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NERVOUS AND MENTAL DISTURBANCES
OF INFLUENZA.

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The historical background of influenza affords a hazy territory for research, conjecture, and speculation. From the days of Hippocrates epidemics have been written of which bear certain resemblances to the present day influenza. Even Homer has been said to have placed an early epidemic on record. Thomas Glass, of Exeter, in his description of the epidemic plague of 1775 calls attention to the plague of the Iliad as presenting features which convinced him of its identity with the influenza. Of the history of these early plagues it is not my purpose to write; this has been done fully well by hosts of students better qualified than myself, and scores of sources are at the beck and call of the historically interested. Hirsch, Webster, Zeviani, Conradi, Kusnekow and Herrman, and many others have traced these from the fifth century to the present time. Throughout this record one finds certain unmistakable indicia of implication of the nervous system, but it is apparently only from about the fourteenth century on, that the details are sufficiently recorded to enable the student of the history of nervous affections to take up the scent and feel that he is on certain ground.

In these early accounts we read of headaches and deliria as frequent accompaniments of this disease; notably in the accounts given by Kusnekow and Herrmann. Sauvages in his celebrated *Nosologie* adopted the classification of cephalitis epidemica for certain of these epidemic descriptions which have notably come to us, chiefly outlined in Mezèray's description of the epidemic of 1510.

Of the many opportunities for uncertainties in the differentiation of different contagious disorders it is not my purpose to speak. It is certain that many whooping cough cases were intermingled in these early epidemics. Polio myelitis is always a definite possibility. But apart from all of the historical intricacies of interpretation, the point to be emphasized is that nervous and mental complications or manifestations have been evident from the earliest times and it is becoming increasingly apparent that the influenza microorganism is one fraught with certain specific activities upon the nervous structures.

One of the features that I wish to bring out in this rather hasty review is that many of the factors which have contributed to the dangers of influenza are based upon this predilection for certain specific nerve structures. Before attempting a generalization, however, with reference to a specific activity upon certain nervous elements, a review of the more general clinical manifestations of nervous disturbances seems advisable.

A great number of these nervous manifestations have come under personal observation in private, hospital, and dispensary practice. Their detailed anamnestic consideration would unduly extend this summary. The extremely extensive literature, which, beginning with the epidemic of 1783, has been recorded with each epidemic in voluminous proportions, contains an extremely rich and varied collection of a vast number of syndromes attributable to the influenza bacillus. The epidemic of 1889 gave rise to several thousand literary productions of all kinds, those bearing on the nervous system alone numbering at the very least several hundred.

I am aware of the fact that a number of observers have claimed that the influence of influenza upon the nervous system has been greatly exaggerated by those who have emphasized this relationship. In fact we find it recorded, in 1837, that Broussais wrote that "influenza itself was a

creation of people without a sou, and of doctors without clients, who having nothing better to do are amused to create this rigamarole." Thus for Broussais, and many others—and we have heard the same expression of opinion at the present time—there was no such thing as influenza although its malign influence and singular severity, especially upon the nervous system, had been emphasized for centuries. Such is the usual forgetfulness of the present for the past.

There can be no exaggeration of the countless number of facts that indubitably attest the enormous significance of the causal relationship between influenza and diseases of the nervous system. Since 1889—when more exact methods of diagnosis, neurological, bacteriological, and cytological have been more widely employed—the actual presence of the influenza bacillus, either in pure or mixed culture or in section, and by other methods of definite identification have more and more aided the diagnosis of nervous syndromes and have helped to clear the way to a more valuable therapy. Pfuhl, Pflüger, Nauwerk, and scores of others have isolated the organisms in various neurological syndromes.

In practically all of the conditions which shall be here mentioned the etiological relationship has been clearly established by different observers, either by direct observance of the organism or by a rigid logical analysis. Snap diagnoses have occurred and will continue to occur, and during an epidemic of influenza it will not infrequently happen that a superficial study of a nervous disturbance may be taken as due to influenza and thus permit a different serious etiological factor to pass by undetected. This may happen, and is particularly unfortunate in the case of syphilis or a tumor for example.

