

In this earnest plea for more systematic therapeutical, clinical and statistical inquiries, I would not be understood as criticising the thorough work now done in connection with asylums. I have merely attempted to point out the necessity for further progress, and have suggested methods which would tend to increase the efficiency of asylum work.

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### THE CLINICAL SYMPTOMS OF COMPRESSION OF THE BRAIN.\*

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The natural course of development of studies, although observed with evident zeal and good success in other branches of medical science, is still neglected in the department of psychiatry. To the modern representatives of psychiatry, it would seem more admissible and more promising to construct, out of a greater or smaller series of observations, a form of disease, or to assign as peculiar and typical, non-essential varieties of circumscribed forms of disease, than to devote observation and clinical research to the elucidation of the symptoms of morbid cerebral processes. In the case of modern German psychiatry more especially, the imprint of systematized and schematized labor is to be found. The thorough working out of the symptoms of cerebral disease is but little regarded. The structure, as a whole, would seem of greater importance than its constituent elements. This is justifiable, perhaps, from an æsthetic, but certainly not from a physical, point of

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\* Translated from the author's manuscript.

view. The daily experience of psychiatric practice, that like symptoms may be associated with the most different forms of disease, and that, conversely, typical forms of disease may pursue a course with very different phenomena—we recall, in this connection, the multifiform symptomatology of epilepsy and paralytic insanity—this experience of itself, aside from theoretical considerations, were sufficient, one would think, to direct attention to the great importance of studying, for the present, the symptoms exclusively.

The new German hand- and text-books are particularly instructive in this direction. They display a wealth of psychiatric systems which can be grasped by the specialist only with difficulty, and by the beginner not at all. This does not depend, however, upon the naturally large number of possible forms of psychiatric disease, for did these exist, all objection and criticism were uncalled for. On the contrary, the richness of the symptomatology corresponds only to the imagination, broad or limited, as the case may be, of the individual author. Evidence of this exists in the slight agreement among authors, and the practical worthlessness of all the forms of disease.

We have for years endeavored to place in a right light, the disproportion between the labor expended in psychiatric studies and its scientific and practical results. We have repeatedly called attention to the defective elaboration of the symptoms of insanity, and laid stress upon the diagnostic and clinical importance of individual symptoms, and we propose to do the same again in the treatment of our present subject, as we shall hereafter endeavor to show, the study of compression of the brain is of an importance not to be under-estimated in order to a knowledge of a series of psychical phenomena. And even if we do not flatter

ourselves, with reference to the connection between compression of the brain and the individual phenomena of psychical disturbance, that he can demonstrate the entire clinical significance of cerebral compression, this allusion should at all events suffice to stimulate us to the utilization of further observations, and finally promote the solution of this cerebral process.

The phenomena of compression of the brain have been mainly studied experimentally. The clinical aspect of the subject, as worked out by Freidreich,\* Griesinger† and others, had reference to tumors of the encephalon, to cases of recent hæmorrhage, or to hæmatoma of the dura mater. That these do not embrace all cases of compression of the brain, is evident. In them we have either to deal with slowly increasing pressure, involving various disturbances of circulation and the effects of irritation, as in tumors; or we may have, as in cerebral hæmorrhages and recent hæmatoma, variations of pressure occurring suddenly, sometimes also serious destruction of the brain substance itself. But it is clear that other causes of compression of the brain must be added to those already enumerated. We only mention here as such causes, first, hyperæmia (arterial or venous), second, œdema of the brain, and third, hydrocephalus. In weighing these conditions, we are frequently compelled to have recourse to analogy. Not all cases of increased cerebral pressure, and especially those of a transient, curable character, are susceptible of post-mortem demonstration. In other cases, perhaps, the pathologico-anatomical hyperæmia or œdema of the brain found after death, will not be sufficiently intense to warrant us in referring the same to compression existing during life; because thus far we

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\* *Lehre von den Geschwülsten innerhalb der Schädelhöhle*, 1853.

† *Archiv. f. k. Heilkunde*, 3.

