01$
Depressed mood	13	53	$\chi^2 = 0.007$	NS
Hallucinations	14	17	$\chi^2 = 14.950$	$p < 0.01$
Delusions	13	31	$\chi^2 = 3.195$	NS
Neurological findings	10	25	$\chi^2 = 1.581$	NS
Duration <6/12	7	55	$\chi^2 = 6.243$	$p < 0.05$
Positive outcome	6	40	$\chi^2 = 1.880$	NS
Confusion	10	27	$\chi^2 = 0.598$	NS

(Kolmogorov-Smirnov Two Sample Test $K = 1.328$; NS)

Table 2 Phi coefficient matrix (Within Symptoms correlations)

	2	3	4	5
1 Depressive Aff.	-0.230	0.460*	0.0231	0.1189
2 Retardation		$\chi^2 = 4.462$	0.0301	-0.372
3 Delusions		-0.353	0.234	0.2850
4 Hallucinations				0.0373
5 Cognitive impairment				

*(Depression and delusions correlate to a significance of $p < 0.05$)

The Data

Mairet's sample has a mean age of 41 years (SD = 10.3) and is composed of 15 females and six males. Each report presents information on family and personal history, current illness, mental state on admission, course of the disease, necropsy results and author's comments. Although there is information on about 33 variables only the clearest descriptions have been chosen to avoid anachronistic interpretations (see table 1). A comparison of Mairet's melancholic dementias with a "reference" sample of pseudodementias recently reported¹ showed no significant overall difference ($p > 0.01$; Kolmogorov-Smirnov two sample test).

On the item by item comparison, three differences emerge: (1) an age difference (Mairet's cases were younger) explained by the fact that the "reference" sample was mainly collected from a psychogeriatric population; (2) a higher number of hallucinated patients in Mairet's sample probably resulting from a "contamination" by toxic-infectious states

superimposed upon melancholic stupors; (3) a longer stay of Mairet's cases probably reflecting the absence of efficient antidepressant treatment during this period.

Mairet's cases exhibited, in addition to delusions of guilt and of nihilism (Cotard's syndrome), suicidal behaviour, confusion, auditory hallucinations, disorientation and memory impairment. During the early stages the depressive symptoms were not accompanied by delirium or organic complications but some of the patients deteriorated due to lack of effective treatment. Some became retarded, refused food and ended up in states of stupor ("lypemia stupide").³⁹ They remained in this state for variable periods of time; many died from metabolic or infectious complications.

Both agonal state changes and the marked variation in the death-necropsy time interval make Mairet's pathological reports unreliable. In fact Meynert drew attention to this general problem in the Berlin Meeting on the Pathology of General Paralysis in 1883.⁴⁰

Analysis of symptom clusters (tables 2 and 3) suggests some correlation between depressive mood and delusions (Phi coefficient = 0.460) and between psychomotor retardation and remission within six months of onset (Phi coefficient = 0.447). Mairet's term "melancholic dementia" therefore seems to capture well the association between severe depression, cognitive impairment and recovery.

Three vignettes are included to illustrate his cases. Madame Dup (Case 1) was a 56-year-old married woman, with no family or personal history of

Table 3 Phi coefficient matrix (Symptoms and Illness Features Correlations)

	Depressive Mood	Psychomotor Retardation	Delusions	Hallucinations	Cognitive Impairment
Acute onset	-0.175	0.121	-0.193	0.137	0.084
Fluct. course	0.306	-0.166	0.141	-0.030	0.062
Duration <6	-0.171	0.447*	-0.316	0.269	-0.069
Improved	0.038	$\chi^2 = 4.2$ 0.066	0.141	-0.030	0.062

*Duration <6 months and psychomotor retardation correlate to a significance of <0.05 .

psychiatric illness. Her current disease started with hypochondriacal complaints followed by delusions of ruin, beliefs that her children were dead or about to be killed, and intense suicidal behaviour. On admission she was agitated, deluded, unable to sleep and refusing food. Her depressive symptoms remitted for a time but in the event became well established and she developed chronic melancholia, disorientation and "dementia". No necropsy report is included.