With these introductory remarks we may take up the consideration of the more widely observed syndromes.

Cranial nerves.—Olfactory: Disturbances of smell are frequent and anosmia is a widespread early symptom. Whether arising from the pressure of the swollen mucous membrane or as a direct response to toxic action, cannot always be determined. In certain patients the loss of smell persists for a long period after recovery from the acute effects of the infection. A few instances of anosmia under personal observation have set in in from four to five days or as late as ten days, after the onset of the acute symptoms. These have not been isolated happenings, as loss of taste was also present in one and loss of hearing an accessory symptom in another patient. Zwaardemaker (2), Bossers, Bardt, Dippe, and others have reported similar cases. Olfactory hallucinations, presumably of peripheral origin are also known (Bardt).

Optic nerve.—Optic neuritis, while a comparatively rare affection, is nevertheless frequent enough to demand serious consideration. Different neurones are involved. Thus retinitis results from involvement of the receptors, giving rise to partial or complete blindness, or more frequently to scotomata. Acute axial neuritis with its characteristic central scotomata is met with. It usually recovers. The most frequent types in my experience are the interstitial and diffuse forms of optic neuritis, the so called retrobulbar neuritis of most textbooks. In interstitial neuritis there results a limitation in the visual fields from peripheral scotomata, but many patients are unaware of the reduction in vision until definite tests are made. Influenza plays a very important rôle in the production of diffuse optic neuritis, here being almost as important a producing cause for this type as syphilis. There is a large literature bearing on this extremely grave disorder. Willbrand and Saenger have collected this in their great monograph. Post-chiasmal involvement of the optic tract may be seen in influenzal meningitis, and rare quadrant hemianopsias may be observed in occipital lobe involve-

ments, as in serous meningitis and in abscess, which latter may result from a pure or mixed infection.

Ocular palsies.—Third, fourth, and sixth nerve involvements, belong to some of the commonest of the many palsies which accompany the influenzal toxemia. They may occur as isolated palsies or are found in combination, and may be accompanied by more widespread involvement of the bulbar nuclei or peripheral neurones of the cranial nerves. The peripheral types are the better understood and also offer the better prognosis. Isolated external rectus palsy as a result of this toxemia I have encountered in a number of instances. It, as well as the more complex or complete types, is frequently preceded by a severe eyeball neuralgia. Accommodation palsies have also been seen by a score of observers, occurring as an isolated event or in combination with external ocular palsies. From a strictly neurological standpoint there is nothing pathognomonic in these ocular palsies by which they may be separated from palsies of other etiology. The prognosis is usually better than the syphilitic, typhoid, diphtheritic, poliomyelitic, or metallic ocular neuritides.

Combinations of ocular palsies, external and internal, with paralyzes of the pharyngeal pillars, or of certain of the laryngeal muscles occur. I have seen two or three such patients, in whom the resemblance to a diphtheritic palsy was very striking. This latter cause was excluded by laboratory tests. Joachim, Jankau and Uhthoff, Heymann, Krakauer, Faye, Fukula, Vaschide, Pflüger, Albrand, Stower, Greef, Bergermeister, Landolt, Weichselbaum, Sattler, Frank, Guttmann, and others, have reported cases.

Trigeminus.—Neuralgia of the fifth nerve is one of the most frequent and painful disturbances in influenza. In our dispensary work it is frequently observed. I have seen it less often in private practice, as many of these patients go to their dentists in the belief that the trouble is a dental one.

Trigeminal zoster I have also seen, and some patients have a very severe trigeminal zoster as a symptom of an influenza. Motor palsies of the fifth nerve I have never seen.

