made to depend, as it ought to do, on the facts of particular cases. Room is being found in the criminal law for the plea of moral insanity and the theory of modified responsibility. It may be noted, in conclusion, that a bold step has just been taken by Germany. The new Civil Code (s. 1569) recognises the lunacy of a spouse as a ground of divorce, but only where the malady continues during at least three years of the union, and has reached such a pitch that intellectual intercourse between the spouses is impossible, and also that every prospect of a restoration of such association is excluded. If one of the spouses obtains a divorce on the ground of the lunacy of the other, the former has to allow alimony, just as a husband, declared to be the sole guilty party in a divorce suit, would have to do (ss. 1585, 1578). The inquiry which this paper has initiated might easily be carried further, but perhaps enough has been said to show the lines on which useful work might be done.

(1) A family council is composed of six blood relatives in as near a degree of relationship to the lunatic as possible; if there are not six, relatives by marriage are then chosen. Such a council is always presided over by the *Juge de Paix* of the district where the lunatic is domiciled (Civil Code, Arts. 407 and 408).

The Physical Signs of Insanity. By F. GRAHAM CROOKSHANK, M.D.Lond., late Assistant Medical Officer Northampton County Asylum.

IT is disappointing to anyone trained in modern clinical methods, and accustomed to hear alienists urge with so much insistence that insanity is a brain disease, to find so little apparent attention paid to what may be called the physical signs of insanity.

It would be foolish to declare that these physical signs have not been observed. But is there not a tendency to speak of them merely as interesting phenomena met with amongst the insane, and to forget that they are consequences of those brain changes which make up the somatic background to what we call insanity? Have we not of late somewhat neglected the old-fashioned method of induction from clinical observations?

The stigmata of degeneration, it is true, have had attention

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enough. But they are not physical signs of insanity ; they are merely marks—valuable, no doubt—of a general protoplasmic vice which reveals itself, so far as the brain is concerned, by idiocy, criminality, insanity, eccentricity, or wayward genius.

We must believe that the physical phenomena of insanity—no less than those of sanity—are, if not strictly dependent on, at least the concomitants of certain activities or changes, cellular or molecular, of brain tissues. And unless there are brain cells whose activities are aimless as far as the body is concerned, and are simply the concomitants of physical states, and unless in insanity the brain changes are restricted to these hypothetical cells, we must admit that the cellular activities which accompany insane states of mind have some resultant effects on the physical economy.

It is to these resultant effects—the necessary result of any brain disease accompanied by insane states of mind—that in the first place we assign the term "physical signs of insanity." But it is sufficiently obvious that, at least in the case of general paralysis, there are modifications of physical functions the result of changes in brain cells whose activities so far as we know are not accompanied by conscious or vividly conscious states of mind. Such modifications are not necessarily physical signs of insanity, inasmuch as they may be the result of purely local brain affections, tumours, etc. But when occurring in the insane they are direct evidence of the insanity being, as we believe it is, connected with brain disease.

Attending the out-patients of any large hospital one frequently enough sees patients who, when tested, fail to recognise or to correctly name objects presented to them. We are told that such persons labour under amnesic defects ; and the defect is regarded as a physical failure and located in one or other brain convolution. In every asylum one may see scores of patients exhibit these particular amnesic defects ; though, to be sure, in their case the defect is only one of many disabilities, and hence does not stand in strong relief. Should we not do well to endeavour to analyse, no less carefully than our colleague of the out-patients, this amnesic defect, and to localise the peccant convolution ? It is true that in the one case the pathologist will find a small hæmorrhage, or may be a thrombosis. In the asylum cases no organic change may be found. Yet a failure of function has an anatomical situation not a whit

the less real because it does not happen to have a gross "organic" cause obvious to our dull eyes.