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lack any reliable measure for such variations of pressure, which probably play a part in the various phenomena of insanity. Data furnished by experimental research in this direction, can not be simply transferred to our field of enquiry. Not only do these experiments produce, in most cases, merely a sudden, considerably increased and unequally distributed compression of the brain, and for this reason give rise to phenomena of another character, but, on account of the limited number of psychical phenomena in the animals operated upon, the train of symptoms induced experimentally, must be of much less importance than in the case of our patients. The morbid phenomena in man and the lower animals operated upon, can only be in accord in the highest grades of compression of the brain—in a degree of intensity in which the functional energy of the cerebrum is entirely, or almost entirely, arrested. In the course of our enquiry, therefore, we can adduce the results of experimental researches only to a certain extent, and with caution. On the other hand, the results of our own autopsies will be of great value, and go to corroborate our views. It follows unquestionably, from the beautiful experiments of Pagenstecher,\* that disturbance and extinction of psychical activity, stupor, somnolence, sopor and coma are among the most prominent symptoms of serious cerebral compression. Other phenomena, such as convulsions, paralysis, *manège*-movements, and rotations in the longitudinal axis, Pagenstecher is not inclined to connect, with equal certainty, with compression of the brain, inasmuch as they were not of sufficient constancy, and since unilateral functional disturbances of the motor centers and purulent pachymeningitis are symptoms to which these motor phenomena may be more fairly ascribed. To

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\* *Experimente u. Studien über Gehirndruck*, Heidelberg, 1871.

compression of the brain, on the other hand, Pagenstecher is disposed to refer general muscular debility, diminished sensibility and reflex excitability, and incontinence of urine.

As the result of frequent post-mortem examinations, we\* have been forced to the opinion that, with reference to conditions of stupor with more or less anxiety, as they occur in many cases as the terminal stage of paralytic insanity, compression of the brain produced by serious internal hydrocephalus, is to be regarded as the pathologico-anatomical basis.

In order to a due appreciation of the relationship of these facts, the stage of paralysis above mentioned must be more closely examined. We do not require for this purpose any casuistical exposition. Cases which belong here are sufficiently familiar to every alienist, and they are by no means among the rare occurrences of psychiatric practice. In many cases of paralytic insanity, a condition of profound stupor supervenes, days and weeks before a fatal termination. The patient, who a while ago was in a lively amœnomanical frame of mind, indulging ideas of grandeur and a thousandfold projects, or who was addicted to endless hypochondriacal complaining, suddenly becomes quiet and perfectly indifferent, stands in a corner depressed, with anxious expression, and trembling from head to foot, can not be induced to speak, and is heedless of the calls of nature. In the majority of cases the *musculature* is tense and rigid; the patient vehemently opposes every attempted change of position, and as a rule refuses food, so that recourse must be had to artificial feeding with the stomach tube. At the same time, the pupils are generally widely dilated and do not react; the pulse is full and strong, and, at all

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\* *Compendium der Psychiatrie*, Vienna, 1881.

events in the beginning, considerably reduced in frequency. Surface-sensibility is markedly diminished, and sensation is not evinced by any motor reaction. The patient looks at the point where he has been pricked with a needle, yet he does not resent the injury, nor even withdraw the affected extremity when the needle penetrates deep into the flesh. Reflex action occurs in the sole of the foot either very tardily or not at all. I do not remember to have ever seen this stage of paralysis of long duration, a fatal termination occurring as a rule after a few days or weeks. Autopsies showed, without exception, signs of severe compression of the brain. The convolutions of the convexity of the brain appeared flattened, the ventricles much dilated, and the basal ganglia, so far as exposed to the fluid in the ventricles, were also broadened and flattened out. The œdema of the brain was of varying intensity.

To these post-mortem appearances found in cases of paralytic insanity are to be added like conditions occurring in other psychical affections, which will be subsequently discussed in the course of our remarks. The phenomena connected with these terminal stages of paralytic insanity, the correctness of which can be proved by the observations of individual specialists, are in entire accord with those which were obtained by Pagenstecher as the results of his experimental researches on compression of the brain. Here, as in his experience, the most marked symptoms of the abnormal pressure in the encephalon are stupor, great impairment or complete arrest of all psychical activity, and diminished or arrested reflex excitability. The symptomatic correspondence extends farther to less certain results of experiment, the condition of the pupil and the diminished frequency of the pulse. Noteworthy is the refusal to eat on the part of some of the animals oper-

ated upon by Pagenstecher; they had to be fed forcibly. In the majority of our cases in this category there was obstinate abstinence. The patient clenched his teeth, closed his mouth, and strenuously resisted every attempt at feeding in the usual manner—a symptom which is not commonly associated with paresis. We simply point out the symptomatic correspondence without at present entering upon the deeper causal relationship, or upon any speculations regarding the same. But we should certainly be justified in so doing when we looked for and found a connection between the so very constant post-mortem appearances and the symptoms observed in the course of the disease, and when, after long experience, we finally accustomed ourselves to refer the symptoms above mentioned directly to compression of the brain, and to regard them as its clinical equivalent.

