

The Pathology of Sudden Death in Mania. By JAMES R. WHITWELL, M.B., Assistant Medical Officer, West Riding Asylum, Menston.

That in the continued and severe excitement which occurs in some cases of mania, either of paretic origin or otherwise, a sudden condition of collapse may occur which may or may not terminate the case is well known; the exact cause of this condition, however, in each individual case, is frequently a matter of the greatest difficulty to decide. In many cases, however, putting aside coarse hæmorrhages, one may reasonably expect to find some pulmonary or cardiac condition of sufficient magnitude and gravity to permit of its selection as the actual cause of death.

Of the cardiac conditions, either organic valvular disease or some muscular incompetence, associated, perhaps, with a fatty change in the organ, is the most common; of the pulmonary conditions, probably congestion and œdema of the lungs and pneumonia are the most frequent, and it is probable that under one or other of these headings many of the cases of so-called "exhaustion from mania" should be placed. It is especially to the pulmonary conditions found in these cases that attention is directed in this paper.

Pulmonary œdema may occur in these cases as a result of at least two conditions, firstly, as a sequential pulmonary lesion to a failure of the heart, a frequent cause of pulmonary œdema apart from mental cases, and secondly, it may occur as a result of pulmonary embolism not necessarily associated with any abnormality of the heart.

Pneumonia may be associated with acute mania in various ways:—1st, it may be that the pneumonia is a causative or concomitant condition which produces death, either by the extensive area involved or by cardiac or other complications. 2nd, it may be that the pneumonia has occurred as an inter-current disease, in the same manner that it may attack a sane and otherwise healthy individual. There seems, however, some reason to believe that the administration of chloral in these cases of acute mania not only frequently tends to assist in the production of the pneumonia on account of its effect on bodily heat, but further, by its cardiac action, may assist in interfering with an otherwise not specially unfavourable prognosis. 3rd, it may be that the pneumonia has occurred as an inflammatory

condition of the lung, superadded to the condition of fat embolism.

The following case demonstrates the condition of acute pulmonary oedema occurring in a case of long-continued mania, associated with the presence of fat emboli in the lung:—

E. L., male, æt. 34, first attack; admitted December 2nd, 1889. A man of fair physique, stature, musculature, and nutrition; face much scarred with small-pox of old date. Right eye shows remains of old corneitis and has a marked anterior staphyloma. Bodily systems, generally speaking, practically normal, and no obvious sign of syphilis to be noted. Left pupil (the only available) reacts fairly well to diffuse light and accommodative efforts. Knee jerk and superficial reflexes present to normal extent and equal. Mentally, patient is continuously noisy and restless, talking, gesticulating, singing and shouting, and can scarcely be kept quiet for a single moment; he frequently sings songs of current interest, and passes rapidly from one to the other, and a note or word given to him is readily fitted with some song in which it may occur prominently. Any word mentioned in his hearing has frequently a more or less accurate and appropriate rhyme adapted to it. All this is done by the patient with his eyes mostly shut, and with his hands and feet wandering about in all directions. He shows little or no tendency to violence, but is particularly mischievous, upsetting everything that is within his reach in the most casual way, not being at all disturbed by the crash which sometimes occurs as a result of his deed. With the exception of a day or two, during which he was fairly lucid and quiet, this condition of mania was steadily kept up for 4½ months, with the physical result that, during the first month, he lost flesh considerably; during the second he began to put on flesh a little and regain a presentable appearance; after this, however, he steadily lost weight until his death, which took place on April 20th, 1890. At two o'clock of this morning, while in the midst of one of his frequent nightly outbursts, he somewhat suddenly became quiet, which drew the attention of the night attendant, who found him lying on his back, with a pale and somewhat dusky complexion, and evidently seriously ill. On arriving I found him unconscious, collapsed, and distinctly cyanotic; pulse 90, of fair tension, and of sufficiently good volume to render the idea of syncope at least doubtful. A sphygmographic tracing, taken at this time, showed a well-marked predicrotic wave, and also some irregularity in force and rhythm, his usual pulse being one of very low tension. Respiration regular, laboured, and but slightly increased in frequency; numerous moist crepitations were heard in the chest, especially at the left base. His condition gradually became worse, and he died on the evening of the same

day. At the necropsy no point of special interest was observed, except in connection with the lungs: the right weighed 545 and the left 1,160 grammes; the latter was intensely œdematous and congested from apex to base, and small pieces, taken at random, showed, on microscopic examination after treatment with osmic acid, numerous fat emboli, scattered throughout the sections, both in the capillary vessels and in the smaller branches of the pulmonary artery; portions only from the left lung examined. The heart showed some patches of fatty degeneration in its muscular substance; the liver also was fattily degenerated. Although the most careful examination was made, both during life and after death, no sign of bruise or injury, either to soft parts or bone, was detected, with the exception of a faint, pale, yellow bruise of very old date over the right eye and temple, covering an area of about $1\frac{1}{4}$ inch in diameter.