Facial nerve.—Palsy of the seventh nerve I have frequently observed when the general symptoms of influenza have been present. It may or may not be associated with an otitis. As not infrequently happens, the neuritis may be severe and yet the general symptoms of the influenza be quite mild, that is speaking from the the usual standpoint—that the nasal and respiratory symptoms are taken as a general criterion of the severity of the disease. I am disposed to believe that this is a great mistake, for many extremely severe cases of influenza go through almost to death with very minor nasal or respiratory indications. It has seemed to me that when the toxin seems to localize its activities in one type of tissue it has a tendency to limit itself there. Thus most of the neuritides that I have observed—and this applies to the neuritides of spinal distribution as well as of cranial localization, including also zoster cases—have occurred in patients in whom bronchial, intestinal, or nasal manifestations have been extremely mild. Thus to deny the presence of an influenza in the absence of these symptoms in a severe type is not good sense. In the epidemic of 1889 and 1890 I recall a particularly striking incident in one family in which one patient died of an influenzal pneumonia, another with a slight bronchitis only, died with a severe zoster, and a third with no influenzal symptoms in the ordinary sense had an external and internal ophthalmoplegia. In two patients seen recently, the mother had a severe old fashioned influenza and was not under my care, but two daughters had a zoster and a mild chorea, respectively, but with only slight indications of the influenza. Thus I have expected not to find the severe neuritides in the severe bronchial cases. It may be that the internist sees these cases and I do not. When the influenza has been mild and

palsies have been present they are referred to the neurologist as nervous cases. This experience as that of others as well would seem to be indicated in the reading of the full historical accounts of former epidemics. Even in those way back in the early centuries, we note that different observers have spoken of, "this epidemic as being noted for the large number of mental cases"; "this epidemic has been noted for the large number of pneumonia cases"; "this epidemic runs to intestinal types," etc., etc. Thus, in the epidemic of 1781, it is recorded that there were great numbers of very severe head symptoms, "cruel pains," and the term "cephalitis epidemica" was coined and used as a standard of classification by Sauvages, as has been referred to. Thus there are direct indications at least that a certain specificity of tissue type involvement may be the usual thing. Complete analyses, which are rarely ever possible, may show this to be a faulty generalization, for there are by no means few instances when diffuse and severe neuritides are known to have occurred with severe pneumonic types. Thus a severe facial cervical zoster type accompanied a severe and fatal exudative edematous pneumonia in one of my patients.

Since the general problem of the determination of localization of disease processes is still so obscure, the generalization is left for subsequent modification and criticism. When one patient with a mild influenza develops a zoster, another a mild optic neuritis, and still a third a tachycardia, diffuse perspiration, tremor, and other symptoms of a vagotonic exophthalmic goitre with other adenopathies, all three resulting from a similar toxic producing agent, it becomes an interesting problem of individual constitutional variation in organ susceptibility—a problem which has been but little touched upon but is of paramount importance, not only in the reactivity to the influenza toxin, but to other types of infectious disease, syphilis for instance. Among others, Potzl, Bartels, Paltauf, and Adler in his *Inferiority of Organs and Their Psychical Com-*

pendent, have broken ground in this fascinating realm for investigation.

Eighth nerve.—The marked tinnitus which is an almost invariable symptom of the early stages is a mixed auditory nerve and physical exudative phenomenon. When the involvement of the auditory nerve is more persistent, deafness results. Vertigoes and nystagmus of vestibular origin are also reported.

Ninth nerve.—The glossopharyngeal palsies have been less thoroughly studied, although the throat complications of the grippe are very widespread. Hoarseness with weakness in swallowing and in phonation are very frequent mild accompaniments. They are conditioned by disturbances which are in part of neurological functioning and in part of physical interference. The great laxness in the tonus of these muscles is directly due to the disturbance in the vegetative control which is so marked a feature of the entire poisoning that it will be made the subject of special discussion.

Tenth nerve.—As will be brought out later, this vegetative nerve disturbance is a fundamental underlying condition in influenza poisoning, and for this reason the symptomatology more directly connected with the pneumogastric will be merely touched upon. These are masked, as it were, beneath the more striking internist situation of an edematous pneumonic flooding—which is so frequently complicated by the effects of the activities of other microorganisms, thus altering the purer (?) picture of a true influenzal vasomotor paresis of the pulmonic vessels with edema, bloody extravasation, etc. I shall pass on to the more usually thought of neurological phenomena, saying at this time only that the peculiar character of the grippe edematous flooding has been so strikingly different as to have attracted attention and record for several centuries and must be elucidated ultimately in the light of the vegetative functions of the vagus (autonomic) and sympathetic systems respectively.

