recorded cases of sudden catalepsy produced by fright,* and these instances may be regarded as bridging over the borderlands between health and disease. An analogous condition to fright rigidity and catalepsy is also probably experienced in nightmare, and I have met two instances of medical students who on awakening when under this peculiar condition of depressed emotion found themselves unable to move for nearly a minute.

At this time, when hypnotism is so much in vogue, it is interesting to note that Heidenhain writes, "that the hypnotic state is nothing more than an artificially produced catalepsy."†

In the foregoing remarks I have traced back the symptoms of disease to the functions of health, and carried both back to their origin in evolution, and by these means I have attempted to give a rational explanation of the symptoms of melancholia.

CLINICAL NOTES AND CASES.

Case of Chronic Meningitis. By J. W. PLAXTON, M.R.C.S., Medical Superintendent of the Lunatic Asylum, Kingston, Jamaica.

Around pachymeningitis curiosity and interest have long circled: the pages of the "Journal of Mental Science" bear witness. Men felt that the current explanation of its development was inadequate, and, if they did not deny its truth, they received it with suspended contradiction rather than acquiescence.

Huguenin's explanation ("Ziemssen," Vol. xii., p. 306), that pachymeningitis, as we usually see it, is merely a case of new tenants in a deserted house, is one which must ultimately oust the older idea.

In support of the newer I put on record in the "Journal of Mental Science" for January, 1889, a case of "Shrinkage of a Hemisphere and subsequent Pachymeningitis." In that case a one-sided pachymeningitis existed, complementary to an equally circumscribed shrinkage of the cerebrum.

Huguenin's explanation will stand good for those cases of which hæmorrhage is the essential fact, and of which antecedent diminution of the natural contents of the skull is almost

* Hack Tuke, "Influence of Mind on the Body," p. 308.
† Heidenhain, "Hypnotism," p. 25.

essential. But the gist of the explanation is this, that we have not to do with a pachymeningitis in these cases at all; we have to deal with an organizing blood clot in the subdural space, nothing else. It is, therefore, high time we dropped such an inappropriate term as pachymeningitis in cases of the kind. Why should we retain a special term for an organizing clot in the subdural space any more than for one in a ligatured artery?

There are cases, of which I have one to narrate, of a true chronic meningitis, the outcome of which is such a thickening of the dura-mater as to render the term pachymeningitis appropriate, but this category has nothing in common with that of the usurping blood-clot—neither cause, course, nor resultant. In the pretender to this name the cause is, if I may say so, a *vis a fronte*, a *vacuano*, in the rightful heir injury, or some one or other of the more obscure causes of inflammation. In their course the two differ, as deposition by the avalanche (hæmatoma) differs from the orderly deposition of water; and the resultants are as conditional, a talus or a stratification.

W. B., male, aged about 40 years, had been a patient in the Jamaica Lunatic Asylum for two years, when he was discharged recovered on July 26, 1886. After an interval of 16 days he was readmitted August 12, 1886, in an acutely excited condition. The police reported that as early as the seventh day after his discharge his conduct had become irrational, and proclaimed mental elation and grandiose ideas. On the second of August he was arrested by the police, and detained two days in the lock-up. Whilst under detention he behaved well, said he did not know why he had behaved irrationally—"it must have been a joke." He was set at liberty with the consent of the medical attendant, and straightway behaved as before. He entered several houses, and conducted himself as though he had been the rightful owner. Re-arrested on the sixth, he could not explain his conduct, was merry and joyous, and at night was noisy and destructive, and so violent as to need the strait-jacket. He was lewd and lascivious, and stripped himself naked. He attempted a carnal assault on a female in the presence of others.

On admission, it is only necessary to remark that his bodily condition was indifferent; that his skin bore evidence of recent troubles, such as a recent cut of upper lip, abrasions, and, amongst others, one bruise on the left side of the forehead. Pulse rate noted as 60.

After admission, for about three weeks he continued more or less noisy and excited, and then seems to have settled down into the beaten ways of the place—at times quarrelsome and inclined to bravado, but working now with the bricklayer, now with the porter.

A legacy from my predecessor, I knew him for more than a year before he died. Big and burly in body, he had the aspect of an epileptic; dull and fatuous in the face, his mental operations were limited, and slow at the best. The occurrence of a fit about a year before his death seemed to set this matter at rest.