Now Dr. Hughlings Jackson has enunciated the remarkable law, that in every case of insanity a negative lesion of highest centres exists, which causes some paralysis, sensory or motor (*Journal of Mental Science*, iii, 1888). Dr. Jackson's evidence, however, relates almost entirely to the insanities of epilepsy and to post-epileptic states; and though Dr. Anderson has brilliantly discussed general paralysis, and Dr. Mercier coma, there seems to have been but little attempt to correlate what observations have been made of the physical signs of insanity in the widest and popular sense of the term.

Many valuable hints, it is true, are scattered through Dr. Francis Warner's papers, and Dr. Turner has contributed some accurate clinical observations (*Journal of Mental Science*, i, 1892).

But Dr. Turner's observations, which were concerned chiefly with the asymmetry of expression seen in the insane, would have been more valuable if he had expressed the physical signs in terms of the nerve centres responsible rather than in terms of the muscles involved. As Dr. Turner says, it is by studying paralyses accompanied by physical states that we shall be enabled to identify the cortical sites whose integrity is necessary for the accomplishment of those physical changes whose psychical concomitants are peripherally expressed by muscular contraction. But in nerve centres movements not muscles are represented, and some of the movements seen in insanity, and called movements of expression, depend really on lower (not cortical) centres, and have little, if any, conscious accompaniment. Hence the apparent dislocation of expression and emotion in insanity. A spasm or weakness of one half the face, and affecting one half the occipito-frontalis, cannot depend on cortical changes. It must be the fault of the seventh nucleus or nerve. Hence to speak of asymmetry of "*expression*" in cases exhibiting this sign is incorrect.

No one expects the psychical state of a tabetic with double ptosis to be necessarily that of sleep or repose; and no one talks of abnormality of expression in such cases.

The essentials of expression do not lie in peripheral muscular arrangements; these may be more or less brought about from different levels. What is important is the state of those brain

cells whose activities are accompanied by the appropriate emotion or other conscious state.

We should then clinically attempt to express the physical state of the insane, (1) in terms of the peripheral resultant, (2) in terms of the governing brain region involved. It is so that we describe a case of brain tumour; it is so that we should describe a case of insanity from brain decay or intoxication.

The most obvious of the physical signs of insanity are, of course, those muscular arrangements truly expressive of the predominant insane emotion. We do not cry because we are sad, as most would say; nor are we sad because we cry, as others would say. We are sad, and we cry, that is all that we know. But the crying, whether in sanity or insanity, is the physical sign of the emotion; the resultant of those cellular activities which are the physical concomitants of sadness.

Psychologists have detailed the motor resultant of these cell states; and Jackson summed the matter up once and for all when he said, "The emotional centre represents all parts of the body, though doubtless the heart and viscera first and most." And a full acceptance of this proposition entails one or two interesting consequences.

I see a man daily who suffers from an overwhelming sense of anxiety, fear, dread; no very uncommon case. The physical signs of his insane emotion are precisely what psychology would lead us to expect; he displays weak voluntary innervation; a certain amount of vaso-constriction; a contraction of certain facial muscles, and, most important, a rapidly-acting heart. His pulse rate is 120. There is no discoverable cardiac disease in the ordinary sense of the word; there is no reason to suppose any.

His rapid pulse rate is simply a part of the expression of his predominant emotion.

We are bound to recognise that there is representation of the heart in the highest brain levels, inasmuch as one result of the activities of those brain cells whose changes are accompanied by psychical changes is a rapid cardiac action. In this insane man the rapid pulse rate is, in fact, a physical sign of brain disease—of perversion of function of the higher brain levels.

There is cardiac representation in the lowest level—that we know. We know of no conscious state accompanying outgoing

processes from that level: if any accompany the incoming processes they must be the ordinary organic cardiac sensations.

It is most probable, then, that the viscera, like the muscles, or rather like movements of the muscles, are represented at more than one brain level. And, just as many of the psychical states corresponding to certain highest level muscle actions—crying and other modes of expression—are those we call emotions, so it seems that the psychical activities correlated with highest level visceral representations are "emotions" or complex states. For example, take respiration. With lowest level interference with respiration we have no conscious state; with voluntary changes in respiration we have certain simple conscious states; with highest level changes in respiration we have complex states of consciousness of which the change is a physical sign, *e.g.* with the arrest of respiration, attention (Ribot).