In the cases of paralytic insanity here considered, the œdema as cause of the compression of the brain was only of secondary importance. It was not of sufficient intensity and too variable to be of much importance as compared with the marked internal hydrocephalus. Neither was there, in these cases, a very high degree of pressure. Compression of such marked intensity occurs only in rare cases of paralytic insanity, the increase being rapid, and they proceed, often with astounding rapidity (within an hour), to a fatal termination. In such cases the autopsy reveals marked œdema, but they run too precipitated a course to permit of their being clearly comprehended.

Other and frequent experiences in psychiatric practice are admirably adapted to the study of compression of the brain of a more marked character.

These are fatal cases of intense alcohol-poisoning. To him who has opportunity of observing great num-

bers of cerebral affections from alcoholism, cases of more severe poisoning will not be unfamiliar in which the patient perishes in one or two hours, or in one or two days. These are the cases which present the same symptoms as those of a compression of the brain which has been artificially produced. The patient's sensorium seems in the highest degree encroached upon; he is soporous, and lies exhausted, motionless, with eyes closed, opening them only in response to strong irritation, and then with great difficulty. The muscles are relaxed, and when stood upon his feet, the patient can hold himself erect only with great effort. The whole body trembles and quivers; the pupils are widely dilated, without reaction, the features are mask-like and void of all expression; fæces and urine are discharged involuntarily. Sensibility seems much diminished; at all events reflex actions do not occur promptly in response to sensory stimulation. The pulse is strong and generally of diminished volume. The temperature as low as 36° C, and even lower. With such symptoms death occurs, as stated above, sometimes in a few hours, and at latest after one or two days.

The autopsy reveals in such cases, without exception, considerable venous hyperæmia of the meninges and very marked œdema of the brain. It seems to us beyond dispute, therefore, that the symptoms above mentioned are to be referred exclusively to the effect of the compression of the brain as the result of the extreme œdema. We are forced to this opinion because the symptoms of compression produced experimentally coincide most clearly with the clinical phenomena which we have described, and the autopsy does not indicate any other pathologico-anatomical origin.



Of much more importance than the cases hitherto cited must be that series of cerebral affections in which the compression does not attain so high a grade, nor proceed to a fatal termination. Moreover, this series is much more extensive and embraces various forms of psychical disorder. Aside from the slow increase and gradual diminution of the cerebral affection, the phenomena of the less intense compression frequently constitute the only symptoms of the psychosis. In other cases again the pressure occurs as a stage of the more complicated cerebral affection, and as such is of very variable duration.

In accordance with the above experience and the points to be subsequently discussed, we shall be able, as a general rule, to regard the condition of psychical motor arrest, termed stupor in psychical symptomatology, as depending upon increased pressure in the encephalon. We can not, however, in support of this broader conception, refer so extensively to the results of experimentation. Neither will each diagnostic point in this connection find its anatomico-pathological equivalent, inasmuch as these cases are examined post-mortem only in rare instances, and after a much more protracted course than those of paralytic insanity and intense alcohol-poisoning referred to above.

It has become the custom to regard, as a stage of melancholia, conditions of stupor beginning with general depression, under the name of *melancholia cum stupore*, in contra-distinction to *melancholia activa* or *agitata*. We have elsewhere insisted\* that stupor is not to be confounded with the clinical phenomena of melancholia, that the condition of stupor stands in no closer connection with melancholia than with any other form of psychical disturbance; and if the systematic

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\* *Compend. d. Psych.*

grouping of different symptoms is to have any significance at all, we must not lose sight of definite characters as belonging to a definite complexus of symptoms. This is not the case, however, if we regard as characteristic the anxious and gloomy frame of mind, with all its concomitant expressions of self-reproach and tendency to suicide, while at the same time we consider the state of total psychical and motor arrest, without spontaneity whatsoever on the part of the patient, likewise a melancholic condition. Depression and fretful self-disparagement, the cardinal symptoms of melancholia, have just as little to do with stupor as with typical mania. It is an error to suppose that there is behind stupor a condition of depression and anxiety. Convalescents from stupor remove all doubt in regard to this point, since they aver that they thought of nothing and were incapable of thinking, intimating that all cerebral activity is impossible.