Six months later the power of co-ordination of the muscles of the lower limbs failed gradually, attained some severity, persisted for a time, and passed off.

He had a convulsion again six months before his death, and then on several (five) occasions during the last two months of his life. Finally, he had a series of thirty (during which he was unconscious), between the hours of 10 p.m. and 7.30 a.m., at which time he died, August 9, 1888, after a residence for the second time of two years.

Sectio cadaveris made August 9, 1888, 2½ hours after death. Body that of a well-made man, fat, and without external markings. Rigor mortis present in arms and ankles, not in knees.

Head first examined. Scalp thick and congested; skull cap and whole bony walls of skull unmistakably thicker than natural, and very vascular; the general thickening in certain places ran into localized excesses of a coarsely porous bone, which overlay lesions of the subjacent membranes and brain.

The excesses were on the inner surfaces of the bones, and occurred:—1. On the inner surface of the left half of the frontal bone. 2. At the posterior inferior border of the right parietal bone, and the contiguous portion of the occipital. 3. In the right cerebellar fossa of the occipital bone. The outer surface of the dura mater, laid bare by taking away the skull cap, showed nothing noticeably amiss, but its inner surface at the spots corresponding to the porous patches of the bony walls without was adherent to the brain within (the membranes intermediating). It was greatly thickened at these places, but elsewhere was not abnormal-looking.

After slitting round the dura-mater with the probe-pointed bistoury, and exposing the dome of the cerebrum, the first thing to be seen was a partial view of a patch of the left frontal lobe, over which thickened arachnoid passed bladder-like; next, raising the frontal lobes, this patch resisted, being in a trifling degree adherent to the inner surface of the dura-mater. The extrication of the bed of the right hemisphere of the cerebrum was resisted in like manner, and came away, leaving behind cortex adherent to the walls over the area of a shilling. Finally, almost the whole of the right hemisphere of the cerebellum was found to be glued to the dura-mater of the right cerebellar fossa. The encephalon, therefore, was adherent to the walls of the skull at three several points, and, moreover, had suffered the extensive changes now to be described at those points.

α. In the left frontal lobe, in the second and third convolution anterior to Broca's convolution, brain, over an area of a crown

piece, had been replaced by a boggy and gelatinous, but not diffluent substitute, glazy in section. The change reached halfway from the surface to the anterior horn of the lateral ventricle. The gyrus in front of Broca's took part in the disease, but Broca's did not.

β. A patch of the superficial area of a shilling occupied a central position on the outer surface of the right occipital lobe. Although the cortex and subjacent medullary matter had undergone change here, it had not undergone removal and substitution, nor did the change penetrate deeply.

γ. In the cerebellum the convolutions of the posterior and outer surfaces of the right hemisphere were so altered as to have become hard and dense to the finger, and resistant to the knife. They had lost their characteristic colour, and showed boldly on the face of the section. Each leaflet looked shrivelled, and was distinct from its neighbour. The ground given up by its contraction had been yielded up to an intrusive gelatinous-looking tissue, which lay between the leaflets, forcing them apart, yet holding them together.

The pia-arachnoid, speaking generally, was but little, if any, thicker than natural, or manifestly diseased.

The cerebrum generally was plump and well convoluted.

The vessels were contracted, hence were small, but no degenerative or other changes were recognized.

The sum of the changes visible on simple inspection, shortly expressed, were, in addition to a general thickening of the bony walls of the skull, local disease of bone, meninges, and encephalon over contiguous, superseded areas.

Cerebrum from the frontal lobe and the cerebellum, with its thickened investiture, were hardened in Müller's fluid, and examined microscopically.

In a stained section from the cerebellum, mounted in balsam, profound alteration from the healthy condition was evident without the aid of the microscope. The dura-mater, arachnoid, and pia, taken as one, occupied $\frac{5}{32}$ of an inch of the margin of the section, of which dura-mater claimed $\frac{3}{32}$. The section, stained in carmine, had passed through healthy as well as diseased tissue, and the contrast between them at once struck the eye. An unaffected leaflet showed, first, a broad lightly-tinted zone, which was the cortex from the pia-mater covered surface inwards to the granular layer; then the granular layer, with its outer edge sharply defined from the cortex, and its inner border well marked, but less sharp, broad, and deeply stained; then, innermost of all, the medullary region faintly tinted.

