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DELIRIOUS STATES*

BY

BERNARD HART, M.D., F.R.C.P.

PHYSICIAN IN PSYCHOLOGICAL MEDICINE, UNIVERSITY COLLEGE HOSPITAL, AND NATIONAL HOSPITAL, QUEEN SQUARE

The word "delirium" is derived from *de*, meaning "out of," and *lira*, the ridge between two furrows on ploughed land, and its original signification was therefore "off the track." Metaphorically it was used to denote any form of mental aberration, and this wide meaning of the term has to some extent persisted to the present day. Notably in French *délire* is used in a far more general sense than in English, and is applied not only to delirious processes in the English sense, but to delusions and obsessions. In the present paper the word is used in its narrower meaning, though even here, as we shall see, its signification is by no means so precise and limited as is sometimes supposed.

Numerous problems worthy of discussion arise in connexion with the consideration of delirious states. Do these states belong to a precise and definable syndrome, with a specific causation and mechanism? Do the deliria resulting from different causal agents present specific features, so that the nature of the agent can be inferred with certainty from the clinical picture? What part do constitutional and psychogenic factors respectively play in the causation of deliria? Does the study of delirious states enable conclusions to be drawn concerning the origin and mode of production of psychotic symptoms in general? Further, there are practical problems of diagnosis, prognosis, and treatment. Naturally it will only be possible here to touch upon the fringe of this great field, and many interesting and important topics must be omitted altogether.

Definition

The term "delirium," when applied to specially selected and characteristic examples such as febrile delirium, denotes a familiar syndrome, apparently composed of precise and easily recognizable features. Clinical observation, however, has made us acquainted with a series of allied states, ranging by imperceptible gradations from the typical febrile delirium to clinical pictures of a very different nature, and the existence of this graded series makes it difficult to mark out any precisely defined syndrome to which the term "delirium" may be properly and exclusively applied. This difficulty is apparent when we seek, in text-books and elsewhere, for a satisfactory definition.

Bleuler¹, after explicitly recognizing that delirium, though a concept everywhere used, is differently defined by different authors, and is not clearly differentiated or definitely limited, remarks that "what one calls *deliria* are mostly states of incoherent thinking combined with hallucinations and delusions, which show a certain activity and run a rapid course." Henderson and Gillespie² define delirium as "a symptom-complex consisting essentially in

disorientation and hallucination, and very commonly having an accompanying affective disturbance, especially of fear." Levin³ says that "empirically, delirium may be defined as a condition in which the patient is restless, lacking in alertness, disorientated and hallucinated." Wolff and Curran⁴ equate delirium in a broad sense with Adolf Meyer's "dysergastic reaction," a term proposed by Meyer for the disturbances "due to disorders of the nutrition and circulatory support of the brain (the effects of poison or abnormal nutrition, anaemia, hyperaemia, and oedema)." Curran⁵ divides organic mental disorders into those which show an irreversible alteration in brain tissue, and those which do not, and he employs the term delirium to designate a group lying within this second class, a group "not perhaps strongly defined," but characterized, "amongst other things, by considerable confusion and disorientation."

It has to be confessed that these definitions are vague, and they certainly fail to mark out any syndrome characterized by precise and sharply limited features. We may conclude, indeed, that no such precisely marked syndrome exists, although it may nevertheless be possible to show that certain phenomena tend to occur in association with one another even in very diverse settings, and that from this constancy of collocation certain inferences may be drawn.