The cortical representation of viscera, if a fact, has still further importance. It is true that many think a "visceral delusion" proof of visceral disease, and are innocently surprised that so little relief is gained by treatment of the viscus. Is not the visceral delusion a proof, not of visceral disease, but of disorder of the "visceral centre?" The peripheral morbid condition, if any, is surely a physical sign of the brain disease. A woman has sexual delusions—central failure. Surely the local pelvic congestion is a physical sign of the central state, just as the tachycardia of my just-quoted case is a physical sign of the brain disease and insane psychosis.

Who would seek to cure a lesion of the Rolandic area by treating the resultant palsy locally? And who would think the brain tumour an "effect" of the paralysed hand? Yet such seems to be the logic of alienists who clamour for gynæcologists and general physicians instead of seeing in these uterine congestions, cardiac irregularities, and so forth, physical signs of disease of certain brain areas, disease none the less real because it may depend on no very gross lesion.

Certainly in some cases, as in that of a lad I know who thinks he has monkeys at his heart, there does exist organic visceral disease. But the fault lies with the central nerve cells, in this case the cardiac centre cells, which, perhaps of never great stability, break down functionally and for association under extra strain.

As a rule, the condition of the special viscus of a patient with delusions relating to that viscus, depends on functional perversity of the central cells representing that viscus, no less than does the twitching of a thumb in Jacksonian epilepsy depend on a lesion of the Rolandic cortex.

If any proof were wanting of the elaborate way in which visceral movements and functions are represented in the highest brain levels, it is surely to be found in the phenomena of vascular "stigmata," and in experiments such as that of Ribot in which, by concentrating attention on a finger tip, pain or discomfort is felt as a result of very localised vaso-dilatation.

It is very probable that just as we see motor or sensory derangements from affections of different brain levels, so visceral delusions may be able to arise at more than one level. At any rate hallucinations may be due to failure at the periphery or lower levels, while delusions are of more central origin.

But to return to the physical signs of insane emotions. Bearing in mind what has been already pointed out, that apparent incongruities of expression do not really depend on the mechanism of expression at all, but on low level or peripheral and independent changes, we should be able, from a study (1) of certain muscular dispositions; (2) of certain visceral states, to deduce at any rate the predominant tone of feeling in the insane, as certainly as we do in the sane. And moreover, the abnormal persistence of certain muscular dispositions or visceral conditions is evidence of nerve disease, actual and localisable, just as the abnormal persistence of an idea or tone of feeling becomes proof of insanity.

Leaving now those physical signs indicative of changes in the brain cells correlated with emotional states, we may direct attention to other clinical evidences of local brain disease in insanity. If any case under consideration be one of "general paralysis," hesitation is not shown in naming anatomical situations as the probable seat of cell changes causative of the muscular states. Yet there is a curious reluctance to do so in cases of ordinary insanity. Though certainly Sir J. Crichton-Browne has pointed out how the abnormal persistence of certain gestures and movements of the insane must be due to abnormal functional activity of certain cortical regions, in the Rolandic area probably.

Again, if certain symptoms of insanity, such as incoherence, defects in writing, failure of powers of recognition, inability to read aloud correctly—all familiar enough in cases of mania and dementia—if all these be studied carefully, isolated, so to speak, they are seen to correspond closely with the aphasias, amnesias, agraphias, and so forth, of the hospital clinic. They are all signs of definite disorder of function of the brain—physical signs of brain disease. Why, then, do we not employ the ordinary clinical terms in describing our cases of insanity, instead of vaguely stating "patient is lost; confused and incoherent." Certainly there is this difference: the lunatic is unaware of his defect; his paraphasia is one symptom among many. The paralytic may be aware of his defect, and it stands out crisply in the clinical picture.

Again the blunting of sensation met with in dements, and the great increase in reaction time, is surely a physical sign, no less than in tabes, of direct nerve failure.

