the asylum show that he continued very suspicious, thought that his food was poisoned, and was aurally hallucinated. His bodily health improved under anti-syphilitic treatment. His eyesight continued impaired as on admission. He was regarded as suffering from a dementing psychosis —a paranoid type of alcoholic dementia—notwithstanding that there was strong proof that he feigned insanity at his trial. Evidence of simulation cumulated steadily, however, and he was returned to the State prison in June, 1906, as "not now insane" and "a clever malingerer," after being under observation in the asylum for nearly eleven months. In the following October, he made a feeble attempt to hang himself with a sheet in his prison cell. In August of the following year, he was re-committed to the State asylum, the medical certificate stating, among other things, that he had shown no signs of being clever at anything, and was considered to be an imbecile. He was much emaciated on his re-admission. He volunteered the information that he was tubercular and should have been sent to the prison camp for such cases. After his re-admission to the asylum, he gave up fasting and gained 23 lb. in weight in three months. He asked for work, and took care of the patients' clothes in the hospital ward for two months as intelligently as any paid employee.

To summarise this case, when first in prison his symptoms all pointed to gross stupidity, imbecility, or mental deficiency. On his admission to the asylum at first he appeared very stupid or deeply demented. Later his symptoms shifted to those of a very delusional paranoiac. When he was returned to the prison he again apparently became very stupid. When he was returned to the asylum, his stupidity all vanished, and he again became a full-fledged paranoiac. As Drew points out it seems unfortunate for the diagnosis of imbecility that it is a constitutional and fairly constant state.

Although he believes that all the symptoms mentioned in the commitment of J. H— were feigned, yet the question of his entire responsibility is still an open one. He seems so much of a degenerate, or deviate, and so difficult to care for in prison, that the author thinks that the ends of justice and humanity may as well be served by making the asylum his permanent home. A. W. WILCOX.

Mental Claudication [*Claudicazione Cerebrale Psichica*]. (*Riv. di Pat. Nerv. e Ment., vol. xviii, fasc.* 7.) *Benigni, P. F.*

Charcot, first in 1858 and subsequently in 1887, originated the idea of intermittent claudication in man, due, as in the horse, to obliterative arterial lesions of the inferior limbs. Many cases of the condition due to progressive endocarditis have since been published, and the phenomena are in all similar. A characteristic and essential feature is the temporary cessation of function in the affected part (limb).

Recently the classical opinion of claudication has been added to by extending its physio-pathological significance to special functions of special organs or systems, and the view of a visceral claudication has thus been created.

In 1866 Potain described the crises of cardiac ischæmia to be due to stenosis of the coronary arteries; he advanced the new and complete theory that angina pectoris was a legacy of ischæmia of the myocardium.

Grocco, in 1892, called attention to cases of true renal disorderliness with oscillation of the amount of urea excreted, compared with the amount of albuminuria, from day to day. He pointed out that there was a true intermittent renal claudication, which frequently preceded the development of chronic nephritis.

Grasset, in 1890, was the first to draw attention to the effect of claudication on the nervous centres, and was of opinion that intermittent symptomatic claudication was the result of arterio-sclerosis, and could be manifested in each of the constituent parts of the central nervous system. Intermittent claudication of the brain was studied by Grasset from the year 1904. This variety manifests itself, according to him, as an assemblage of symptoms, of which the principle are amnesia, intellectual fatigue and aphasia. In support of his contention he cites the case of a nephritic individual who suddenly had tingling sensations in one hand, and then, without loss of consciousness, became aphasic, not finding words to express his thoughts; after several hours these disturbances disappeared. He describes the cause of these symptoms as a passing insufficiency of the circulation of the third left frontal convolution. As a result of his researches Grasset was of opinion that there was a true cerebral claudication related to spasm of the arterioles of the region of Broca. At the same time he indicates very transiently a mental form of cerebral claudication which may be produced by periods of mental strain after excesses of intellectual work, and is an expression of cerebral fatigue.

Dr. Benigni, in this paper, treats in detail of the points he considers of importance in the pathogenesis and anatomical basis of the phenomena of intermittent mental cerebral claudication. The pathogenesis is often ascribed to transitory ischæmia through slackening or temporary suppression of the circulation, and is a result of the organ not receiving sufficient vital nourishment, and thus lacking in capacity for daily work.

The pathological anatomy of the causal fact, *viz.*, arterio-sclerosis, does not merit special distinction. It may be of inflammatory or of degenerative origin, and identical in the vessels of the brain and in those of the limbs.