This case is a type of what occurs clinically in a certain percentage of maniacal cases, and is very apt to be regarded as a case of exhaustion from mania or death from fatty degeneration of the heart, and, doubtless, each of these conditions, in many cases, are factors in the fatal result. Still, the pulmonary condition adds a new and unexpected feature to the case, which must be a most prominent element in, if not the absolute and immediate cause of death. Exhaustion from mania is too indefinite a term to be discussed here, and is largely a screen in the absence of more definite knowledge, but in fatty degeneration of the heart producing syncope, one could scarcely find a pulse of such fulness and tension as occurred in this case, and, in addition to this, the dusky pallor and cyanosis all tend to lead one to the diagnosis of fat embolism in the lung as the actual cause of death. It is difficult to see in this case why there should be none of the well-marked embolic infarcts, but only apparently an acute œdema of the lung; many cases, however, both experimental and clinical* (*vide* Bergmann), are on record which have shown a very similar condition on post-mortem examination.

Of the connection of fat embolism with pneumonia the following case is an example:—

J. G., male, æt. 35, second attack; admitted February 7th, 1890. A man of fair physique, stature, musculature, and nutrition, and of dark complexion. Left eye has a large leucoma over the centre of the cornea. Bodily systems practically normal. Right pupil reacts but slightly to diffuse light, but well to accommodative effort. Left knee jerk much brisker than right, the latter being

* Bergmann, "Berliner Klin. Woch.," No. 33, 1873.

about normal. Superficial reflexes practically normal and equal on the two sides. Mentally, patient is apparently much demented, is dull, apathetic, and quite regardless of his position and circumstances, has no cognition of his surroundings, cannot tell his home address, nor is he aware of any dates. With but little mental or bodily variation, patient remained in this state till May, when he became somewhat excited, and pulled a good deal of hair out of his head and beard; this excitement, however, only lasted a short time, and he soon returned to very much his mental condition on admission, and remained so until September of the same year, when he entered upon a state of continuous and acute mania, which lasted till his death in November, and during this time he was continually noisy, restless, and troublesome, singing, shouting, tearing, and jumping about whenever he was not under the influence of sedatives, and he steadily lost flesh throughout the whole period. At about two a.m. on the 8th of November he was noisy and excited, and quite suddenly, in the midst of his excitement, fell backwards, his face became of a dusky cyanotic hue, he had considerable dyspnoea, and his pulse was irregular, but not of very good tension. This condition of collapse and dyspnoea steadily increased until death, which took place about two hours after the commencement of the seizure. At the post-mortem examination the left lung showed oedema of the base and posterior part, but not to any great extent, and considerable emphysema anteriorly. The right lung, throughout its middle and lower tubes, showed a large number of wedge-shaped embolic areas, with their base at the surface of the lung, varying in size from an inch square downwards, and, independently of these more condensed patches, there was considerable oedema of the greater part of the lung. In the upper lobe were also a few infarcts, but the most noticeable feature of this lobe was a large patch of pneumonia in the grey hepatisation stage, which abutted against and merged into some of the embolic areas, and would seem to be an inflammatory condition superadded to a previous attack of embolism, which gave rise to so few symptoms, perhaps owing to the relatively small area involved, as to pass unobserved. Microscopic examination, after treatment with osmic acid of sections taken from portions of the lung in the immediate neighbourhood of the infarcts, showed very numerous fat emboli of various sizes. The changes in the other organs, noted post-mortem, were comparatively unimportant; no special fatty change in any of the organs. Commencing cirrhotic change in both kidneys and considerable hepatic venous congestion. No sign of bruise or injury of any kind observed, either during life or after death, though special search was made with this object in view.