The general motor weakness of persons with melancholia is obvious enough; why should we not call this paresis? If it were marked on one side of the body only we should do so; as it is general we ignore it, or talk of lack of will power. But surely it is a weakening of muscular power depending chiefly on defective central nerve activities.

In mania of the acute and delirious types surely excessive reflex activity is obvious, and the movements are inco-ordinated.

Dr. Mercier has suggestively shown that every case of coma is really a case of total paralysis. And in advanced dementia is there not very real paresis, with almost total loss of truly "voluntary" movements?

I know an asylum attendant of great sagacity and native shrewdness. He is always in the habit of speaking of feeble patients as "much paralysed," and incurs no little ridicule in consequence. But I remember the case of an old man, to most people a case of senile mania, who displayed restlessness and great weakness. The attendant in question persisted in saying the man was much paralysed. At the post mortem disseminated cerebral sarcomata were found, subcortical and in the motor areas. The man was paralysed truly enough. Would he not have been so, save for the name, if the failure of

his Rolandic areas had depended simply on lack of nutrition and arterial disease?

The motor weakness, the feebleness of advanced dementia, is true paralysis, a physical sign of disease of the middle level. In fact, if, looking at motor physical signs alone, we consider that in melancholia there is general weakness of innervation (especially of finer highest level actions), that in mania there is failure of complex co-ordinated movement, and unfettered activity of lower states, that in deep dementia only automatic and quasi-automatic movements are retained, and that in coma, where the bodily powers, together with the mental, fine to the vanishing point of death, practically only movements of the vaso-motor and respiratory organs persist; we have the "types of insanity"—melancholia, mania, dementia, and amentia—arranged in the order which Dr. Sankey years ago declared to be the clinical order. And this order of the "types" or rather "stages" of insanity corresponds in essentials, if not superficially, to the order of the stages of general paralysis.

The physical signs of a comatose man, whether the coma be due to trauma, poisons extrinsic or intrinsic, or to organic disease, are, as Dr. Mercier has shown, those of a man in whose brain all functions above the lowest (bulbo-spinal) level are abrogated.

Respiration is of the bulbar type; no modifications of respiration or circulation can be produced by stimulation of the highest centres.

Voluntary movements are not performed; only the lowest "most organised" of automatic movements.

Sensation is absent and so are sensory processes. In dementia sensory processes, though not absent, are greatly blunted; new complex movements cannot be acquired; such movements as are performed are only "organised" habitual movements.

Reflex activity, though not abolished as in coma, is very sluggish, and it is almost impossible to produce, through the mechanism of the highest level, pupillary, cardiac, and respiratory changes. In fact, with the disappearance of the emotion, the power of expression has gone. Speech is limited; amnesia is profound; agraphia and alexia, mind blindness, and word deafness very common. In fact, the physical signs are those of a brain in which the cortical faculties are reduced almost to

the uttermost, and, in katatonia and allied states, it is to the basal ganglia that control of the musculature seems abandoned.

In mania the physical signs clearly enough seem those of unfettered reflex activity, of destruction of highest controlling levels. The exaltation and excess of nerve tension spoken of by some writers mean, not real excess of nerve action, but uncontrolled action of a lower type, the exaltation being merely as the spasm of the legs in lateral sclerosis. The finest and most complex movements are badly performed; sensation, so far from being more acute, is actually blunted.

The state of the viscera—the cardio-vascular and respiratory viscera—demonstrates also the loss of highest controlling powers. The physical signs of mania, in fact, are the signs of a brain in which the highest level is degraded, and functions of the body are governed and ordained by the middle level.

But in melancholia the physical signs, well enough known, the sluggish innervation, the poor circulation, the feeble respiration, the impaired acuteness of sensation and sensory processes relating to the external world,—all point to a general enfeeblement with commencing decay or impairment of the highest level.

Looking in this way at the physical signs of insanity, one is forced to ask, was not Sankey right in calling the chief types of insanity (melancholia, mania, and dementia) stages of one progressive process?