This is not the case with melancholics. Psychical processes take place, if in dreary monotony of distressing ideas. The melancholic is never so apathetic and void of all spontaneity, never so listless and indifferent as the patient in a state of stupor. Apart from the intercurrent stages of extreme excitement which frequently supervene under the influence of great anxiety, the melancholic always evinces a certain degree of activity, a necessity to give vent to his woes and self-depreciation, which is entirely excluded by stupor. We may refer, in this connection, to the frequent cases of senile melancholia, which are diagnostically obscured by stupor only in extremely rare instances, indeed almost never.

The essential feature of stupor is the almost entire interruption of all cerebral functions, psychical as well as sensory and motor. Psychically, a complete chaos

prevails. It is just as little a question of mental discordance, as of ideas and thoughts of any kind whatsoever. This is clearly shown by the diminished conduction of sensory irritations, the suspension of all sensation of pain, and finally, the absence of all voluntary motor activity. From this true stupor, however, must be distinguished the speechless and motionless condition frequently observed, during short periods, in patients under the influence of strange delusions; although the specialist should experience no real difficulty of diagnosis from this source.

Similarly, the refusal of food in patients in a condition of stupor, must be viewed in a different light from a like refusal in those whose actions are controlled by morbid ideas, for while in the latter abstinence has its evident psychical motive, it depends in the former upon an organic basis at present unknown, but which may be considered from the same point of view as phenomena arrived at by experimental research.

The description of stupor here given, corresponding, we believe, to clinical observations, renders it in the highest degree probable that the condition, in the view we have taken of it, represents the true picture of primary and acute dementia.\*

In view of the thorough similarity between the symptom-complexus of the psychical condition here described, and that of the corresponding experiment, the theory of a like cerebral cause in our cases, can not be disputed. Add to this that that stage of general paralysis which corresponds symptomatically, in every particular, with cases of insanity with stupor, is dis-

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\* Esquirol called stuporous insanity *dementia acuta*, and Schüle describes (aside from melancholia attonita) acute stupor, where it can not be regarded as a stage in the course of a psychosis, as *primary acute dementia*.—  
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tinguishable, post-mortem, as the result of a compression of the brain which had existed during life; and after referring to individual post-mortem appearances, noted by ourselves after cases of protracted stuporous insanity, which were characterized by a *high degree of chronic œdema* of the brain, we shall have exhausted the data which we have to advance in corroboration of our views. As regards the effect, of course, it is quite immaterial whether the compression in the one case is produced by extensive hydrocephalus or by general œdema of the brain.

We can not here investigate the circumstances and conditions under which such œdema or hydrocephalus, with the symptom-complexus peculiar to them, may have developed. In all cases of acute stuporous insanity, as well as in numerous others in which stupor forms but an inconsiderable stage in the course of the entire psychosis, we lack the necessary premises to this end. We are just as little able to explain the course of development of a high degree of compression of the brain by œdema, such as has been observed by us in some cases of concussion of the brain. We recall, more especially, the case of a young man who fell violently on his head, without showing any external injury. After a brief period of unconsciousness, the patient awoke in a condition of extreme sensory benumbedness, and with a complete arrest of all psychical functions. The pupils were widely dilated and did not react; there were severe tremors of the entire body; he could maintain the erect posture only with great difficulty, and bore an external resemblance to a person highly intoxicated. His discharges were passed involuntarily. Reflex actions were entirely wanting. The patient, when pricked with a needle, directed his eyes to the place of the injury, but made no withdrawal attempt.

The whole *musculature* was rigid, almost tetanic, and there was an energetic resistance on the part of the patient to all passive movements. On account of a persistent refusal of food, it was necessary to feed the patient with the stomach tube. About five days after admission into the clinic, he died quite suddenly.