Writing of the pathogenesis Benigni is of opinion that the principal cause of the phenomena, while in the circulatory system, does not occur without inhibitory central impulses. If, for instance, an important vessel in a limb is closed the abolition of movement is not instantaneous but gradual. If the collateral circulation is established the abolition of movement disappears. If, on the other hand, the most important nervous conduction is interrupted the paralysis is instantaneous and complete and cannot be compensated by collateral circulation. In the region of the nervous system it is known that the function of a given part is more under the control of nervous action than under the dominion of the circulation. It is a well-known fact that the nervous centres and nervous ramifications can temporarily functionate without nutriment; for instance, the heart of a frog, after removal from the body, pulsates through the intrinsic vitality of its own nervous ganglia, and the various segments of insects and reptiles survive, after they are detached from the body, by nervous impulse. Thus an organ

is not vitally affected until the inhibitory action of the nervous system is exhausted.

It may be possible to have the phenomena of claudication with perfect integrity of the circulation, and solely through nervous influence. Grasset recognises claudication as the result of simple spasm of a vessel, a spasm which expresses, in point of fact, a nervous origin. It may also occur when the arterial walls are quite healthy (nervous angio-spasm).

Grocco has demonstrated in two cases that angina pectoris may be due to an affection of the nerves of the heart, the blood-vessels being perfectly normal.

The peculiar characteristics of claudicational disturbances are their transitoriness, and their dependence on vascular lesions. When the phenomena are carried into the realm of mental disease there is generally a circulatory disturbance. The vessel diseases that are found in many forms of mental disease play an important part, direct and indirect, in the origin of functional diseases of the brain. These vessel diseases may follow as much from excess of function as from alterations of an acute or chronic kind. The integrity of the vessel-walls may be endangered by impurity of the blood, the result of intoxications and infectious diseases. These morbid causes may induce a precocious arterio-sclerosis, resulting in many conditions which are probably at the root of the explanation of claudicational phenomena in early and presenile cases, epochs in which are manifested the larger number of the morbid mental forms, progressive and transitory. Again, excitement of the vaso-centres of the bulb or medulla may induce vascular spasm or dilatation. Thus there exists a form of claudication (Roth) in which the circulatory disturbances of the trunk of the femoral nerve centres are a causal element. The claudicational phenomena have here been provoked, not by vessel alterations, but by mechanical disturbances, such as muscular contractions, erect postures, etc.

The chief causes of the anatomical lesions in mental cerebral claudication are the same as those forming the ætiology of many mental diseases, *e.g.*, alcohol, tobacco, lead and uric acid diathesis, among infectious maladies, syphilis, the toxins of typhus, scarlatina, influenza, etc., which are all of ætiological importance in inducing arterio-sclerosis. The affecting causes, and perhaps also the affections themselves, would be considered in such cases as the pathogenic agents.

The suspension of the functions of a claudicating organ ought to be necessarily of short duration, or it will last hours and days without prejudicing the significance of intermittent claudication.

Claudicational cerebral disturbances of mental order are met with frequently in the course of common mental maladies. Thus it is not rare to note in melancholic, plethoric, arterio-sclerotic individuals the crises of true amnesia associated with temporary hemiparesis, hemiæsthesia, cephalalgia, etc., with hallucinatory disturbances, mental confusion, more or less severe, depending on obliterating arteries of the cerebral vessels, having characteristics of true intermittence, and being therefore comparable to the intermittent claudication of Charcot. The greater part of the transitory cerebral manifestations in the gouty belong to the same class. Such manifestations are also of motor order, as in

nephritic delirium. Many forms or episodes of mental alienation, according to Dieulafoy—acute delirium, mania, lipemania, etc.—can occur as passing manifestations of lesions of the cerebral vessels. According to some authors these transitory mental phenomena are ascribed mainly to toxic elements; others again hold that the toxic material injures first the cerebral vessels and causes intermittent manifestation, the result of true intermittent claudication.

Cerebral claudication may explain the episodes of confusion with psycho-motor agitation of short and rapid duration in persons who have recovered over months or years.

