

CHAPTER 1

Shared pathologies

German E. Berrios¹ and Ivana S. Marková²

¹*Emeritus Chair of the Epistemology of Psychiatry, Emeritus Consultant Neuropsychiatrist, Department of Psychiatry, University of Cambridge, Cambridge, UK*

²*Reader/Honorary Consultant in Psychiatry, Centre for Health and Population Sciences, Hull York Medical School, University of Hull, Hull, UK*

Definition

Until recently “Shared Pathologies” was the official DSM-IV-T [1] name for clinical phenomena having in common the fact that persons, through their socio-emotional relationships, may share mental symptoms or disorders similar in form and/or content. Such temporal concurrence has led clinicians to calling such complaints shared, communicated, transferred, or passed on. Although the A+B combination (*folie à deux*) is the commonest form of the disorder, this can also occur in families (*folie à famille*) or even larger social groups (schools or other institutions). This, together with the fact that the terms *shared* and *communicated* are (covertly) explanatory, has impeded the formulation of an adequate operational definition.

Both clinically and historically, *folie à deux* remains the core clinical phenomenon. Recently, in U.S. psychiatry, the category “297.3 Shared Psychotic Disorder (Folie à Deux)” [1] has been replaced by “298.8 (F28) 4. Delusional symptoms in partner of individual with delusional disorder” [2].

A similar concept appears in the blue (descriptive) World Health Organization (WHO) book [3]: “F24 Induced delusional disorder: A delusional disorder shared by two or more people with close emotional links. Only one of the people suffers from a genuine psychotic disorder; the delusions are induced in the other(s) and usually disappear when the people are separated. Includes: *folie à deux*; induced paranoid or psychotic disorder.”

And in the green (research criteria) WHO book [4]: “F24 Induced delusional disorder”:

(A) The individual(s) must develop a delusion or delusional system originally held by someone else with a disorder classified in F20—F23.

- (B) The people concerned must have an unusually close relationship with one another, and be relatively isolated from other people.
- (C) The individual(s) must not have held the belief in question before contact with the other person, and must not have suffered from any other disorder classified in F20—F23 in the past.

However, clinical experience suggests the existence of other presentations. For example, cases have also been reported of “contagious” obsessionality and hypochondriacal and suicidal behavior. Furthermore, if “communication” or “transfer” is to be considered as a definitional criterion, then phenomena such as the transfer of anesthesia or motor paralysis from one side of the body to the other (with the help of magnets) or indeed from one patient to another have to be included.

Lack of an adequate operational definition has precluded meaningful epidemiological research. It would be hasty, however, to conclude that the shared pathologies are clinical curiosities. Indeed, their peculiar multi-subject structure calls into question the individualistic metaphysics on which the definition of mental disorder is currently based, and challenges the plausibility of current neurobiological models of mental disorders (more on this below).

History

It is now about 150 years since folie à deux entered the nosological catalogue. Historians disagree on who reported it first. For example, Lazarus [5] states, “it was originally described by Lasègue and Falret” but Gralnick [6] and Cousin and Trémine [7] have shown that it all depends on how “locus classicus” is defined. The latter is a notion that can be characterized as resulting from the historical convergence of a name, a concept or mechanism, and a behavior [8]. Thus, if “contagion” [9] is considered as the concept involved in the convergence then Hoffbauer should be considered as the initiator; if “induction” were to be considered instead then it would be Lehmann. If the emphasis was to be on the behavior involved then the first to report the phenomenon would have to be Baillarger or Dagron. Finally, if the term *folie à deux* itself is to be used as a criterion then Lasègue & Falret should claim the accolade.

Deciding on priority has bedeviled the history of folie à deux since its inception. The official story goes that although some earlier alienists may have noticed folie à deux it was Lasègue and Falret who, in presenting a case to the *Société Médico Psychologique* in 1873, rounded it off as a new clinical phenomenon [6, 10]. Lasègue & Falret went on to publish the same paper in 1877 in two Journals: *Archives Générales de Médecine* [11] and *Annales Médico-Psychologiques* [12].

The historical reality is more complex. In his “Rectificatory note concerning the history of communicated insanity—folie à deux,” Régis [13] noticed that Lehmann had identified Baillarger as the “first” who had reported cases suffering from this disorder in 1857. Régis went on to confirm this claim and stated that in his “Quelques exemples de folie communiquée” [14] Baillarger had not only reported four cases but also provided the very diagnostic criteria that were to reappear in the work by Lasègue and Falret [11, 12]. In the debate that followed Arnaud [15] tried to redefine the *locus classicus* in favor of Lasègue and Falret: “the scientific era in the study of folie à deux only starts in 1873”; and Halberstadt agreed [9]. But what did Arnaud mean by “scientific era”? Why did he dismiss Baillarger’s report as “non-scientific”? It must be concluded that in Arnaud’s hands the term *scientific* was little more than a rhetorical device used to resolve an ongoing rivalry between two psychiatric coteries.