Madame R (Case 2) was a 44-year-old married woman with family history of psychiatric illness (mother was nervous, extravagant and irritable; maternal uncle was an alcoholic, said to have died of paralytic dementia). She had no personal history of psychiatric illness until age 40 when, after the death of her mother, she fell into atypical and protracted grief. Two months before admission she developed insomnia, became agitated, suicidal, felt persecuted and believed that people accused her of dishonesty. After admission she became highly excited, but remained paranoid and irritable. After this hypomanic episode she became haggard, depressed, apathetic, amnesic and confused. She had no neurological symptomatology. After some fluctuations and remission her illness became chronic. No necropsy report is included.

Madame Hug (Case 9) was a 46-year-old woman with a long history of nerves and one episode of psychotic depression 4 years earlier. A few weeks before admission she had become depressed, suicidal and attempted to kill her daughter. On admission she remained markedly depressed, retarded, hallucinated and cognitively impaired. Her illness followed a fluctuating course. She was discharged five years after admission in a state of "vast improvement".

Mairet on Dementia

Up to the first decade of the 19th century the term dementia was used to name both states of insanity accompanied by psychosocial incompetence and the related status of civil and legal incapacity.⁴¹⁻⁴³ Pinel⁴⁴ listed dementia as a separate form of insanity but did not distinguish it from mental retardation. Esquirol⁴¹ typified dementia as an acquired, irreversible and severe form of intellectual dilapidation (hence different from "idiotisme"), secondary to organic brain disorder and chronic insanity. Some of his criteria were challenged. For example, cases were reported who had recovered or who presented no positive postmortem findings.⁴⁵⁻⁵¹ Morel⁵² and others suggested that a "vesanic" form of dementia could complicate chronic insanity and that this fact could account both for the occasional recoveries⁵³ and for the absence of brain pathology.

The concept of "vesanic" dementia was in due course also challenged. This followed from the realisation that, to be of any clinical use, the concept of dementia needed definition in terms of psychological function. In the event memory became a popular candidate.⁵⁴ This soon led to the finding that the "defect state" of insanity affected memory in a different and far more superficial way than the organic (particularly senile) dementias. So new psychological characterisations of "vesanic" dementia began to emerge. Baillarger, for example, described it as a "chronic failure of the *association of ideas* in a person with an intact intelligence" (p 628).⁵⁵

Mairet, however, considered his "melancholic dementia" as organic in nature. He identified a clinical marker to differentiate it from the vesanic, defect, states: "the weakening of the intelligence occurs, as shown by the clinical facts, more or less soon after the onset of the condition. This short latency differentiates melancholic dementia from vesanic dementia" (p 138).

But Mairet also distinguished "melancholic dementia" from other organic dementias. It consisted in "a state of confusion of mind (*embrouillement de l'intelligence*) of which the patients were aware"; "they complain of inability to disentangle their thoughts (*debruiller leurs idées*)" and "their memory becomes impaired for recent events" (p 130).⁵ Mairet classified these cases as a separate psychopathological group: "according to our observations they correspond to states of so called anxious (*lypemanie anxieuse*) and stuporous depression (*lypemanie stupide*)". He also noticed the good prognosis: "when melancholic dementia follows a fluctuating course: remissions may be more or less complete and more or less long lasting" (p 136).⁵

Clinical analysis shows that a number of Mairet's cases, even those who followed a progressive course and died, suffered from severe psychotic depressive states which often terminated in stupor and its lethal complications. The fact that some of these patients became cognitively impaired, within a short period of time ("soon after the onset of their condition") and eventually improved suggests that they might have been cases of "depressive pseudodementia". Some had even personal and family history of affective disorder. Mairet, however, considered their pathology to represent a limited form of general paralysis.

19th Century Psychiatry and General Paralysis

The history of general paralysis has been told before.⁵⁶⁻⁵⁹ First described by ALJ Bayle as an "arachnitis chronique", it has been claimed that the disease was a "new phenomenon" resulting from "a

mutation in the syphilitic virus towards the end of the 18th century (p 623).⁵⁷

Historians who have put forward the view that the 19th century had a "Psychiatry without Psychology" (p 623)¹⁶ are fond of claiming that the discovery of general paralysis provided the alienists of the period with useful evidence in favour of the anatomical view of mental disease and "added weight to the prevailing theories that mental diseases are physical diseases of the nervous system" (p 399).¹⁶ They also lament that this "proved a step backward for the general psychopathology of the severe neuroses and psychoses, that is to say of mental diseases proper" (p 399).¹⁶ Non-clinical historians of psychiatry have gone further: "General paralysis became the model of a psychiatry conceived exclusively as a natural science". "during the second half of the century psychiatry felt able to deal with all psychopathological phenomena according to Bayle's model" (p 224).⁶⁰