Hypoglossal nerve.—Disturbances of muscular control of the tongue are few, and unilateral atrophy of this structure, while recorded (Leyden), is a rarity. Taste disturbances of a mixed nature are a frequent finding and the universal disgust for food, which is a mixed psychical and cranial nerve disturbance, is too well known to demand special attention (Frey-Laache).

Spinal distribution.—Neuralgias, neuritides, with palsies and zosteres of every regional distribution—central as well as peripheral—have been seen, either as isolated localizations or as widespread and serious polyneuritides. Even the most severe grades of multiple neuritis, grouped under the symbol of Landry's paralysis, are known. Many of these isolated neuritides with their consequent palsies resemble poliomyelitis cases closely and a clinical differentiation is at times extremely difficult. Poliomyelitis and influenza have often been associated; indeed there are not wanting those who have claimed them as identical. There is a rich Scandinavian literature upon this question, but with our present knowledge this viewpoint seems untenable.

I have seen comparatively few spinal neuritides and a few zosteres of influenzal causation, yet they are among the best documented cases in the neurological literature, and a passing word may be said concerning the more frequently observed types.

Neuralgias are extremely frequent. In some epidemics nearly fifty per cent. of those affected have had severe neuralgias. Supraorbital and infraorbital localizations are among the most frequent, and seem associated with the near lying sinus engorgements. Trigeminal neuralgia has been mentioned. It is occasionally very intense and chronic. Intercostal neuralgia is frequent and is to be separated from the extremely frequently felt sense of constriction of the chest. This latter is usually a vagus sympathetic syndrome, as has been noted by Kinnicutt, Edgren, Braken-

ridge, etc. Scapulo-humeral and brachial neuralgic types are the most frequent of the upper extremity neuralgias. Sciatic neuralgias and neuritides are extremely common. The entire distribution is rarely involved in a neuritis sufficiently severe to develop a palsy, although this sometimes happens, and even bilateral sciatic palsies are known.

Polyneuritis.—A rare, but nevertheless, a most important series of polyneuritis cases are on record. Personally I have happened to see but two cases in private practice when the causal relationship could be carefully investigated. A number of suspicious cases have been seen in my City Hospital service, but the etiological factor had to be surmised rather than proven. In one of my cases a complication with a possible rabies polyneuritis of the Landry type obscured the picture. These have been written concerning these polyneuritis cases (Diemer), which have entered the literature as definite since Dumènil called attention to them in 1866.

The multiple neuritis is apt to come on in the period of convalescence, in from ten days to three weeks, more or less like the postdiphtheritic palsies to which they have often been compared, and, at times, attributed. Grippe polyneuritis is preeminently a motor neuritis although even severe neuralgic pains may precede its development. The lower extremities are more severely involved as a rule than the upper, and one side of the body is apt to be more affected. The muscles of the back seem to be spared. The cranial nerves are involved with the severe cases, even the pneumogastric. The diaphragm has been paralyzed (Bonnet). The distal muscles are more involved and the extensors more than the flexors. Central vegetative disturbances are not marked, though at times present, and the secretory and trophic alterations minor. The atrophy which follows seems to follow the peripheral spinal type and the prognosis is usually good. In marked contrast with polyneuritis of

alcoholic etiology, Korsakow's psychotic states are rarely observed.

Some help in the differential diagnosis from a palsy of central origin, poliomyelitis, may be gained by a study of the sensory changes. In the polyneuritic types there are not infrequently changes in bony sensibility (see Williamson), and epicritic heat and cold tests reveal differences; light touch and the sense of position also may be involved. These signs are usually entirely absent in the poliomyelitides, unless the poliomyelitis virus has produced a diffuse and severe transverse myelitis, or more rarely a neuritis, but even here a careful sensory examination will tend to show that the alterations in sensibility follow out a peripheral or a segmental metameric formulæ (Head) respectively. In the severe types which follow the so called Landry picture it is doubtful if a differential diagnosis can be established without laboratory aids.

