Some writers have attempted to classify insanity into emotional, ideational, impulsive, and intellectual insanity, according to whether the case showed more prominently an emotional or ideational disturbance, etc. I say more prominently, for there never was yet known a case in which either of these artificial divisions of mental phenomena was alone affected; the brain-functions were never so dissected, as it were, by disease.

Again, some authors divide the cases of insanity according to their supposed causes. They speak of mania *à potu* as a distinct malady, while others fix upon a mere concomitant, and describe a puerperal mania, or phthisical mania, making a mere accident the godfather to the disease; as, again, in kleptomania, oinomania, etc.

Surely this is a great and very unnecessary departure from the rules of pathology. We might as well speak of the varieties of fractures, as fractures from falls, from blows, from pressure, etc.; or describe puerperal fractures, scrofulous fractures, and so on. In general pathology, too, a disease once ascertained in its early stage continues to be called by the same name throughout all its stages. A case of pneumonia is pneumonia from beginning to end; the first, second, and third stages have not distinct titles, and are not disconnected in works on medicine. It would be as rational to classify disease by its duration, as cases of one week, or two weeks, three weeks old, etc. Yet this is very much what is done by some writers in their classification of mental diseases.

We read in reports of such a case as epileptic mania complicated with paralysis. Surely one disease is enough at a time, and as surely our nomenclature should signify an unity, not a trinity, of morbid processes. Of course, we cannot expect to achieve a nomenclature which is perfect; such a consummation presupposes a complete knowledge of all diseases. But, admitting that our system must be only a provisional one, we ought to guard ourselves against allowing such an auxiliary to become our master instead of our servant; against perpetuating error, instead of aiding in the discovery of truth.

When we speak of acute mania, do we not imply that we are alluding to a disease which is of a distinct and separate morbid species? or, rather, do we or do we not mean a disease as distinct in all its characters, in its progress and termination, as small-pox, syphilis, cancer, etc.? If we do not, we should acknowledge the fact at the outset, and say that mania is a symptom merely, like cough, jaundice, tormina, etc. But mania finds a place in most published treatises on insanity, and occupies, indeed, a prominent position in most classifications, and is divided also into several varieties.

If mania be a disease *per se*, of course it must commence as mania, continue and terminate, always a mania; and if so, I would ask, is such a malady ever met with?

I extract from a French author the following example of the mode of describing the condition. "Mania," he says, "was thus described by Esquirol. A cerebral affection of chronic character, ordinarily without fever, and characterised by perturbation and exaltation of the emotions, the understanding, and the will. It breaks out suddenly, following some strong emotion, an alcoholic excess, or a prolonged incarceration, and mounts in a few hours to its highest degree."

I have, with this view in mind, waited now some twenty years without finding anything in my experience to agree entirely with this description. If this description be that of a distinct species of disease, I have never met with any clearly marked instance of acute primary mania; and my experience now ranges over several thousands of cases.

I have several times supposed that I had such a case under observation, but on thorough investigation I have never been able to satisfy myself of the fact. I use the word primary to avoid misconception, though I do not admit its propriety: all true morbid species of course are primary. I have also received patients certified to be labouring under acute mania, cases commencing, that is, as mania and so continuing; but I have never been able to verify such a diagnosis. I have searched reports, newspaper accounts, trials in which persons have been accused or tried for sudden outbreaks of maniacal violence, or whose crimes have been excused on the ground of insanity; but, whenever any history of the accused's case has been given, I have never found that the outbreak was the first symptom of the disease. The cases which I have received as examples of acute primary mania have been of the following character.

Firstly—and these appear to correspond most nearly to the description given of acute mania—are cases of general paresis, in what the French call the expansive stage. I believe it to be almost universal that maniacal symptoms occur very early in the course of general paresis. There is a stage of excitement evidenced at first by great garrulity, which speedily goes on to an outbreak of maniacal violence. It is not uncommon, as we all know, for this stage to pass away before any motor symptoms make their appearance, and the patient may be even discharged cured at this juncture, and his case may seem to justify a diagnosis of primary

mania; but the motor symptoms, according to my experience, invariably appear, and in the sequel all the other characteristics of paresis. It would not be correct, however, to call the same pathological conditions by two names. The disease is general paresis from first to last; one morbid process only is in operation in the patient's system from beginning to end.