Range of Delirious States

It has been remarked above that, while febrile delirium forms an easily recognizable and characteristic syndrome, a series of states can be found which range by imperceptible gradations from this typical picture to conditions with apparently remote relations to febrile delirium. Tracing out this series of states, we have in the first place the disorders to which the term delirium is unhesitatingly and universally applied, the deliria resulting from the abuse of drugs, notably alcohol. Delirium tremens, with its confusion, disorientation, hallucinations, associated fear affect, and short course, conforms in all respects to a typical delirium, but it is to be noted that we find a range of cases proceeding from what may be called an attenuated delirium tremens, with less severe symptoms and a longer course, through the conditions which have been termed alcoholic hallucinosis, to chronic hallucinatory psychoses in which some of the characteristic features of delirium such as confusion are almost entirely lacking. Chloral hydrate is capable of producing a disorder symptomatically identical with delirium tremens, and numerous drugs—bromides, cocaine, and many others—may be responsible for closely similar clinical pictures.

Next we have those conditions in which an endogenous toxic process may reasonably be presumed to be operative. Febrile delirium of course itself belongs here, and in addition

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typical delirious states may arise in illnesses which may or may not be accompanied by fever, such as uraemia. Here also should be mentioned the deliria which may precede the outbreak of an infectious disease or occur during its post-febrile period, and perhaps post-operative deliria and those sometimes manifested in states of extreme inanition. In the next group may be comprised those cases in which the assumption of a toxic origin is based mainly on analogy, such as the deliria occurring in senile psychoses, general paralysis, and in traumatic conditions, though, as we shall see when considering the theories put forward by Hughlings Jackson, the assumption may be erroneous.

We then have to deal with a series of cases which, while certainly not conforming to the typical picture of a delirium, present obvious similarities thereto, and include in their symptomatology the confusion, restlessness, and hallucinatory tendency which seem to be the essential features of delirium. It will not be amiss, indeed, to regard these states as attenuated deliria. Here belongs the acute confusional insanity of English psychiatrists, a clinical entity originally described by Meynert under the name of amentia, and then by Chaslin under the name of primary mental confusion. This condition is characterized by confusion with imperception and disorientation, hallucinations of all the senses, motor restlessness, a duration of several months, and a fairly good prognosis. The apparent causation of these cases is particularly interesting because, while on the one hand the disorder may follow definite toxic states either exogenous or endogenous, it may also occur as a sequel to excessive fatigue, and emotional stress. It is a plausible inference to suppose that the link is by way of chemical processes associated with the fatigue or emotional state.

Next, certain considerations may be mentioned which indicate clearly the difficulties encountered in regarding delirium as a definite syndrome with specific characters, relationships, and causation. The first is the circumstance that typical deliria may occur in the course of other disorders such as schizophrenia, and that these manifestations shade imperceptibly into symptom-complexes characteristic of those other disorders but not of deliria. The second is that deliria may occur in psychogenic disorders such as hysteria. These might be regarded as resulting from the associated emotional states in the manner described above, but we are met with the further difficulty that such deliria shade into the so-called "twilight states," such as hysterical somnambulisms, in which elaborate and coherent thought-processes and behaviour occur, without obvious affect, and without anything that can reasonably be called confusion.

Finally it has to be noted that the clouding of consciousness to which the term confusion is applied may in deliria show varying grades of depth. There may simply be a mild impairment of comprehension, and some interference with the co-ordination and elaboration of impressions. If the disturbance deepens, a state develops in which hallucinations and delusions may easily be formed. A further deepening leads to a condition of somnolence and ultimately to coma. The consideration of this type of grading points to a significant relationship between the confusional phenomena of deliria and the mental torpor observed in conditions of increased intracranial pressure such as occur with cerebral tumours.

How Far is "Delirium" a Unitary Syndrome?

We are now in a position to consider how much unity the syndrome "delirium" possesses, and how far essential features can be picked out therein which, when found similarly collocated in diverse settings, enable definite inferences to be drawn concerning their causation and pathogenesis.