Soon enough a small industry developed around folie à deux. According to the phenomenology of the cases found and the transmission mechanisms proposed, four types were described: folie *imposée* (as described by Lasègue and Falret [11, 12]; folie *simultanée* (reported by Régis in his doctoral thesis of 1880) [16]; folie *communiquée* (reported by Marandon de Montyel in 1881) [17] and folie *induite* [18]. By the turn of the century, the main risk factors had also been listed: association, dominance, lack of blood relationship, premorbid-personality, gender, and type of delusion [9].

The concept of folie à deux crossed the English Channel swiftly. Savage wrote on it in the *Journal of Mental Science* [19], Tuke in the *British Medical Journal* [20] and in *Brain* [21], and Ireland [22] included a discussion in his book *The Blot upon the Brain*. By the end of the 19th century, all that could realistically be said on the subject had been summarized by Tuke [23]:

- (a) The influence of the insane upon the sane is very rare, except under certain conditions, which can be laid down with tolerable accuracy;
- (b) As an almost universal rule, those who become insane in consequence of association with the insane, are neurotic or somewhat feeble-minded;
- (c) More women become affected than men;
- (d) It is more likely that an insane person able to pass muster, as being in the possession of his intellect, should influence another in the direction of his delusion, than if he is outrageously insane. There must be some method in his madness;
- (e) The most common form which cases of communicated insanity assume is that of delusion, and specially delusion of persecution, or of being entitled to property of which they are defrauded by their enemies. Acute mania, profound melancholia, and dementia, are not likely to communicate themselves. If they exert a prejudicial effect, it is by the distress these conditions cause in the minds of near relatives;

- (f) A young person is more likely to adopt the delusion, of an old person than vice versa, specially if the latter be a relative with whom he or she has grown up from infancy;
- (g) It simplifies the comprehension of this affection, to start from the acknowledged influence which a sane person may exert upon another sane person. It is not a long road from this to the acceptance of a plausible delusion, impressed upon the hearer with all the force of connection and the vividness of a vital truth;
- (h) It is not easy to determine to what extent the person who is the second to become insane, affects in his turn the mental condition of the primary agent. Our own cases do not clearly point to this action, but there have been instances in which this has occurred, the result being that the first lunatic has modified his delusions in some measure, and the co-partnership, so to speak, in mental disorder, presents a more plausible aspect of the original delusion (Vol. 1, p. 241).

Current publications do little more than repeat what has been said in the classic texts.

Clinical phenomena

According to the received view, the clinical categories *folie à deux* and *folie communiquée* were first constructed in France by Lasègue & Falret [11, 12], and soon enough they surfaced in English as “communicated insanity” [22, 23] and in German as “induced insanity” [18]. However, equally important in Germany were the publications by Wollenberg on psychical infection [24] and the magnificent doctoral thesis by Max Schönfeldt on induced Insanity [25, 26]. Interestingly, in the German literature the term *induction* included the additional meaning that the psychosis seen in B (the “inducee”) might result from stress caused by living with A, a psychosis sufferer [27].

In the event, the French expression *folie à deux* was to predominate [6, 10, 27–36] and the disorder it names has since been reported in different cultures and clinical settings [37–41]. A number of explanatory mechanisms have been suggested [5, 7, 35]. For example, based on a review of 103 cases, Galnick [6] identified four sub-types: *folie imposée*, *simultanée*, *communiquée*, and *induite*. As we have seen above, this classification is little more than a medley of 19th-century French and German views on putative etiological mechanisms. In addition to *folie à deux*, clinical phenomena such as suicidal behavior (the Werther effect) [42, 43], hysterical symptoms [44], and obsessions [45, 46] should also be included in the group of shared pathologies. In this short chapter, there will only be space to deal with *folie à deux*.