The autopsied case of v. Leyden (1893) is one of the first cases of influenzal Landry's polyneuritis on record. Bernhardt, Eisenlohr, Buzzard, Savage, Holmen, Westphalen, and others reported early cases in the 1890 epidemic. Senator in the discussion of v. Leyden's patient called attention to an important point in the pathology of this affection which will be taken up later—namely, not only the tendency to edematous infiltration but to the minute hemorrhagic infiltration or extravasation in the nervous tissues. This is not a massive hemorrhage and in certain patients with influenza dying of pneumonia Foa has described this type of infiltration and edematous swelling in the cord structures.

Space does not permit entering into a discussion of the respective parts played by peripheral and central changes in this rare but very sinister type of disturbance. V. Leyden, Bailey, Ewing, and others have discussed the polyneuritic aspect, while Bing, Van Gehuchten, Giovanni, Raymond, Strümpell, Medin, and others have taken it up from the aspect of poliomyelitis.

An interesting polyneuritic syndrome is that of *pseudotabes*. These cases have been described as acute ataxias by some observers; I have seen a few only. The absence of positive Wassermann signs has aided in the diagnosis. Dejerine has discussed them fully. Putnam, Sottas, Livierato, and others have described them. Ataxia, Romberg's sign, sensibility changes, and loss of knee jerks are the usual symptoms. The majority of the influenza polyneuritides are motor, at least we have Bosser's statement to the effect that they are exclusively motor, but there are many observations showing that the sensory neurones may at times be deeply involved. Pressure over the nerve trunks in these ataxic cases is usually painful. The Lasègue sign is usually positive, whereas in the true tabetic, nerve tenderness and the Lasègue signs are usually absent. Epicritic sensibility changes are more apt to be present in these polyneuritic *pseudotabes* cases, and the distribution of the sensory modifications is apt to be peripheral rather than radicular. A few cases of radiculitis from influenza are on record (Feinberg).

Spinal cord changes.—Myelitides: Not only are extensive changes in the peripheral nerves possible symptoms of influenza, even in what appear to be mild cases, but active and severe involvements of the spinal pathways and of the spinal meninges take place. The extremely severe types of ascending myelitis—related to and, perhaps, indistinguishable from the severe ascending neuritides—Landry's type, have already been discussed. They are rare. Dorsolumbar myelitis resulting in a flaccid or a spastic paraplegic picture are more often encountered. It is, however, the most frequently observed type of influenzal myelitis. I recall but one case seen in private practice. The grippe myelitides are apt to be mild, however, and often clear up very satisfactorily. The onset is apt to be slow, the symptoms developing progressively. This is more true of the spastic types. The flaccid types usually have a more furibund aspect; several hospital cases

seemed to show this variation. Varying grades of involvement are to be expected.

In the more distinctly hemorrhagic cases the onset is more acute and there is a tendency to the formation of disseminated foci. This develops, not only in the observation of the spinal symptoms, but is also seen in the occurrence of other focal involvements in other parts of the cerebrospinal axis. Thus optic neuritis and ophthalmoplegias have occurred with the spinal myelitis syndromes. Two personally seen patients with the disseminated type resembled, what is often termed, acute multiple sclerosis. Bramwell, Maixner, Marburg, Massalongo, Nolde, Oppenheim (six cases), Rendu, and others have reported similar findings. Some of these patients go on to recovery and others run a more chronic course and are often viewed as true cases of multiple sclerosis of the so called secondary type. The influenzal myelitides usually have a good prognosis; fatal cases, however, are on record and would probably be more often reported here in this country were it not for the many obstructive conditions surrounding opportunities for postmortem observation.

Eulenberg and Determann have reported curiosities in the form of spinal foci which have caused the Brown-Séquard syndrome. Capillary exudations and minute bleedings occurred in small areas involving but one half of the spinal cord. In Determann's case tetany also was present and recorded.