Is not every case of insanity in a sense a case of general paralysis—a stage in a progressive dissolution of brain, sometimes partial, sometimes general, sometimes arrested, more often not to be arrested? Is it not the fact that the more complex the causation of an insanity, the wider the brain area seriously affected, the less curable the lesion, the more the clinical picture resembles that of a stage of general paralysis? The term "general paralysis" is, of course, a convenient one for certain brain dissolutions which run a certain course and have more or less well ascertained causes and pathology. But the cases confounded clinically with the general paralysis of the text-books are those of general brain dissolution from alcoholism, from general arterial disease, meningeal disease, or disseminated tumours. Is not general paralysis, then, not a thing apart, but the perfect example of progressive brain dissolution, imitated more or less perfectly by the other insanities.

It is true, and herein lies the chief difference, that the abrogation of brain function in the ordinary insanities is less often due to gross and permanent organic changes; but then, the more general and the more permanent the change, the closer becomes the resemblance to a stage of true general paralysis.

As Hughlings Jackson said, in every case of insanity there is a negative lesion causing sensory or motor paralysis, and it is to the observation and enumeration of these paralyses—the physical signs of insanity—that our clinical efforts should be applied.

So far I am afraid I have dealt chiefly with generalities. I will endeavour to enumerate some of the physical signs of brain disease in the insane. Some classification is necessary, and at present it seems best to follow anatomical and clinical paths, though there necessarily must then be some confusion between physical signs of insanity—signs met with *only* in the insane, and physical signs of brain disease met with in the sane as well as in the insane,

Let us take first the cranial nerves and the nerve tracts from the nucleus to the cortex:

1. Perversions of smell in delusional insanity, indicating aberrant functioning or faulty associations of highest centres, probably in gyrus fornicatus.

Blunting of sense of smell in dementia, indicating defect from highest centre downwards.

2. Perversions of the sense of sight in delusional insanities indicating defects or faulty associational paths in highest visual centres; marginal convolutions.

Hemianopias in post-hemiplegic insanities, and in insanities, associated with unilateral gross lesions, indicating disease in occipital lobes or lower tracts.

General failure of visual acuteness, colour sense, etc., most marked in dements; general failure of visual nerve-paths.

(Word and mind blindness in cases of mania and dementia, failure of cortex around marginal convolutions.)

3. Recurrent and temporary palsies and spasms; mydriasis (unilateral), ptosis, squint, myosis, and retraction of eyelids (upper), indicative of functional disturbance in the third nucleus or any of its component parts. Seen chiefly in manias.

4. Squint (oblique), occasionally with maniacal excitement.

5. In dementia, general blunting of sensory processes connected with fifth nerve.

6. Internal squint common in mania; usually due to temporary weakness of one external rectus; defect of sixth nerve or part of nucleus.

7. Facial spasm and weakness; asymmetry of frontal muscles, indicative of defects in seventh nucleus.

8. Auditory hallucinations and delusions; in most cases probably defect, associational or otherwise, of highest centres: temporo-sphenoidal lobes.

Deafness (general) in dementia. Word deafness and amnesic aphasia in chronic mania, dementia, etc., failure (extensive) in temporo-sphenoidal lobes.

Vago-glosso pharyngeal nerve.—Tremor and deviation of tongue—perhaps a central defect.

Spinal accessory nerve.—Shallow, slow respiration without emotional variation, in dementia.

Lack of expectoration power—of laryngeal and palatal reflexes—in advanced dementia.

Sympathetic system (cervical ganglia). Paralytic myosis; unilateral and bilateral flushings of face; unilateral sweatings; seen in various forms of mania, and especially in epileptics.

Sensory tracts.—Repeated observations have shown sensation, and so necessarily sensory processes, those of touch, heat, cold, and pain, to be blunted in melancholia, more so in mania, more in dementia; we know them to be abolished in coma.

Spinal reflexes we find, like all nervous processes, sluggish in melancholia, in mania exaggerated from loss of cerebral inhibition, and in dementia almost in abeyance.