With the exception of an intense arterial hyperæmia and œdema of the brain, the post-mortem examination revealed nothing abnormal. Again, I have at present a female patient under treatment, who, in the course of a melancholia, developed, during the puerperal state, had thrown herself from the window of her residence, into the court-yard. The patient arose at once after the fall, spoke to those who had witnessed the event, and returned, unassisted, to her dwelling. She did not become unconscious till about a quarter of an hour after the fall, and then for a short time, and awoke in a benumbed and stuporous condition, which was, at times, interrupted by a dream-like, but slightly active, delirium. The further course of the affection, which was characterized, psychically, by a high degree of apathy and sensory benumbedness, has now been dragging on for months. Refusal of food has persisted since the fall; likewise a peculiar rigid and unstable and motor condition; involuntary discharges; much diminished sensibility and arrest of all reflex excitability. The patient's condition resembles, in all its symptoms, albeit the stupor is not of so high a grade, cases of compression of the brain confirmed by autopsy, and reminds one of a dement. Neither immediately after the fall, nor in the further course of the psychical disturbance which followed the same, and which differed entirely from the depression of mind which preceded the fall, have any symptoms developed which would point toward a cerebral hæm-

orrhage or any other injury of the brain substance, and which, we think, therefore, may be rightly excluded. We rather ascribe the condition in this case to an œdema of the brain, brought about by the concussion, and refer the clinical phenomena to the effect of an increased compression of the organ.

The development of the cerebral compression, the clinical symptoms of which present themselves after an epileptic attack, in *post epileptic stupor*, would seem much clearer than in the foregoing cases.

After repeated epileptic seizures, sometimes even after a single seizure, there follows in man, as well as in animals experimented upon, a stage, varying in duration from minutes to several hours, in which the individual remains completely benumbed, almost motionless and wholly stuporous, in no way reacts, is insensible to pain, staggers about as if drunk, in short, presents all the symptoms described by Pagenstecher, as occurring in animals artificially exposed to an intense cerebral compression. Regarding all these cases, the assumption is supported and justified that the suddenly developed anæmia of the brain, as the result of vascular spasm, which, in accordance with all experience in this direction, must be considered the immediate cause of epileptic convulsions, is substituted by an excessive dilatation of the vessels of the cerebrum. The vascular spasm is followed by a transient paralysis of the muscular coat of the vessels; the atonic condition of the walls of the vessels produces dilatations of the blood-ducts, with a corresponding fullness of the same, and probably resulting, subsequently, in œdema of the brain-tissue itself. In fact, post-mortem examinations of persons who have died during an epileptic seizure, as a rule reveal nothing more than hyperæmia and an excessive œdema of the brain.

By far more evident than the development of compression of the brain in cases which are of importance from a psychiatric point of view, is the connection of the already existing pressure with the clinical symptoms as considered above. The explanation given by Pagenstecher that the pressure experimentally produced, compresses the vessels of the organ, thus interfering with its regular and normal nutrition, is probably only a part of the interpretation. We can assume with certainty that, apart from the diminished calibre of the blood-ducts, the compression of the brain substance itself is of equal importance. In regard to the effect of the pressure, it is of course of no consequence whatever whether the pressure is produced by an injected substance, as in the experiment, or by hydrocephalus or œdema of the organ. We must therefore, in reference to our cases, also take into consideration the twofold effect of the pressure, viz., the compression of the blood-ducts as well as that of the tissue of the brain. Both will render the clinical symptoms comprehensible. It will be easily understood how under the influence of this double effect, viz., the deficient nutrition of the brain and the compression of the nerve tracts, cerebral function may become impaired or almost entirely arrested. It can certainly not be attempted to illustrate details in the clinical picture from these gross, morbid, cerebral processes, nor can the post-mortem appearances serve the purpose of explaining any fine points in the symptom-complexus. Yet the correct comprehension and interpretation of the most marked symptoms as the stupor, the arrest of all spontaneous brain function, the lack of all initiative activity, the impeded or arrested conduction of external impulses, the characteristic motor relaxation, in their dependence upon the compression of the brain, is suffi-

cient to enable us to recognize the causal connection between the cerebral morbid process and the clinical phenomena. But at the same time the unfavorable prognostic significance of protracted stuporous conditions will be more easily understood from this consideration. If the insufficient supply of nourishing material, as the result of the compression of the nutrient ducts on the one side, associated with the direct compression of the nervous tissue of the brain, lies at the foundation of the affection, there is nothing surprising in the frequent unfavorable termination of the psychosis *quoad sanationem*. We know from similar experiences concerning other organs that each of the two factors referred to suffices *per se* to induce histological changes of a persistent and irremediable character, and how much more must this be the case where we have the combined effect of both factors.