Curran¹ in a study of 103 cases, of very diverse origin, endeavoured to determine, amongst other problems, the characteristics which delirious states appeared to possess in common. He found that confusion (difficulty in grasp, and clouding of consciousness), and restlessness (ranging from highly co-ordinated and apparently purposive movements down to simple movements barely distinguishable from tremor), were universally present. In addition the patients responded with abnormal facility and intensity to stimuli arising from without and within, unless the disorder was so profound as to border on coma. Visual hallucinations were very frequent, as were also delusions or misinterpretations of paranoid type. Curran holds that "confusion," if the term is correctly used and not loosely applied to any case which does not respond briskly and appositely, such as retarded depressives or patients showing schizophrenic perplexities, is only found in definitely organic reactions.

Henderson and Gillespie² adopt a similar standpoint and include deliria in the acute variety of "organic-reaction types," usually ascribable to a temporary toxic process affecting the brain substance, as opposed to the chronic organic-reaction types, in which there is a more severe tissue change and usually a progressive degeneration. So far as I am aware, these authors do not specifically state the meaning which they ascribe to the term "organic," but they would appear to apply it to conditions in which either an actual alteration of brain substance or the presence of a toxic or metabolic agent acting on the brain substance is known or inferred to exist. Régis and Hesnard³ uncompromisingly assert that mental confusion is the "psychose-type" of the exo- and auto-intoxications "*partout et toujours la même sous ses diversités symptomatiques.*"

A Problem in Three Parts

Consideration of the views expressed by these various authors indicates that the problem with which we are here concerned falls into three parts. Is there an "organic-reaction" which can be discerned and differentiated in whatever setting it may appear? Is there within this "organic-reaction" a "delirium" syndrome which can be sharply marked off? And thirdly, if this second question is answered in the negative, are there at least in delirious states features tending to occur together with a constancy which enables inferences to be drawn concerning their nature and causation?

It is generally agreed that to the first question an affirmative answer can be given, though only within certain limits. The organic-reaction is usually described as manifesting itself in processes of two kinds, which may be combined in various ways. On the one hand there are deprivation phenomena due to loss of function or loss of control, such as appear characteristically in the organic dementias. On the other hand there are confusional states, often hallucinatory, such as are exemplified in the deliria. Clinical experience amply confirms this formulation so far as a large range of cases is concerned, and typical instances presenting the features described can be immediately recognized and differentiated without difficulty. It has been seen, however, that as we move away from these typical instances we find a graded series of cases which ultimately takes us to schizophrenic and other pictures with but little obvious relationship to characteristic organic conditions. We cannot therefore say that an "organic-reaction" can be discerned and differentiated in whatever setting it may appear, but only within a certain range of cases where the features composing it are pronounced. If those constituent features, loss of function, loss of control, and confusion, could be sufficiently precisely defined, it might be possible to observe and differentiate them throughout the whole series of graded cases to which

reference has been made, and inferences of great value might then become possible. Unfortunately none of these features as yet permit of such precise definition, and therefore the step forward cannot at present be made with any certainty.

To the second question, whether within this "organic-reaction" a syndrome "delirium" can be exactly marked off, the answer must obviously be negative. It is clear that here also, while typical deliria form easily recognizable pictures, they shade off by imperceptible steps to pictures of an entirely different kind.

In attempting to answer the third question, whether in delirious states specific features occur together with a constancy permitting inferences to be drawn concerning nature and causation, we are met with the difficulty already mentioned with regard to "organic-reaction" in general, the difficulty of precisely defining those specific features. We may accept Curran's statement that restlessness and confusion are universally present, but the former obviously admits of no clear-cut definition, and it is very questionable whether this is not equally true of the latter. Curran's equation of "confusion" with "difficulty in grasp and clouding of consciousness" certainly describes a phenomenon familiar and recognizable in its typical forms, but not one with exactly definable properties and limits. Régis and Hesnard's claim that confusion is a specific character of toxic states, constituting a picture everywhere and always the same in diverse settings, is surely unduly optimistic.