As the affected tissue was neared, the granular layer narrowed, and was lost to the eye, and the whole section showed an almost uniform depth of coloration, the medullary region being indicated by a triflingly less depth of tint. The extreme outer edge was also

differentiated by comparative pallor (this is not unusual where chromates have been used for hardening), but not at all points. Occasionally the colouring deepened evenly from centre to edge.

Microscopic examination; power $\frac{14}{1}$. When examined with this low power the divergence from the normal state of things seen with the eye was confirmed. When attention was turned to the granular layer, as it passed progressively from the healthy to the affected regions, that layer was seen to narrow until but the faintest indications of its persistence remained. The loss was from the deeper layers, and the position of the trace to which it finally became reduced showed that the cortical layer had shrunken also to just one half its depth in the unaffected leaflet.

The membranes exhibited with this power ($\frac{14}{1}$) the external boundary of denser dura-mater; the sub-arachnoid region widely areolated, and the pericerebellar pia-mater thickened, and deeply coloured, because crowded with cells and blood-vessels; but there was no sharp line to be drawn between any two of them. At one or two points the pia-mater, with its cells, invaded the cortex itself, and was confused with it.

With higher powers of the microscope ($\frac{500}{1}$) the evidence of the naked eye and the lower powers was confirmed in detail. The lower strata of the meninges were seen choked with cells, and fusion of the pia and cortex, where such has taken place, were seen to have occurred along the track of entering blood-vessels.

Cortex and granular layer (minus granular) exhibited an almost even staining, and in excess of the medullary region. From the granular layer, the granules by which it is known had entirely disappeared. The gradual loss in the transitional region was seen more distinctly, and the only indication of the position it should occupy was the trace spoken of above as visible under the lower power. This trace was now seen to be a continuation from the granular layer of large nuclei, irregularly placed three, four, or five deep in a narrow band. They were for the most part oval, with the long axis almost always perpendicular to the surface of the convolutions; occasionally the outer end was obtusely pointed. They resembled most closely the nuclei of the muscle cells of the middle coat of one of the smaller arteries. They did not seem, however, to have any such relation, nor to have any cell territory of their own, nor do my preparations show that they possess processes.

No cells of Purkinje could be demonstrated in the altered cortex, but, on the other hand, they were few and difficult to demonstrate in the healthy parts. My failure in this may be due in a measure to the length of time the tissue had been soaking in Müller's fluid.

The cortex was diminished in breadth, and denser than in the normal parts of the sections.

The blood-vessels showed much more numerously; their sheaths

were wide, and the peri-vascular spaces full of cells—exactly resembling the cells which crowded the inmost layers of the pia, and the granules of the normal granular layer—leucocytes.

Thick plugs of cell-crowded pia-mater dipped down between convolutions.

A section made from the tissue, which in the frontal lobe of the cerebrum had taken the place of the vanished convolutions, showed a web of blood-vessels and capillaries, with the intervascular spaces bridged by fine fibrils in every direction; the fibrils being prolongations from single connective (neuroglia) tissue cells, which must have been bathed in fluid, for the whole tissue is a sponge, and section-making only possible after infiltration with paraffin. After solution of the paraffin, a loose filmy web was left, which it was impossible to mount with its parts in relation.

The connective tissue cells stain magnificently, as they always do when taken from the neighbourhood of a lesion of this kind, and it is here that I have always best seen the connection of the processes of such cells with the walls of capillaries.

The alterations which the microscope showed to have occurred may be enumerated as:

1. In the frontal lobe: Absence of cerebral matter beneath a wide area, and replacement by, or, perhaps, it would be more correct to say, its reduction to, a connective tissue, loose-meshed and vascular.

2. In the cerebellum: Condensation and contraction of the cortex, loss of the cells of Purkinje, obliteration of the granular layer, and a general increase of vascularity.

3. Thickening and fusion with each other of the dura mater, arachnoid, and pia mater, and, rarely, fusion of the pia with the cerebellar cortex. An orderly overgrowth; in fact, a chronic inflammation.