These claims require modification. Historical analysis suggests that: (1) the concept of general paralysis was never stable; (2) it took the best part of 30 years to gain acceptance as a separate disease; (3) it drew the minds of the alienists more towards psychological and philosophical issues than towards anatomy: and, (4) it did not polarise French (or European psychiatrists) into brain-mind choices. In this respect Ackerknecht is right to state that: "the distinction between somatic and psychological schools of thought never developed in France in terms of an *a priori* opposition as it developed in Germany".⁶¹

Bayle's contribution, as Bercherie has perceptibly observed,⁶² was *not* anatomopathological, for even his contemporaries were aware of the incompleteness of his descriptions. Due to his youth Bayle's clinical experience and histological training were rudimentary. Like Jaspers, about a century later, this contribution was to the field of ideas. Bayle's insight was to suggest that a temporal framework be added to the concept of disease. Hence it is more helpful to interpret his "discovery" of general paralysis as a challenge to the "cross sectional" concept of medical diagnosis. Clinical entities he believed needed redefinition in terms of specific temporal patterns of symptoms.

The longitudinal view of disease required, according to the new view of disease developed during the early 19th century, a sort of "ontological backbone" that guaranteed its continuity in time. His belief in the "anatomopathological view" led Bayle to consider the "anatomical lesion" as the temporal bearer of the disease. Bercherie's puts it thus "for the first time in the history of psychiatry there was a morbid entity which presented itself as a sequential process

unfolding itself into successive clinical syndromes". . . . "in this diachronic process the nosological entities of Pinel and Esquirol constituted but episodes. . . ." (p 75).⁶²

This diachronic view of mental disease, often attributed to Kraepelin, is therefore a product of the early 19th century. The notion of "unitary psychosis" developed during the middle of the century but an offshot of it—and so were the later efforts by Kahlbaum and Magnan.⁵⁷ During the second half of the century, however, things became easier. The concept of functional lesions had replaced the earlier one of anatomical lesion.¹⁵ The idea of "degeneration", which was longitudinal in meaning, had become popular and provided a functional explanation for most forms of behavioural failure and decline, including dementia.⁶³ The earlier links between degeneration and anatomy became in fact tenuous by the end of the century.^{64 65}

Clinical Issues

The association between somatic and psychiatric symptoms that characterised general paralysis was explained in two ways during the 19th century. Everyone accepted that the illness was related to a "pericéphalite chronique diffuse" but no agreement existed as to how the brain lesion determined symptomatology.⁵⁶ Manic-ambitious, melancholic-hypochondriac and dementia with paralysis subtypes were recognised. The "unitary theory" stated that all three clinical forms constituted stages of a single disease with the order of their appearance depending upon the spreading of the cerebral lesions. This, which was Bayle's original view, found staunch supporters in Falret, Billod and Parchappe.

The "dualist" hypothesis, which Baillarger sponsored, stated on the other hand that "paralytic insanity and paralytic dementia were *different* conditions".

The issue at stake, however, had less to do with the nature of the brain lesions themselves than with the way they related to behaviour. The question was how is it possible to explain, in terms of the diffuse and generalised brain lesion, the "typical" content of the delusions and, indeed the "typical" demeanour of the paralytic patient? Baillarger believed that the chronic periencephalitis could only explain the motor disorder. For the psychological content of the delusions an account of a different order, a pathogenesis, was required: "the typicality of the delusions in paralytic insanity has no connection with the chronic periencephalitis. . . . for these delusions can also be found in subjects without brain lesion and hence must have different origin" (p 389).⁵⁶ The fact that the psychiatric symptoms had no real connection with the anatomical lesion,

Baillarger said, might also explain why patients may recover.

Since Fournier,⁶⁶ if not before,⁶⁷ the idea had been entertained that general paralysis might be related to syphilis. It is a point so far missed by psychiatric historians that, during the second half of the 19th century, there was resistance to accept that the identical clinical states exhibited by syphilis sufferers constituted, in fact, instances of Bayle's disease.⁶⁷ ⁶⁸ The creation of the special term "Pseudo General Paralysis" to name the general paralysis caused by syphilis illustrates this well.⁶⁹ Even a delusional (grandiose) and a dementia form of "Pseudo General Paralysis" were described.⁶⁹

If it is the case that 19th century alienists were totally organic in their approach, why did they not accept the syphilitic aetiology as a gift from the gods? To do so would have provided them with the best example of a Virchow-Koch type of disease. Explanations of this historical fact must start by demythologising Bayle's disease. First of all there is no clear historical evidence to show that 19th century alienists considered general paralysis as something special or as *the* paradigm-disease. They saw it rather as an example of a condition clinically characterised by a composite of both mental and physical symptoms, which maintained its identity in the midst of change.