In these replies we are really recording a difficulty which constantly besets progress in psychiatry. Manic-depressive psychosis, schizophrenia, and paranoia are clinical entities which in their typical ranges present characteristic and immediately recognizable pictures, but they shade off into one another and into other psychoses. The concept of reaction-types in place of the older concept of disease-entities gives us a better understanding of these transitions, but it will not take us very far unless we are able to describe and differentiate those reaction-types in precisely definable terms. Until that time arrives, diagnosis and prognosis must necessarily depend upon clinical experience and intuition rather than on deduction from established principles. The deliria, however, do provide a group of cases from the study of which there is a good prospect of opening up an avenue leading in the desired direction, because we already know at any rate some of the causal factors responsible for the deliria. If the phenomena ascribed to those causal factors can be precisely formulated, and a specific connexion established between cause and effect, so that this specific connexion can be traced out throughout the graded series of allied states, we should be able to penetrate far into the general problem of the aetiology of psychoses.

Causation of Deliria

If we confine our attention to typical deliria it can confidently be stated that in the great majority of cases a "toxic" origin can be demonstrated or reasonably inferred. This is at once evident in the deliria arising from the abuse of various drugs, and in delirium tremens, though in all these cases we do not know whether the causal agent is the drug itself or some substance of the nature of an antibody generated by the organism. It is also a reasonable inference that the deliria of acute specific diseases result from a "toxin" produced by the primary disease, although again we have no knowledge of its precise nature or mode of manufacture. Further it is a plausible assumption that the deliria occurring in senile psychoses, general paralysis, and cerebral traumatism may be mediated by toxic processes, though here the modification of brain substance directly responsible for the delirium may be produced not

by a circulating toxin but by the immediate action of the disease processes or the trauma.

If we next turn to those conditions standing nearest along the scale of allied states, the cases included in the group termed "acute confusional insanity," we find them associated with preceding factors of two kinds. In the first place there may be factors of an obvious exo- or endogenous toxic type, such as externally introduced poisons, or puerperal conditions. In the second place there may be factors such as states of excessive fatigue or emotional stress, and in both it is a reasonable inference that the delirium is due to chemical substances generated in association with those states.

Finally, it would be a plausible assumption that, where definite episodic deliria occur in the course of other psychoses, such as schizophrenia, their causation is also to be ascribed to a transitory toxic process, and this might apply also to hysterical deliria. But the assumption becomes more and more strained when we remember that the deliria of schizophrenia shade imperceptibly into schizophrenic symptom-complexes of a very different kind, and that hysterical deliria shade into somnambulisms and other phenomena occurring in settings in which a toxic origin seems remotely improbable.

In this connexion it is necessary to realize that, although in certain typical deliria a toxic agent undoubtedly plays a causal part, we have no knowledge as to its precise mode of action or the links by which it leads to that final modification of brain substance which may be presumed to be the immediate cause of delirium. There is no reason why this final modification should not be equally producible by agents of a quite different nature. For example, the connexion between delirium and toxic processes may be no more specific than that existing between hemiplegia and any one of the causal agents primarily responsible for it such as cerebral haemorrhage, thrombosis, or trauma, all of which are very different from one another but similar in their capacity for producing the destruction of brain substance which immediately results in hemiplegia.

Our knowledge of the causation of deliria is indeed limited to the statement that in a majority of cases deliria are preceded by factors bearing a general resemblance to one another and therefore included under the somewhat indefinite term "toxic." Of the precise processes by which delirium is mediated we know nothing. In discussing at the present time the possible pathogenesis of delirium we have therefore to leave the sphere of knowledge and enter that of hypothesis and speculation.