I think that other cases, considered at first sight to be examples of primary mania, are secondary attacks of recurrent insanity. In such cases, the attack, though it be the second or third, may not be known, or it may be concealed by those who bring the patient to the asylum. The premonitory symptoms of these secondary attacks are often of very short duration, and show but slight deviation from the ordinary condition of the patient, to detect which it may require a well-tutored observation. The symptoms of maniacal character quickly succeed; they are often well-pronounced, and seem to the friends and non-medical observer to be the first phenomena of the attack; but such cases are not instances of primary mania.

If it be the fact, then, that no case of insanity commences as mania, we should expunge the name from the list of diseases, and use the word only as a term for a symptom or series of symptoms of a given character. And if it be true that no primary case commences with violence, the knowledge of the fact becomes of the utmost value in a medico-legal point of view also.

ON THE ACTION OF ALCOHOL.

By JAMES ROSS, M.D., Waterfoot, near Manchester.

I WISH to supplement my paper "On the Action of Alcohol," which appeared in the JOURNAL of October 4th, by a few additional remarks. When I wrote that paper, I was quite aware that Dr. Beale, in common with Professor Binz and many others, was of opinion that alcohol was one of a class of agents which checked the movements of white blood-corpuscles and other masses of protoplasm; and that from this fundamental action, all the other effects of these agents upon living organisms was to be explained. I was not, however, aware that Dr. Beale had to a very large extent anticipated the views which I now hold so far back as 1863. The author has kindly sent me a small pamphlet of his published at that date, entitled, "On Deficiency of Vital Power in Disease, etc.," and the coincidence between some of his observations on the action of alcohol and some of my statements is altogether remarkable.

I will, with permission, cite one or two of the more striking passages. After describing the morbid changes which take place when a slight abrasion of the cuticle inflames, he proceeds. "Now what happens when a drop of alcohol is applied to such a sore? Momentary pain, followed in the course of a few minutes by great relief, or complete cessation of pain, and diminished vascularity. But how does the alcohol bring about such changes? If alcohol be added to any serous fluid, the albumen is precipitated. If delicately granular cells are placed in alcohol, and afterwards examined under the microscope, every one knows that they will appear shrunken, and will have altered much in form; and they would resist disintegration by pressure to a greater extent than they did before. The surface of the wound is covered with a dry crust, produced by the hardening effects of the alcohol; and some of the rapidly growing cells are quite destroyed, while others become surrounded by an envelope of hardened matter, which prevents the possibility of their absorbing nutriment, and giving rise to new cells so rapidly as before. Not only so, but the permeating power of the nutrient fluid itself is diminished by the tendency of the alcohol to coagulate it. The most superficial of the cells, composed entirely of germinal matter, would be destroyed by alcohol, though not so quickly, perhaps, as by the actual cautery, nitrate of silver, sesquichloride of iron, sulphate of copper, etc."

Dr. Beale then goes on to make interesting remarks on the action of alcohol upon the vessels and nerves; and he finally discusses the action of the agent when taken internally. After noticing that many agents have the property of coagulating albuminous fluids and solutions of extractive matter, he says: "The favourable action of such remedies is probably due to their direct influence on the fluid constituents of the blood. They, no doubt, also diminish the rate at which blood-corpuscles are disintegrated, and at the same time they tend to render the walls of the blood-vessels less permeable to fluids. But of all remedies, I believe alcohol acts most rapidly in this way, and in these particular cases (pneumonia, etc.) most efficiently. The properties alcohol possesses of hardening animal tissues, and of coagulating albuminous fluids, are well known; and these properties must not be forgotten, when its effects in the animal body are discussed. Of course, when