Before leaving the spinal cord syndromes, and particularly the differentiation of Landry's paralysis, poliomyelitis, etc.—questions which are of much importance neurologically and which are still in need of more complete clarification—a word may be said concerning the attitude of which the Scandinavian, Borgström, is the chief representative. He holds that there is a great polymorphism in the group of organisms which cause influenza and poliomyelitis. He thinks they are interchangeable, and has entered the polemic field chiefly against Wickmann in an attempt to prove, on the basis of the personally ob-

served cases in Sweden, that influenza and poliomyelitis are the same disease. Wickmann's so called abortive cases, he maintains, are certainly to be included in this conception. His analyses, however, are filled with faulty presuppositions, his neurological technic in examination, particularly of the vegetative nervous system and of the sensory nervous system, is so faulty that it is evident that he oversteps the mark. At the same time it has been considered worth while at this time to dwell for a moment on the fact that severe spinal cord disease, while a particularly rare form in influenza involvement, nevertheless is one of the things that does happen, and that the poliomyelitic form is a possibility. The pathological differentiation of the types of lesion is still to be decisively pronounced upon. A great deal is known of the pathology of poliomyelitis; very little of that of influenza. In certain cases of influenza dying of pneumonic complications changes in the spinal cord have been observed.

Brain involvements.—From the very earliest times the cerebral involvements in influenza have been noted. The almost universal headache, the frequent occurrence of delirium, with or without high fever, have seemed to accentuate the belief among nearly all of those who have had experience with influenza that the brain structures are involved early. For the most part it is true temporarily, for the headache, to speak of the most prominent conscious symptom, usually passes with the severe pains in other parts of the body within three or four days, but in some epidemics the cerebral involvements are very pronounced and extremely severe. In all the epidemics certain cerebral symptoms are present.

While the headache of influenza resembles in most respects that of a number of other infectious diseases, still it is characteristic enough to have earned a special title early in the science of nosology. Epidemic headache, cruel and severe, was the appellation given it by Sauvages. It is cruel; at

times it is fiendish, and three marked types are distinguishable. There is an early headache, which is primarily due to vegetative functional alterations in blood pressure, in the nutrition of the vegetative nervous structures of the trigeminus, particularly of the *nervi vasovasorum*. This seems more closely related to the reaction to the toxemia of the grippe organism. It is a headache which is usually all over the inside of the head, giving a sort of sense of internal explosion, as if the head would burst. The type is frequently spoken of as a congestive headache. A number of other toxins seem to induce a closely similar vegetative nerve reaction as an indication of the attempt at vascular control. At times this headache, still in the toxic anaphylactic functional realm, may be more sharply localized. This localization, frontal, may be associated with more severe local infectious signs, such as nasal and frontal sinus predominance; occipital and lateral, when the mastoid sinus is predominantly involved.

When an invasion of the meninges occurs by the Pfeiffer bacillus, the various localized or diffused mild or severe types of influenzal meningitis occur. The headache becomes usually more of a dull character, and following the type of meningitis, active more rarely, comatose, lethargic more frequently, the headache seems to run with the meningitis and is mingled with the general meningitic series of symptoms.

A third type is particularly interesting and important. I have seen a large number of postinfluenzal headaches of a particularly severe and protracted type. So intense and so prolonged have they been that they have come in consultation as possible brain tumors. The postinfluenzal neurasthenoid syndrome is not now under discussion. I am speaking of patients who have not been very sick with the influenza, save perhaps they have all shown an extremely intense reaction to the toxemia; they have been sick for the most part not

over two or three days. There has been, with these cases, a very severe general reaction with a marked sense of great illness. The sthenic reaction type has been characteristic. After recovery, which has been uneventful, they have developed a severe generalized or, more often, occipital headache. This has been peculiar, in that if the patient does nothing he may be free from pain, but the moment he attempts any labor, reading, writing, concentration of effort, the pain is so intense as to force him to desist all effort. Three such patients could not even write a letter or read a paragraph in a newspaper without the onset of the headache; otherwise they were in excellent health. While I am inclined to believe that behind this postinfluenzal headache situation possibly certain definite psychical components may have been present, the fact remains that the influenza brought the pain into the foreground of active consciousness. Its function I could not learn. These headaches have persisted from three to eight weeks and have all cleared up almost as quickly as they came.