So that the reader forms a concrete idea, a case of shared pathology from Tuke [21] is reproduced:

The father, William Cairn, admitted Feb. 26, 1886, was 70 years of age, a farmer, and believed himself to be pursued and persecuted by the whole House of Keys; that he was the owner of extensive property, out of which he had been kept by that House and the high bailiff. He asserted that mobs had been raised to destroy his houses and cut down his trees. He had, he said, been assaulted by the men who had robbed him, with crowbars and pickaxes; when he endeavoured to obtain redress of these grievances, he had been prevented by telegrams and ghosts. His wife, ten years younger, asserted that her property had been sold against her will; that she had telegrams from invisible wires to say she must hang herself in consequence; and that her neighbours had put blood on the door and over the house. The daughter of these people, admitted on the same day, was 26 years of age; was silent and morose, with the exception of saying "first-rate" to enquiries about her health. Her mind, in fact, was too demented to allow of her entertaining the delusions of her parents. How long she had been affected is not stated, but Dr. Richardson informs me that she had returned home from service some time previously, and he is of opinion that the insane ways of her parents had much to do with inducing her present condition of mind. As to the man and his wife, the first symptoms arose about sixteen years ago after the loss of a little farm. They began to think they were entitled to property of great value, and eight years ago they went to London to Somerset House, to establish their claim, and have, their relatives say, spend "many a bright pound" in their search after the imaginary wealth (p. 413).

Epistemology

To understand why after the 1850s alienists thought it possible for insanity to be "communicated," two themes need exploring: (1) changes affecting the concept of insanity, and (2) theories and mechanisms of human communication (e.g., mimesis, imitation, contagion, infection, sympathy, etc.) [9].

Concept of insanity and the individualistic metaphysics of disease

Since the 18th century, "medical nosography" (that is, the description of disease) has been based on John Locke's notion of Individualism [47]. Like property, human rights, thoughts and selves, disease was also considered as an exclusive "personal" event. Reaffirmed as a unit of analysis, the individual and his skin became the absolute, natural boundary. By the same token society was modeled upon the Newtonian atomic paradigm and conceived as a mere collection of atoms (individuals). To get the latter to communication a theory of interaction was needed and Locke's solution was associationism, an epistemological (and later psychological) theory that set the rules as to how

information might pass from one individual to the other. Because disease was a very personal event, its transmission from one person to the other needed explanation. To this effect mechanisms such as epidemics, contagion, mimesis, imitation, sympathy, empathy, and others were put forward. Constructed during the 19th century, alienism (now called psychiatry) fully adopted this individualistic notion of madness (disease).

Within this epistemological framework, the idea that madness could be “shared” or “passed on” was in principle unintelligible. This is why when clinical observation suggested that such sharing did actually occur, alienists had to resort to metaphors borrowed from physics and biology. It is in this sense that they claimed that mental disorders could be communicated, induced, caught (via contagion), and so on. In other words, a person A (the inducer) could pass on his madness to person B (the induced). The flow and direction of transmission between A and B was made in terms of features attributed to each that in practice reflected the social prejudices of the time. For example, A was said to be strong, male, superior, older, and the like, whereas B was claimed to be weak, a female, neurotic, dependent, younger, and so on. In general, metaphors taken from physics and mechanics were preferred to social accounts already available at the time such as empathy, sympathy, and imitation.

However, the idea that diseases (like selves) [48] may be shared by groups becomes less unintelligible if: (1) ontological individualism is set aside, and (2) diseases themselves are not fully reduced to their organic substratum. In this sense, the latter become processes or concatenations of events that as such can exist in multiple times and spaces (e.g., a cluster of persons) [49]. In the long term, whether this latter view is to become popular will depend more upon its usefulness in managing disease than upon some theoretical need to preserve the individualistic ontology of disease.

Theories and mechanisms of human communication

The central meaning of “to share” is “to cut into parts” [50], hence *oratio recta*, “shared pathology,” should mean that parts of one symptom or disorder are being given out to different individuals. In practice, however, shared pathology means that although A + B exhibit similar symptoms or disorders, A has developed them first and passed them onto B. For completion’s sake, it could be said that the clinical phenomenon is open to three theoretical interpretations: (1) one disorder is apportioned in shares to A + B; (2) one disorder is passed on (transmitted, communicated, etc.) from A to B; or (3) A + B show the same disorder but this fact is aleatory.

As currently defined, shared pathology refers to the second interpretation, that is, the situation or process whereby a disorder moves from one individual to the other. This situation, in turn, is open to three interpretations: (1) A *passes* it onto B (either intentionally or not); (2) B *imitates* or copies A,

regardless of A's views or actions; or (3) a hidden (third) agent occasions the disorder to pass on from A to B.