Movements; functions of the motor tract.—As indicating disturbance of the highest (motor) level, we have—in melancholia—a difficulty of imitating new movements; in cases of deeper dissolution a failure of execution of higher movements; in advanced and acute mania a failure of all complex movements other than those purely automatic; in dementia a restriction of movements to a few well-organised (voluntary) movements.

Looking at *muscular* states we have, indicative of general nerve failure, the loss of tone in melancholia; indicative of greater failure of higher levels, the low level "reflex" tone of mania. In other cases we have more complicated conditions

indicative of varying depths of dissolution; the katatonic, cataleptic rigidity of some cases, the total paralysis and flabbiness of coma.

The general wasting of some cases of insanity doubtless means cord invasion (anterior horns), just as the greyness of hair, skin conditions, and bedsores indicate disturbance of what are called trophic centres. All these because general are less obvious than if partial.

We have sluggish peristalsis in melancholia, and sphincter relaxation in mania and dementia—doubtless dependent on failure of the appropriate cord centres, just as priapism in mania is no less a sign of cord activity (morbid, unfettered) than when the spine is fractured or we have a myelitis.

Certain other signs must specially be mentioned.

We are inclined to connect a feeble, small-volumed pulse—that of poor innervation—with melancholia, a bounding, dilated one—that of diminished inhibition—with mania, one unaltered by emotion with dementia. So, too, one is inclined to connect a sluggish medium pupil with melancholia, one in which there is spasmodic myosis or mydriasis with mania—again the unvarying pupil with dementia.

May I now venture to anticipate some objections and criticisms?

One may be told that these "paralyses" of insanity are not the paralyses of the hospital ward.

Certainly, there is a difference; the hemiplegic has will, but no power. The comatose general paralytic and, in less measure, the dement, has neither will nor power. That in mania the paralyses are transient is true enough; in other cases it is, in fact, the universality of the paresis that prevents us seeing it.

Again, it may be said that many of these signs that have been mentioned are not "paralyses" at all; that an internal squint of a maniac or a smoothed left forehead is not due to paralysis of a sixth or seventh nerve, but to excessive action of a third nerve or of the opposite seventh. It may be; but in any case there is localised disturbance of brain function. The detail matters little; the real point is that these things are evidence of brain disease, even though the disease be purely functional. Perhaps some one will see that all this is obvious; that one flogs a dead horse.

My only answer is that in no text-book that I know of are the physical signs of insanity set forth or mentioned as evidence of brain disease. Perhaps it is a question of description and expression; still the proper expression of facts is surely a matter of importance.

We have to-day in medicine, and in our speciality in particular, a vast and unwieldy accumulation of facts. It seems to be forgotten that the proposition of hypothesis is a necessary part of induction, and that without theory there is no useful observation. Let us be unafraid then of cultivating a wholesome imagination, corrected by, and not in opposition to, observed facts.

On some of the Rarer Skin Diseases affecting the Insane.

By THEO. B. HYSLOP, M.D., Medical Superintendent, Bethlem Royal Hospital; Lecturer on Mental Diseases, St. Mary's Hospital; Demonstrator of Psychology, Guy's Hospital.

IT would be quite impossible during the few minutes at my disposal to deal in an exhaustive manner with the numberless varieties of skin affections met with in asylum practice. I have therefore selected from an immense mass of material a few of the rarer affections, and shall deal with them in such a way as to call for your experiences and criticisms rather than make any personal attempt to lay down the law with regard to any of them.

While fully recognising that some skin diseases may be classed among the neuroses, I believe that several writers on this subject have classed as neuroses diseases which are not more prevalent among the insane than the sane, and which on inquiry have no distinct relationship or evidence of neurotic origin. All asylum physicians are familiar with the brown muddy tints in mania, the cracked and scurfy conditions in melancholia, hypochondriasis, and stupor; also the brown discoloration in general paralysis somewhat suggestive of Addison's disease. Attention has also been directed to pallor, leaden hues, mottlings; the wine-coloured skin of dements; the semi-

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