Meningitis.—Spinal types as well as cerebral types are known. The bacteriological evidence is now beyond cavil, for the microorganism has frequently been obtained by lumbar puncture, cerebral puncture, blood culture, postmortem culture, and by staining methods in postmortem examinations. A great variability in grades of infection is known. The simple vascular preinfection stages have already been spoken of; these are usually the more benign types and recover soon. Possibly the severe headaches which have just been mentioned may represent serous meningeal types, without infection or with minimal localized infection. Serous meningitis, then, may be a possibility. I know of no definite proof of this for the only possible type which could be proved, i. e., the focal infectious type. One patient operated upon for possible brain tumor showed a focalized serous meningitis. The history of onset of the difficulty closely following a

severe influenza made this etiological factor a possibility, but culture experiments with the fluid were negative and as the patient still lives, the etiological factor is still uncertain.

Acute meningeal cases, found at all ages, more frequent apparently in childhood, especially in the milder type, may be of this congestive or hypertensive type with minimal focalized infection. A second degree of more serious involvement constitutes the suppurative meningitides of pure Pfeiffer type, or mixed with other microorganisms, notably the pneumococcus and streptococcus. The Pfeiffer microorganisms have been isolated, closely, following the discovery by Pfeiffer in 1889, by Pfühl, 1892; by Slavyk, 1898; by Trouillet et Esprit, Mao, 1903, and many others.

Influenzal meningitis differs little from other types of meningitis. It is usually an extremely severe disease and the differential diagnosis is difficult without lumbar puncture or blood culture. Grasty has called attention to a difference in the leucocyte count of influenzal meningitis stating it to run rarely above 15,000, while other purulent meningitides are apt to run as high as 30,000 to 40,000. Forbes and Snyder in a more recent study of leucocytes in influenza in general find an absence of hyperleucocytosis as a general feature of the disease, with or without any meningitis.

To the neurologist the meningeal and encephalic syndromes are still a very large grab bag, out of which, by careful clinical and laboratory observation, much may be chosen with certain degrees of definiteness. Still there are numberless patients, viewed in the large, who develop meningeal or encephalic syndromes of extremely perplexing characters.

In the epidemic of 1890 I was a hospital interne and my first personal and professional baptism was in the influenza epidemic of that year. Since then from time to time I have seen many of the syndromes which have been spoken of here. Occa-

sionally there has been presented a type which has received of late some special mention, in which it has not been certain whether one has to do with botulism (see English reports), poliomyelitis, or an unknown infectious disorder involving the structures of the midbrain. The French have been working at it as lethargic encephalitis and attention has been already directed to it here, when speaking of paralysis of the oculomotor nerves.

The type of disorder referred to has been present in Austria, England, Italy, and France and has been given several names. It is characterized by acute onset with chilliness, headache, and fever; nausea and vomiting are occasionally present. Then a series of symptoms develops in which great lethargy and cranial nerve palsies occur. The lethargy, at times spoken of as narcolepsy, is very profound. It may come on slowly with heavy eyelids—complicated by organic ptoses in the eyelids—and an irresistible torpor. The patient may be aroused, wake up, answer in responsive or irresponsible monosyllables and sink again into deep unconsciousness. The patient may not be waked up sufficiently to be fed, urination and defecation taking place in this deep stuporous state. Occasionally this is broken by nightmares or at times a muttering delirium. Death may ensue, the patient developing Cheyne-Stokes respiration and going out. In the patients who recover, which is the rule, the lethargy slowly diminishes and the patient comes to himself gradually.

The cranial nerve palsies are chiefly of the oculomotor group; either external, internal, or double ophthalmoplegias are observed. This paralysis is a nuclear palsy, solely motor, without the neuralgic pains spoken of under the head of the neuritides and oculomotor palsies. The cranial nerves affected are chiefly of the mesencephalic localization—third, fourth, sixth. The paralyzes are usually partial, dissociated and incomplete. Ptosis is usual; diplopia not uncommon; the pupillary disturbances rare, at

times very pronounced. Jacob and Hallez have noted transitory Argyll-Robertson signs. Paralyzes of accommodation are frequent (Harris).