Interestingly enough, all three options were discussed during the 19th century:

- 1 A → B concerned the old view that certain behaviors could be imposed or induced onto others (regardless of their will); indeed, this possibility was also reflected in the popular 19th-century pedagogical philosophy that supported the view that a teacher was able to shape the behavior of a pupil regardless of the latter's conscious wishes.
- 2 B could also "imitate": A; indeed, by the end of the century imitation had been proposed as a general mechanism of socialization, for example by Tarde [51]. In this regard, notions such as sympathy, empathy, imitation, mimesis, emulation, and the like were included in the process.
- 3 This mechanism concerned the old medical notion of contagion (i.e., the view that miasmas, spirits, microbes, and so on could facilitate the transfer of a disorder from A to B) [52]. The fact that no such agents were known to exist in regards to mental disorder led 19th century alienists to talk about psychological or moral contagion. Indeed this was a common explanation for suicide epidemics, folie à deux, addiction to opium, and other conditions. It is also possible to include under this rubric clinical situations where the transmission of symptoms from A to B is effected by another person. For example, in experiments using a magnet carried out by Babinski, symptoms such as paralysis and anesthesia were moved either from one to the other side of A or from A to B [44]. Conceptually, these cases seem to belong in the shared pathologies category even if ordinarily they are not included.

Prosper lucas and his 19th-century classification

The analysis of how certain mental disorders can be transmitted from A to B proposed by Prosper Lucas (1808–1885) remains unsurpassed to this day. In his doctoral thesis, "*De la Imitation Contagieuse. Ou de la propagation sympathique des névroses et des monomanies*" [53], he proposed a three-fold etiological classification:

- 1 Phenomena resulting from voluntary mimicking
 - Physiological
 - Pathological
- 2 Phenomena resulting from involuntary imitation (sympathetic)
 - Physiological
 - Pathological
 - Neurosis of movement or sensation
 - Neuroses of mental faculties
 - Complex neuroses
- 3 Phenomena that start as voluntary mimicking and become involuntary

Lucas's model is based on the combination of two polar dimensions: voluntary versus involuntary and physiological versus pathological. At the beginning of the 19th century, Bichat had explained the first dimension on the basis of differences in muscle type and innervation. This distinction was soon transferred to the mind and "voluntary" and "involuntary" thoughts started to be differentiated. The dimension physiological-pathological developed in the wake of the construction of the discipline of physiology itself, based on the distinction between structure and function. Like structure, function could also range from the "normal" to the "pathological."

Lucas introduced two additional concepts: imitation and sympathy. At the time, medical science still conceptualized imitation as a faculty of the mind (something similar to what is happening now in regard to the function of mirror neurons). Sympathy, in turn, was defined as a functional interdependence between the organs of the body, and by extension, between separate individuals [54]. Among the pathological phenomena Lucas included a variety of "neuroses," which he still defined in William Cullen's sense (imported into French medicine by means of the translations of Pinel and Bosquillon) [55]. There is no space in this chapter to analyze Lucas's work in more detail.

Conceptual mechanisms

The names used in the past to refer to the various shared pathologies reported in the literature reflect not only descriptive but also explanatory biases. Terms such as *induced* or *communicated* seem to be referring to hypothetical mechanisms that the authors very rarely make clear. Other concepts such as imitation, empathy, sympathy, and transfer are also mentioned in this context and some have interesting conceptual histories. Only imitation will be briefly explored in this chapter.

Imitation

Together with mimicry and mimesis, imitation constitutes a family of notions that refer to the copying the behavior (overt and subjective) of others. This action can be conscious or unconscious and its motivation varies from mere jest to admiring emulation. Known since classical times, these three notions have been put to a variety of uses. For example, mimesis plays a central role in the theories of art and the representation of nature proposed by Plato and Aristotle [56, 57].

Likewise, since early in Christianity, imitation (of God and Christ) became a principle of ethical behavior and a religious path toward the acquisition of grace. It can be found at the very basis of the concept of *theosis* or deification, that is, of the process whereby by his actions man emulates God [58]. On account of its importance, imitation is discussed by all the fathers of the

Church (from St. Augustine on) culminating in “Imitation of Christ,” a classical work by Thomas à Kempis where it is enjoined that the mere “copying” of Christ (that is, of his holy behavior and preaching) should be replaced by an emulation of his interior life and withdrawal from the world [59, 60]. Luther disliked the concept of imitation and sought to replace it by passive conformism with the divine rule [61]. During the 19th century imitation returned as a pedagogic device, as something that children should do in order to become socialized and educated [61]. By the end of this period Tarde [51] proposed imitation as the central element in social development and cohesion and used it to explain all manner of social processes such as fashion, acculturation, national feelings, patriotism, and so on. Imitation has therefore been variously conceptualized. Originally considered as a power or capacity, by the 19th century it had become an instinct, something that animals and human beings did naturally. A difference was also introduced between mimicry, imitation, and mimesis and it was claimed that mimesis was an exclusively human function [62, 63, 64].