Theories of Pathogenesis

In considering the character and genesis of the modification of brain substance presumed to be responsible for delirium an important place must be given to the theories put forward by Hughlings Jackson. The application of these theories to the special problem of delirium has been reiterated and amplified in a recent paper by Levin.³

Jackson's views with regard to the structure and mode of functioning of the nervous system are too well known to need description here. The essential points bearing upon our present purpose are as follows. The nervous system is a sensorimotor mechanism composed of a hierarchy of "centres" or "layers," the higher and more recently evolved centres being not only more complex and specialized, but also serving to control or inhibit the activities of the lower centres which, being older in the scale of evolution, are more firmly organized and automatic. A centre can be affected by lesions of two kinds: "destroying" lesions, whereby the functioning of the centre is diminished or abrogated; and "discharging" lesions, whereby the function of the centre is stimulated. If a higher centre is the seat of a "destroying" lesion then

the lower centres which should normally be under its control are freed from that control and proceed to manifest activities in accordance with the lower evolutionary level corresponding to those lower centres.

In his Croonian lectures Jackson⁷ applies these principles to delirium. He holds that in this condition there is on the one hand a loss of function in the highest centres, due to a "destroying" lesion, which produces only negative manifestations, such as disorientation and other incapacities. On the other hand there is uncontrolled activity of the *healthy* lower centres, which produces the positive manifestations of delirium, such as restless activity and mistakes in identification. Levin in his paper endeavours to explain the occurrence of hallucinations in delirium along precisely the same lines.

It is to be noted that Jackson's theory is consistent with and helps to explain many of the facts of observation mentioned above. The clinical relationship of delirium to coma becomes immediately intelligible if coma is ascribed to a more or less complete inhibition of function by a "destroying lesion" of highest centres, and delirium to a similar but more incomplete inhibition. The gradation of states from typical deliria to obviously allied but not identical pictures is easy to understand if the essential factor is simply the inhibition of a higher centre manifesting itself by purely negative features, and therefore allowing a great diversity of positive symptoms, because those symptoms are conditioned by factors completely independent of the primary lesion. Further, although toxins of various kinds may all be capable of producing a "destroying lesion" of higher centres, there would be no reason to suppose that a delirium produced by one toxin would differ essentially from that produced by another, because the manifestations of a paralysis of function would be the same whatever the cause responsible for it. This is in accord with the clinical observations of many authors who have failed to find specific peculiarities in the delirium resulting from various toxic processes. Finally, the theory would support the view that an organic-reaction type can be distinguished, characterized by confusion or other features, so long as those features can be shown to be negative in character and hence ascribable to a loss of function.

Further, the view that the onset of a delirium is partly dependent upon a constitutional factor, as shown by the observations of Curtius and Wallenberg⁸ in cases of pneumonia, and by the greater liability of children to develop delirium in febrile illnesses, would fall into line here. Whatever the constitutional factor may consist in, it can easily be regarded as something affecting the tendency of the "higher centre" to break down under stress. Moreover, if it could be established that individuals developing psychoses of any kind in later life had in previous febrile illnesses manifested delirium in a proportion significantly higher than the average, this would support an interesting extension of Jackson's theories to the genesis of psychoses in general.

The chief limitation of Jackson's theory is that, while it provides a satisfactory explanation of the *existence* of a delirium, it contributes but little to the understanding of its *content*. The positive phenomena comprising the latter are ascribed to the uncontrolled activity of lower centres, manifesting themselves in accordance with the evolutionary level which they have attained, and Jackson strives to show that such a symptom as a patient mistaking his nurse for his wife can be interpreted as the activation of a well-organized mechanism existing before the development of refinements in discrimination possible with the evolution of higher levels. This is not very satisfying, however, and it is easier to accept the more general statement of Wolff and Curran⁴, that the content and individual characters of a delirium are dependent upon the individual equipment

and experience of the subject, a conclusion based upon a careful investigation of a series of cases in which the influence of these latter factors is very clearly shown. It is to be observed that, in the present state of our knowledge, such factors can be formulated more illuminatingly in psychological rather than in neurological terms, and hence that psychogenic conceptions must be taken into account in endeavouring to explain the content of a delirium. The obvious relationship of delirium and normal dreaming would find its explanation here.