After such detail it remains to bring the rays of observation to the focus of explanation. The disease had two centres, and no more—the left frontal and the right occipital regions. At these centres, from without inwards, from the bony wall to the viscus, all the structures were implicated. Inspection and the microscope accounted for the disease in the walls as a chronic inflammation, a true meningitis; and, in the cerebellum, had no other explanation to offer. The cicatrix, which in the left frontal lobe had replaced so much of the brain matter, is not to be explained in the same manner; it was not caused by extension inwards of the inflammatory process, nor was it from this point that the disease set out. The first suggestion is negatived by the microscopic appearances, those of quiescence; the second by the fact that the membranes covering the cicatrix, although distinctly involved and adherent, were adherent

to a trifling extent only, and that the posterior region of disease was distinct from this.

It was at first difficult to ascribe to a single cause the meningeal and visceral lesion (cicatrix); but, after some thought, the conjecture which seems to best satisfy the given conditions is concussion, and to concussion, I believe, the beginning of the disease is to be attributed.

The cicatrix in the frontal lobe I interpret as marking the seat of maximum intensity of the injury, probably of hæmorrhage, and of the neurotic softening which followed (?).

As strengthening my supposition, the lesions occupy extremities of an axis of the skull, though on different sides of the brain. The man had been extremely violent before his second admission, and it is remarked that he had a bruise on the left forehead when admitted.

The man had been insane four years, and whilst insane these meningitic processes had been active within his cranium. It would be satisfactory if their interconnections could be made out, but with the best intention the foundations are much too slender for anything but a conjectural explanation. (Perhaps it is fortunate we have no history. In Jamaica mirage is almost as prevalent as in Tarascon.)

If the conjectured concussion could be carried back to a time antecedent to the insanity, no one need, I think, hesitate to connect the insanity with the accident and its consequences as cause and event; and I have already given reasons why the concussion should be carried back to the time of his second admission at least; but, further than this, there are certain additional facts which bring me to the belief that the conjectured injury did indeed precede his insanity, and cause it.

This extension of opinion is grounded on the age of the cicatrix in the frontal region of the cerebrum. The cicatrix was perfect, all necrotic products had been removed, and the activities of the adjoining cerebral tissues were quiescent. Again, the immediate recurrence of insanity on his return home from a life of indolent quietude to one of excitement and riot (there is evidence of this) suggests the fanning into flame of old fires.

If it may not be conceded that the psychic disturbance was from the first dependent on the lesions discovered after death, I submit that it is probable that the insanity of his second and final residence in the asylum was so. Firstly, for the reasons already given; secondly, for the reason that the characteristics of his condition were that his wits were dull and his sense

blunt, and that during the earlier months of his residence, especially at the time of his admission, he exhibited well-marked grandiose delirium, and other peculiarities which led to the suspicion of general paralysis, a disease in which cortical and meningeal changes have part. There is no need to take further note of the epilepsy and inco-ordination of muscles than to bear in mind that "congestive" attacks accompany general paralysis, and inco-ordinated movements indicate interference with the functions of the middle lobe of the cerebellum.

My opinion is that this man's case was one of concussion of the brain in the first instance, with, as an immediate result, a local destruction of nervous matter, and the kindling of a chronic meningitis, the mental disturbance being dependent and resulting.

I am here brought back to my starting point, that pachy-meningitis is an unusual term, adopted for a condition which is now understood to be little more than the organization, almost abortive, of a sub-dural blood-clot. With such a condition my case has nothing in common but situation, but there is danger that the term will be applied to both.

I spoke incidentally at the outset of the case in which hæmatoma was the result of shrinking of a hemisphere; and to contrast that case with the one just related has been most interesting.

In that case the cerebral lesion was confined to the motor area—in this case the disease overran large areas of the cerebellum and cerebrum, but all outside the pale.

In both the objective symptoms were complementary; in one the disorganization was advertised in the striking capitals of crippled limbs and contracted muscles; in the other it lay enshrouded in the dim characters of clouded intelligence and in mistranslatable convulsion; a ray of light entering in with the transient disturbance of motor co-ordination.

Case of Insanity associated with Chorea in Advanced Life. By N. M. MacFarlane, M.B., Clinical Assistant Perth District Asylum, Murthly.

B. McD., domestic servant, æt. 64, was admitted into the Perth District Asylum on April 6th, 1888. Came from Perth Poorhouse, where she had been an inmate for two years. There was no family history to be got, and her personal history was meagre. Symptoms of insanity in the form of delusions of suspicion, hallucinations of