absorbed by the blood, it does not actually coagulate the albuminous matters; but it probably renders them less fluid, and reduces their permeating property. Alcohol interferes with the disintegration of blood-corpuscles; and in cases where this is going on very rapidly, and where fluid is passing through the walls of the vessels in considerable quantity, in consequence of the walls themselves being stretched and too readily permeable to fluids, alcohol is likely to be of service; but where these changes are occurring very rapidly, and the patient's strength fast ebbing, it may save life. We may, therefore, explain the beneficial action of alcohol, without assuming that it is a food, or contributes directly to the process of nutrition." And again, he says: "This view of the action of alcohol accords with many broad facts familiar to all. It accounts for the shrivelling of the hepatic cells, the shrinking of the secreting structure, and the increased hardness and condensation of the entire liver, which results from the continual bathing of the gland-structure by blood loaded with alcohol. It accords with the gradual shrinking and condensation of tissues which occur in persons who have long been accustomed to excess. The tendency to increased formation of adipose-tissue, which occurs in persons who live generously, and seems to be augmented by alcohol, may be explained upon the same view; and the stunting in growth which follows its exhibition to young animals is readily accounted for."

These extracts will show that Dr. Beale has, to a very considerable extent, anticipated me by ten years in the theory of the action of alcohol which I advanced in the JOURNAL a short time ago. For a fuller exposition of his views, I must refer my readers to the little pamphlet* itself; since no number of detached extracts can do complete justice to an author's opinions.

I am happy to be able to add that, in a very kind private communication, Dr. Beale disclaims the idea of accusing me of intentionally appropriating his ideas without due acknowledgement; and that he cordially congratulates me on coming to the same general conclusion from a different point of view. The hypothesis was originally suggested to me by Mr. Herbert Spencer's remarks upon narcotics, reference to which I have already given. I must leave others to judge whether what I have said upon the subject has given to the hypothesis additional significance.

PULMONARY APOPLEXIES (HÆMORRHAGIC INFARCTIONS) IN CASES OF CEREBRAL APOPLEXY.

By J. HUGHLINGS JACKSON, M.D., F.R.C.P.,

Physician to the London Hospital.

IN his highly interesting Harveian Oration (BRITISH MEDICAL JOURNAL, July 5th, p. 5), Professor Rolleston refers to Brown-Séquard's important experiments, which show that injuries of certain encephalic centres are followed by pulmonary hæmorrhages. I wish to point out that some of the "experiments of disease" on the higher nervous centres of man produce the same result. I have alluded to this briefly in my article on Apoplexy in Reynolds's *System*, 2nd edition, vol. ii.

I shall in this article use the term pulmonary "apoplexy". It is true that to use this term for local internal hæmorrhages is to write inaccurately; but I never, or scarcely ever, hear used any other term than pulmonary apoplexy to describe what is better called pulmonary hæmorrhagic infarction. In the expression "cerebral apoplexy", the term apoplexy is used strictly; but it is well in this paper to qualify it by the word cerebral. In the two expressions, pulmonary apoplexy and cerebral apoplexy, the word apoplexy has quite different meanings.

After cerebral apoplexy—the effects of any comatous lesions being included in this term—numerous conditions of lung are found. As regards differences in mere quantity of blood in these organs, there are two causative factors—1, the robustness of the patient when taken ill; 2, the rapidity of his death. As a rule, the more robust the patient, and the more rapidly he dies, the more blood in the lungs; but in one case of death in an hour from large cerebral hæmorrhage, I found that the patient's lungs weighed together only twenty-two ounces. They were very like—quite like, I think—the lungs of patients who have died in cholera collapse. This case puzzles me very much, as I have found the lungs much congested in patients who have only lived apoplectic half an hour or less (as after meningeal hæmorrhage). But, as a rule, the lungs are the more congested the more rapid the death, provided death

does not occur *very* rapidly, does not occur within, let us say—and I only state my impression—two hours.

I have above spoken as if there were only degrees of congestion of the lungs in cerebral apoplexy; but in a considerable number of cases we find other appearances *post mortem* which certainly cannot be passed by as "congestion". It is of these that I wish to speak. I omit further reference to well known conditions of posterior congestion, and need not detain the reader by speaking of œdema and acute emphysema.