A problem which presents itself here is how far the content of a delirium is entirely to be ascribed to such factors as "individual equipment and experience," whether those factors be described in neurological or psychological terms, or how far it may in certain cases be partly due to direct stimulation of "lower centres" by a "toxin." That is to say, whether Jackson's assumption that the positive phenomena of delirium arise from the uncontrolled activity of healthy lower centres is wholly correct, or whether those lower centres may not also be the seat of a "discharging lesion." So far as I am aware, only a purely speculative answer can be returned to this question.

Relation of "Psychogenic" and "Organic" in Delirium

In view of the fact that in delirium the clinical picture is obviously determined by processes in part "organic" and in part "psychogenic," it provides an exceptionally favourable field for the examination of the relation between those terms and between the processes which they denote, and I propose to conclude my paper with a brief consideration of this question. It is necessary first to clarify the situation by defining the sense in which the terms "psychogenic" and "organic" are to be used, because a great deal of confusion of thought has resulted from the ambiguities and radical diversities of meaning with which both those terms, as also the word "functional," are employed by different writers.

"Functional" sometimes indicates a disordered condition for which no structural basis has yet been discovered, while sometimes it is equivalent to "psychogenic." In the former sense it refers rather to a stage of knowledge than to an essential character of the condition itself; in the latter it would seem to be superfluous. "Psychogenic" is sometimes employed to denote disorders arising as a sequel to emotional stress or shock, but as the connexion between the emotional state and the disorder is usually conceived in purely chemical or physiological terms this use would seem to be undesirable and misleading. As most commonly applied it indicates a disorder in which the causal factors are considered to be "mental" as opposed to disorders in which the causal factors are considered to be "neurological" or at any rate "physical." The implication is that in the former group "neurological" or "physical" causal factors do not exist, an implication against which the neurologist must emphatically protest, as he necessarily holds the faith that all behaviour processes, whether normal or abnormal, will ultimately be explicable in terms of the nervous system. "Organic" sometimes may mean simply "non-psychogenic," or it may be applied to a disorder in which alteration of brain substance, or a toxic agent causing a modification of brain substance, is known or inferred to exist.

I would suggest that the term "psychogenic" is best used to connote a *mode of explanation*, in which the causal processes responsible for a particular sequence of phenomena are conceived in psychological terms and by the aid of psychological concepts. It does not imply that those causal processes are incapable of being conceived in neurological terms, and certainly not that they are essentially and for ever incapable of being so conceived, although in practice it does generally imply that in the present state of knowledge the psychological mode of con-

ception is more helpful in understanding and controlling the phenomena in question.

The most useful distinction between the connotation of "functional" and "organic," as applied to disordered processes, would seem to be that in the former the causal factors act through the ordinary channels by which those processes are normally activated, whereas in the latter the causal factors do not act through those channels. For example, a hysterical hemiplegia is termed functional because the absence of movement in the paralysed side is produced by the ordinary machinery whereby one side of the body is normally kept still, whereas in an organic hemiplegia the absence of movement is due to an external factor which does not act through the ordinary channels, but is as it were impinged upon the machinery from the outside. The hysterical hemiplegia is further called "psychogenic," because we can conceive the causal processes, which ultimately utilize the ordinary machinery to express themselves, in psychological terms more profitably than in neurological terms. In the future these processes may prove capable of being represented in terms of the activity of the central nervous system, and with this advance in knowledge it may be found that the psychogenic mode of explanation may profitably be discontinued.

If we apply this terminology and these principles to the problem of delirium, it is clear that in the great majority of cases the *primary* causal factors are "organic," and consist in a modification of brain substance produced either by a toxin or a degenerative process, and not by stimuli acting along the channels whereby that area of brain substance is normally activated or inhibited. It is conceivable, of course, that the neurological substrata underlying processes at present best represented in psychological terms may be capable under certain circumstances of producing a similar modification of brain substance, and such a process may explain some instances of hysterical delirium. Only in this sense, however, can we speak of a delirium being *primarily* psychogenic.