That embolism of a portion of the lung is a not very unfrequent cause of pneumonia has been proved by the

observations of Virchow,* Panum,† and Cohnheim,‡ and, judging from the surrounding circumstances discovered in the lung in the case of J. G., there is every reason to believe a pre-existing embolism was the cause of the pneumonia; if this is so, it would explain in a most satisfactory manner the frequency of pneumonia as an inter-current disease in mania, and would, in addition, add another factor to be considered in connection with the prognosis of inter-current pneumonia in cases of mania. It is noticeable that the infarcts in fat embolism, when present, are mostly near to the surface of the lung, in which case they are readily observable after death. Where, however, they are not to be discovered, fat emboli may be present and produce acute œdema of the lung, and it is possible that, in these latter cases, the emboli are mainly blocking vessels in the body of the lung, which, perhaps, are not terminal in character.

Fat embolism of the lung was first observed in man by H. Müller§ in 1860, and has since been observed in pyæmia,|| diabetes,¶ injury to bones, mania,** and the status epilepticus;†† and, in addition, has been produced experimentally by various methods and persons. Dr. Jolly‡‡ has drawn attention to this condition in mania, detailing a case previously recorded by Dr. Flournoy, and surmised that, as injuries of the bones were absent, the introduction of fatty matter into the circulation was by reabsorption from the self-inflicted bruises, which are apt so frequently to occur in cases of violent mania, and, in evidence that this is possible, quotes a case of Dr. Fitz§§ (cited by Flournoy), in which mechanical injury of subcutaneous adipose tissue had led to reabsorption. Halm,||| however, has not been able to produce fat embolism by mechanical injury of the subcutaneous tissue in dogs, and in the cases detailed in this paper there was certainly no evidence of any recent injury discovered, though diligent search was

* Virchow, "Gesammelte Abhandlungen zur Wiss. Med.," 1862.

† Panum, "Experimentelle Untersuchungen zur Physiologie und Therapie der Embolie," 1864.

‡ Cohnheim, "Untersuchungen über die embolischen Prozesse," 1872.

§ H. Müller, "Würzburger Med. Zeit.," 1860.

|| Wagner, "Archiv für Heilkunde," 1862.

¶ Sanders and Hamilton, "Edin. Med. Journal," July, 1879.

** Flournoy, "Contributions à l'étude de l'embolie graisseuse," 1878.

†† Clouston, "Journ. Ment. Sci.," July, 1879.

‡‡ Jolly, "Archiv für Psychiatrie und Nerven [Krankheiten]," Bd. xi., Ht. 1; "Journ. Mental Sci.," July, 1882.

§§ Fitz, "Boston Med. and Surgic. Journal," May, 1878.

||| Halm, "Beiträge z. Lehre v. d. Fettembolie," 1876.

made with that special object in view. In Dr. Clouston's case also, that of an epileptic who had had a steady series of fits for two days and died comatose, no injury was noted. In this patient there was discovered post-mortem fat embolism of the lungs and pia mater, and, in addition, extensive fatty degeneration of the liver, kidneys, and heart; it was also observed that the cancellated tissue of the bones was very open, and was filled with a grumous fluid. It would appear that our knowledge on this subject is not sufficiently complete to be able to refer with any degree of certainty to the source of the fat in each individual case; there is, however, some evidence to show that, in all probability, fat embolism can occur without any rupture into any pre-existing fat in the body, in which case it is probably due to some obscure change in the blood, producing a condition of lipæmia, perhaps having its origin in the bone marrow, the result of the long-continued and severe mania or convulsions. I may therefore summarize as follows:—

- 1st. A not unfrequent cause of sudden collapse which may or may not result in death in cases of mania is fat embolism of the lung.
- 2nd. That it is to be suggested or diagnosed during life by the presence of the following points:—
 - i. Sudden collapse, with coldness of extremities, etc.
 - ii. Dusky pallor of face, sometimes marked cyanosis.
 - iii. Some dyspnoea: respiration may be shallow, sighing, or laboured.
 - iv. Pulse of fair volume frequently, usually irregular.
 - v. Stethoscopic examination revealing pulmonary œdema or secondary embolic pneumonia.
- 3rd. That it is to be suspected after death by:—
 - i. The observation of intense local œdema of one or both lungs.
 - ii. The occurrence of actual infarcts in the lung.
 - iii. The presence of localized pneumonia, which may or may not be associated with infarcts.
- 4th. That the actual source of the fat is not at present known, but
- 5th. That fat embolism of the lung can occur in these cases without any discoverable injury to either bone or subcutaneous tissue.
- 6th. That it may possibly be due to a change in the blood, brought about by the long-continued maniacal excitement.