Until the end of the 20th century, writers conceived of imitation as a function of the mind. This changed when in the 1980s, neuronal clusters were reported that seemed to fire in response to imitative behavior [65]. Much debate has since been had on whether mirror neurons constitute the imitative brain engine par excellence or whether they fall short from explaining social imitation. All told, it remains unclear whether mirror neurons can discharge all the explanatory responsibilities that have been attributed to them [66]. For example, attributing to neurons full functional autonomy and agency leads to the obvious danger of a regression *ad infinitum*, that is, of the need to postulate another controlling neuronal cluster, and so on.

Part of this problem relates to the ambiguous use of the term *mirror*: Mirrors reflect passively images flashed onto them but do not start any imitative activity. By a semantic sleight of hand, mirror neurons are now used to explain imitation, that is, to start imitational behavior on the part of agent [67]. In this sense, they are no different from the old accounts that used psychological powers or faculties of the imitating agent such as sympathy, emulation, and so on.

Conclusion

The clinical phenomenon now known as shared pathologies has been well known and discussed since the 19th century. Indeed, discussion in the 20th century (and later) has contributed little to its explanation. Difficulties with providing an adequate operational definition explained the limited epidemiological information available on its incidence, prevalence, and cultural distribution. This notwithstanding, the shared pathologies are interesting both

clinically and conceptually, the former because they draw the attention of the clinician toward the family setting in which mental disorders occur. There is a tendency these days to neglect such context due to the overemphasis on the individualism of disease encouraged by the neurobiological model. The latter is of interest because a proper analysis of the shared pathologies will make the clinician call into question some cherished assumptions as to the individualistic nature of mental disorder and will encourage her to explore new concepts and ways of explaining the complaints expressed by sufferer.

How then can the shared pathologies be explained? The old concept of imitation could be a useful start. It might be said that for a variety of reasons human beings copy (whether consciously or not) the behaviors (including the symptoms) of relevant others. Imitation could be driven by solidarity, a wish to identify with others, and so on. Exploration of these psychological and social mechanisms has not yet been exhausted and should be pursued, not by reducing it to mechanistic neurobiological language (such as mirror neurons) but by keeping the discussion within the semantic space, the space of meaning where much of the drama of mental illness occurs.

One of the interesting issues arising from the existence of these phenomena concerns questions around how humans form and maintain their mental symptoms and disorders. On this nothing has been so far said in this chapter but it is, as far as we are concerned, the most promising option to understand the shared pathologies. For, irrespective of the underlying mechanisms that the clinician may want to postulate for, say, the psychosis in A, it is unlikely that it can be explained to account for B's.

According to the Cambridge model of symptom-formation, this process starts when new information enters awareness and causes emotional distress, encouraging the sufferer to want to share it with an interlocutor. The information can be of biological origin (generated by a neural network in distress) or symbolic in nature (originating in a social interaction). Like all other material entering awareness, this information is in an inchoate state, that is, pre-conceptual and pre-linguistic (ineffable). Because it is often novel it cannot manage in the usual way (like, say perceptions) by means of conventional templates that are applied to it in a habitual, non-conscious manner. Upon becoming aware of the novel information, the sufferer is forced to choose a configurator (from his or her bank of personal, familial, social, and cultural templates). Once configured into a speech act, the information is communicable and can be passed onto an interlocutor with whom a further negotiation can take place. A crystalized and recordable (in the case notes) mental symptom emerges at the end of this process [68].

This could explain how a shared pathology takes place. One option is that in A, the symptoms are formed as configurations of a biological signal. A and

B have a social relationship and share an intersubjective space. In the regular exchanges that follow, symbolic material is transferred into the awareness of B, who must handle it accordingly. In the ordinary state of affairs, B will configure it as what it is: a worry about A. On rare occasions, however, B may choose a configurator that expresses sympathy, identification, or imitation with A and this could lead to B's introjecting the symbol and configuring it as a mental symptom that would then be similar to that of A. The point here is simply to explicate how mental symptoms that may have the similar phenomenological presentation are the result of different configuratory mechanisms and hence have different etiology. Interestingly enough, one of the 19th-century explanatory models of shared pathologies lists as a cause of disorder in B the stress caused by living with A [27]. An explanation of this nature should carry important implications not just for understanding mental symptoms but also for approaches taken to their research and to their clinical management [69, 70].

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