We see in some cases, on slicing the lung, not only a general reddening of the dependent parts, but *patches* also. These patches are not confined to lobes, although they affect the lower lobes most. They vary in tint from a black to a granite colour; and in some lungs we find both colours and intermediate degrees of colouration. The sections are margined, slightly raised; the masses can sometimes be felt from the surface, but very indistinctly, as nodules. The black nodules vary in area of section from a half-crown to a split pea. The granite-coloured ones vary similarly. These appearances—the differences of stages being well marked—were very striking in the case of a largely built patient aged 50, whom I saw a few months ago with Dr. Stephen Mackenzie. In this case, a few patches were broken down into a pulp.

I think it is plain that the above description of the dark patches is that of "apoplexies"—hæmorrhagic infarctions of the lung. The large black patches are indeed, I consider, unmistakably "apoplectic". The patches (nodules) in heart-disease are, however, for obvious reasons, harder. In a case of coma from injury to the head in a very robust man, I found lungs which were very like, if not quite like, the "damson-lungs" of heart-disease; and I found the same in a case of apoplexy from opium-poisoning. The suicide was a healthy man eighteen years of age.

As to the pathology of the granite-coloured patches, there will be a difference of opinion. As we find them along with black patches in some cases, and as there are all degrees of intermediateness, I suppose them to be decolorised pulmonary "apoplexies". This is what Brown-Séquard has said. But I often hear them called pneumonic. There is, I suppose, the hypostatic pneumonia of Piorry. But, whatever the pathology of the patches may be, I think that the term pneumonia is an unfortunate one. There is not clinical pneumonia. Pneumonia should be kept for an acute affection—of one lung in most cases—which has a fairly definite course, beginning by a severe rigor, and attaining its climax in about a week. When the patches I have mentioned are so numerous as to be confluent, we may find the dependent parts of the lungs bulky and solid. But even then both lungs are nearly always affected—one more than the other, it is true, in many cases; and they are not affected in one or more lobes only, but in all lobes and in the dependent parts of these. When, however, there are a few scattered granite-coloured masses, the condition will occasionally be called lobular pneumonia. The geographical part of the expression is, I think, correct; it is a *lobular* affection, but it has not the clinical associations of lobular (catarrhal) pneumonia. It is lobular; but there is no evidence that it is inflammatory—that is, inflammatory *primarily*. Sometimes the pleura is inflamed over a patch seated near the surface; but the same can occur in the pulmonary "apoplexies" of heart-disease, which are certainly not inflammatory primarily. It occurs also in the hæmorrhagic infarctions of pyæmia. The latter, by the way, present stages of decolorisation analogous to those of which I speak in severe cases of coma. It is true, they go on to suppuration; and so, although very rarely, may the apoplexies of heart-disease.

The general opinion of medical men in this country, so far as my knowledge goes, is, that pulmonary "apoplexy" in heart-disease is owing to impeded *exit* of blood. It is not, however, easy to understand how this process can lead to *localised* hæmorrhages, even if it be easy to understand that it can lead to general congestion, splenisation, etc. The more likely explanation seems to be, that the "apoplexies" result from pulmonary embolism. Virchow's experiments, indeed, demonstrate that embolism does produce pulmonary apoplexies. The embolic material in cases of heart-disease is believed to be blood coagulated during partial stasis on the chordæ tendinæ; and I suppose the same explanation holds in the pulmonary apoplexies of cerebral apoplexy. Every one knows that splenic and renal blocks are *red* when recent; and the accepted explanation of "red softening" of the brain is, that it is dependent on *obstruction* of small *arteries*. It is true that there are difficulties in understanding how increased quantity of blood, and even extravasation, result from plugging of arteries; but of the facts of the matter there is no doubt. It is warrantable, then, to infer that the pulmonary apoplexies of heart-disease or of cerebral apoplexy arise in a like manner from blocking of arteries (pulmonary). It is not necessary to deny that impeded exit has something to do with the production of pulmonary "apoplexies".

* *On Deficiency of Vital Power in Disease, and on Support; with Observations upon the Action of Alcohol in Serious Cases of Acute Disease.* By Lionel S. Beale, M.B., F.R.S. London: T. Richards, 37, Great Queen Street. 1863.