If this primary organic process has occurred, the particular features which the delirium displays may be conditioned by factors in which the primary process is not concerned. If we accept Hughlings Jackson's view that these features are due to the uncontrolled activity of healthy lower centres, then this activity will presumably be explicable in the future in terms of neural mechanisms not affected by the primary process. It would therefore not be "organic" in the sense defined above, except in so far as there may be modifications of brain substance due to "constitutional" or "congenital" conditions affecting the functioning of the mechanisms in question. At present it is probable that this "lower centre" activity of Jackson may be best understood in terms of psychological processes and concepts, and hence be properly treated as "psychogenic." In this way we can understand the frequent statement that while the form of a delirium is organically determined its content is largely influenced by psychogenic factors, and the conclusion reached by Wolff and Curran, and other investigators, that the content of a delirium is dependent in large part on the individual equipment and experience of the subject. If, on the other hand, it is considered probable that the lower centres are directly influenced by circulating toxins and therefore not "healthy," then their activity would be partly ascribable to an "organic" process, and not entirely to "functional" processes.

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PULMONARY TUBERCULOSIS AND PREGNANCY*

BY

JAMES YOUNG, D.S.O., M.D., F.R.C.S., F.C.O.G.

PROFESSOR OF OBSTETRICS AND GYNAECOLOGY, UNIVERSITY OF LONDON;
DIRECTOR, OBSTETRICAL AND GYNAECOLOGICAL UNIT, BRITISH POST-GRADUATE MEDICAL SCHOOL, LONDON; CONSULTING GYNAECOLOGIST, ROYAL INFIRMARY, EDINBURGH; CONSULTING OBSTETRICIAN, ROYAL MATERNITY AND SIMPSON MEMORIAL HOSPITAL, EDINBURGH

Of all the facts in regard to the influence of pregnancy on pulmonary tuberculosis with which the obstetrician is brought face to face, the most significant is the devastating influence of labour and the puerperium on the course of the disease. This is well brought out in a survey of the history of forty-six cases which I have carried out for the purpose of this communication. In this group there was a total of fourteen deaths, or 30.4 per cent. If we restrict the study to those cases whose follow-up has extended to one year or more and to those who have died within this period we are left with thirty-six patients, of whom fourteen died, giving a death rate of 38.8 per cent. Of this total eight, or 57.1 per cent., died during the interval from a few days up to three and a half months immediately after the confinement; twelve, or 85.7 per cent., died within two years of the confinement.

The damaging influence of pregnancy and labour is revealed in another way. Out of the eight cases in which death occurred within three and a half months after labour it was found that the pulmonary disease had, in seven cases, existed for periods varying from one to ten years. This demonstrates very clearly the manner in which pregnancy and labour bring to bear upon a chronic disease an acute, critical, and often lethal influence. The same lesson is brought out by the evidence which has been furnished by many observers to the effect that a latent lesion may first reveal itself subsequent to the birth of a child. There are two such cases in my series, in which the onset of the disease became first apparent soon after a birth previous to that for which she was subsequently under treatment. It is obvious that this aspect of the subject is more likely to come within the ken of the physician and the tuberculosis officer than the obstetrician.

Effects on Temperature

The way in which labour and the puerperium so often cause a flare-up in the disease is demonstrated dramatically by a study of the temperature charts of a series of cases. We can in this way recognize how frequently, even in those cases where activity is not in doubt, there occurs a rapid deterioration in the local and general condition. In twenty-seven cases, or 58.6 per cent. of the forty-six cases which have been the subject of this investigation, there was an appreciable depreciation in the clinical condition in the puerperium. In eight, or 17.4 per cent., death occurred within three and a half months.

In view of this evidence, which we may regard as a commonplace in the experience of those obstetricians who have observed a considerable number of such cases, it may seem somewhat surprising that there are still some few

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