The 19th century definition of general paralysis was very wide and series of cases reported during this period suggest that at least one in three subjects were suffering from functional or organic psychoses. Furthermore there was no accepted "typical" anatomopathology of general paralysis as the Berlin Debate of 1883 clearly showed.⁴⁰ The overinclusive nature of the diagnosis explains the polymorphous symptomatology attributed to general paralysis during this period and also the reported recoveries. For example, because of the belief that affective symptomatology (whether manic or depressive) was an early manifestation, patients with bipolar disorders were considered as suffering from general paralysis. In the event many ended up in stupor, dilapidation and cognitive impairment. (*vide* Case 1 in Baillarger series).⁶⁹ Some of Mairet's cases belong to this category.

Baillarger in an important paper in 1889 threw open the question of the clinical boundaries of general paralysis and showed that by the 1880s the original unitary view had started to disintegrate.⁶⁹ He concluded that: (1) Patients may exhibit all the symptoms of general paralysis without suffering from it; (2) Pseudo general paralysis frequently improves and, when it does not, it progresses onto a form of "dementia simple" without paralysis (pp 207–208).

Mairet's Views

It is against this background that Mairet's work must be understood. Following Baillarger's injunction he sought to research into the "Pathogenesis of delusions" (p VII). For this he chose to work on a syndrome "characterised by a mixture of depressive delusions and organic dementia" which at necropsy might show "a localised form of periencephalitis". His research strategy was to collect a homogeneous sample during the early stages of the disease. He did not seem to have wanted to "establish a new morbid entity" as a reviewer accused him of doing.⁷⁰

In melancholic dementia: "after a prodromal state of varied duration the depressive delusions appear suddenly and occupy the forefront of the condition, they are occasionally accompanied by signs of weakening of the intellect and of organic involvement of the brain; the somatic symptoms resulting from it tend to become chronic but may also improve" (p 119).⁵ Camuset⁷¹ misunderstood Mairet's intentions and insisted that "melancholic dementia" was not a new disease. He even contrived to publish in the same volume of the Journal a paper showing that about 45% of his 173 cases did in fact suffer from the depressive form of general paralysis.⁷¹ In this regard current figures concerning the prevalence of the depressive subtype of general paralysis oscillate between 8%⁵⁸ and 27%.⁷² Kraepelin estimated it to be about 7%.¹⁹

Camuset's criticisms were misdirected. Mairet's aim was to: (1) identify lesions which were sufficiently circumscribed to be related to the observed mood state; (2) to analyse melancholic delusions into their component parts; and, (3) to relate each to a different brain site. Baillarger seems to have been the only one at the time to grasp the significance of Mairet's work (p 376).⁵⁶

Mairet found, or believed he had found, (unfortunately he seems to have both examined the cases and carried out the necropsies) that patients with clear cut depressive delusions showed circumscribed lesions in the inferior aspect of the temporal lobe. He then tested his hypothesis by carrying out experimental excisions in dogs.

Mairet was not concerned with aetiological questions. On this score he stated, rather perfunctorily, that in the patients in question the lesions in the "lobe sphenoidal" might have arisen, by proximity, from infections in either ear or eyes or, alternatively, from general causes such as grief, overwork and overexcitement. He related the depressive delusions to certain areas: "we have come to give importance to the temporal regions as sites for depressive ideas (*idées de tristesse*), particularly the sphenoidal area" (p 207).

However, when it came to explaining the appear-

ance of cognitive impairment Mairet believed it resulted from a diffuse involvement of the cortex (p 196). His experimental work led him to state: "one can reject the view that the form of the delusion is directly related to the lesion". . . . "one should believe instead that lesions in the sites mentioned give rise to emotional changes in the direction of sadness (sentiment triste)" (p 257). With some precience he went on to suggest that, in case of melancholia, sadness must be considered as the primary defect out of which, in a later state, melancholic delusions and hallucinations may emerge.

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