In progressive paralysis how are the crises to be interpreted which are frequent at the beginning and in the course of the disease, *viz.*, epileptic and apoplectiform attacks with hemiplegia, hemiparesis, or monoparesis of short duration, temporary motor aphasia, congestions, vertiginous attacks, all symptoms of a transitory order; the states of mental confusion that arise suddenly, and are more or less profound, and which end with the same rapidity as some of the motor affections with which they are generally accompanied; the sudden impulses on account of which the patient is thrown headlong into threats or fears and verbigeration with chaotic delirium and complete disorientation, with loss of consciousness, in short, true mental arrests with complete loss of consciousness that are accompanied by true motor attacks? All these mental disturbances have the special characteristics of a more or less sudden rise and sudden recovery. Again, in senile dementia we find true confusional attacks—fleeting dreams—in which the patients become so disorientated as even to lose the correct idea of personality. With these confused states we can associate serious illusions, motor agitations, anxiety and fright. Such states remain for many hours—even for more than a whole day; they clear up with a certain rapidity, and the patients regain their usual lucidity.

The cerebral vessels in senile dements show evident atheromatous alteration in the large and small arteries, hence the circulatory disturbances can be easily initiated, and carry with them intermittent disturbances of a functional nature. This happens much more easily in brains already weakened by an atrophic process of the nervous elements.

In arterio-sclerosis mental phenomena are noticed, often transitory, such as delirium, more or less severe, depressed or agitated psycho-motor states, with impulsiveness. They are of short duration, and the patient becomes more or less apathetic and demented.

Benigni is of opinion that not only the diminution of the afflux of blood to an organ, but any transitory disturbance of the cerebral circulation, as the increase of bloody effusion, is able to cause an intermittent claudication of the organ.

Some of the acute mental disorders noticed in dementia præcox, as sensory frenzies, the rapt condition of certain melancholic states, and perhaps also certain states of epilepsy, can be compared with the phenomena of mental claudication. We know, however, that cerebral sclerosis is often found in youth, when toxic or infective causes irritate, and one or other of the many causes capable of bringing about ischæmia may be present, whence it is possible that these emotional states and toxic blood conditions may cause cerebral angio-spasm, and the forms

of mental disturbance already noted. In the course of dementia præcox, sudden fleeting episodes of mental confusion with agitation may arise, on account of which patients, at first tranquil and apathetic, are thrown into dangerous or fearful states, in which they break furniture, tear up clothing, and are given over to a lively emotion, shouting words or incoherent phrases, and refusing food. They then rapidly calm down, without knowing anything of their sudden agitation, or they say that their confusion was the result of a sudden fearful sight that they felt like a blow on the head, although they can record little or nothing of the occurrence.

Benigni's theory regarding mental cerebral claudication is that toxines enter the circulation suddenly, in greater quantities than usual, and are able to act directly or indirectly, through their effect on nerve cells, on the vessel walls, causing angio-spasm or paresis. Should the vessel walls be diseased, as they generally are, the claudicational phenomena are easier of production. HAMILTON C. MARR.

The Power of Comprehension and Observation in Dementia Præcox [*Auffassungs- und Merkfähigkeit bei Dementia Præcox*]. (*Psychol. Arbeiten, published by Prof. Emil Kraepelin, vol. v, Part VIII.*) *Busch, A.*

The tests described by Dr. Busch where made at Heidelberg in 1903. Reis had already discovered that the power of comprehension in the paralytic was much more impaired than that of the hebephrenic, who often had normal comprehension—a discovery that is not surprising in view of the fact that the clinical picture of hebephrenia shows the powers of comprehension and observation to be very slightly disturbed in comparison to the impairment of will-power. On account of the fact that this side of the psychical change differed in some degree from the clinical picture in the group of patients suffering from dementia præcox, it seemed interesting to make the tests again, and, if possible, more fully.

Reis used the Cron-Kraepelin rotating drum for his tests, but this instrument has the disadvantage—especially in the case of dementia præcox patients—that the patients, being left to themselves to read off the writing, after a time are inclined to become so indifferent that their efforts cease altogether. For this reason it was thought expedient to use Finzi's shooting plate in conjunction with the rotating drum, an arrangement which had these advantages: (1) It was impossible for the patient to lapse into a state of indifference, for after each passing of the plate he had to state (*a*) whether he had read anything, (*b*) what he had read. (2) The apparatus does not place such a strain on the patient as the rotating drum, which requires constant attention for six or seven minutes.

A detailed description of the instruments will be found in the works of Cron-Kraepelin and Finzi. In the tests with the shooting plate the opening was 1·9 cm. and the length of time the plates were visible was 22 seconds. The duration of visibility in Finzi's own tests—he worked entirely with normal and educated people—was 16·7 seconds.

The tests were made on nineteen patients diagnosed as dementia præcox cases, and on six healthy persons, attendants in the institution.