Double facial palsy may occur; trigeminal, hypoglossal and glossopharyngeal palsies have been noted. Sensory changes may also occur, and other variable symptoms such as convulsive seizures, contractures, hyperesthesia, anesthesia, catatonic or cataleptic states. Sergent's white line is fairly constant. Although too few cases are recorded to give reliable statistics, the mortality seems to be fairly high. Sainton quotes thirty-five per cent. in the French series and twenty-five per cent. the English series. The severe type seems to be marked by great thermoregulatory disturbance. The fever mounts rapidly and does not fall. The signs of infection are very profound. Death takes place in from eight to twelve days. The subacute type shows a rapid rise in temperature, then it falls, and has an up and down course between 99° and 102° F. for some length of time—four weeks to two months. Lumbar puncture is usually negative, a fact of considerable importance in separating this disturbance from epidemic cerebrospinal meningitis. No signs of meningeal irritation are present and the steplike mode of progression so frequent in the meningoencephalitis of infectious origin is not present.

In the autopsied cases reported on by Sainton, Pierre Marie et Tretiakoff, and Caussade, attention is called by the first observer to the incongruity that exists between the severity of the symptoms and the paucity of the findings. We are here reminded again of Senator's suggestions respecting the minute characteristics of the changes in the cord in the myelitides, and certain cases of hemiplegia without visible signs—edemas probably—come to mind. Minute hemorrhagic suffusions or microscopical hemorrhages seem to mark the congested areas in the mesencephalic structures. Histologically the hemorrhagic suffusion is most marked. In Marie's cases degeneration of the cells of the locus niger was a

marked feature. The general character of the lesion is that of a polioencephalitis histologically undifferentiated from other types of polioencephalitis, not including the syphilitic or tuberculous or malarial types. Whether the influenza bacillus is able to cause this type of lethargic meningoencephalitis is still to be proven. The cases reported have all of the features of an acute infectious disease. In the early epidemics of 1889-1900 such case reports began to appear in the literature. Henry Young called it grippe catalepsy. Later studies of Longuet (1892), Wolf, Bozzali (1900) reported cases which were attributed to influenza. Thus far in the recent studies no definite organism has been reported.

Other types of encephalitis have been reported since 1890 involving not only the cerebrum, but the cerebellum as well. Guttermann (1900), Pfühl, (1892-1897), Nauwerk, and others have isolated the organism from the infected foci. A great diversity of clinical pictures has resulted from the many possibilities of such infectious foci. Abscess has been the terminator in some of the cases.

Influenza hemiplegia with or without aphasia has been, personally, the most frequently observed type of symptom in this field. The otologists undoubtedly observe the abscess cases from ear or mastoid extension, which are either purely influenzal or mixed infections. Monoplegias, choreas, epilepsies, and abscesses are among the possibilities which have been reported. Influenza, as providing the necessary upset to precipitate a cerebral softening in an arteriosclerotic of sixty to seventy years, has been not infrequent in my experience. These softenings have occurred in various parts of the brain and have given rise to a very diverse syndrome varying from the slightest types of motor contractures or loss of sensibility to the advanced softening of a terminal dementia. Aphasias and mental confusions have been not infrequent and have for the most part had a good prognosis.

Polioencephalitis superior, as a syndrome, is usually evidenced by the sudden appearance of a localized convulsion or, in young persons, by a series of generalized convulsions. The patients then may develop a mild delirium often with a pseudohysterical coloring, possibly with laughing or witticisms (frontal softening) or they develop coma, indicating deeper involvements of the cerebral structures of the midbrain (polioencephalitis inferior of Wernicke). These symptoms occur usually within the first week of the influenza, in the patients personally seen. The third day, usually marked by intense febrile states, 103° to 106° F., has been the chief day of invasion. There are almost invariably meningeal signs as well, *tache cerebrale* is frequent, goosefleshing and other severe pilomotor reactions, Sergent's white line. Kernig's sign is occasionally elicitable in the comatose state. Lumbar puncture or blood culture may reveal the influenza bacillus.

As has been stated the residual symptoms, should the patient clear up from his coma, will depend entirely upon the area or areas involved in the encephalic process which pathogenically is a greater or less functional disturbance consequent upon an edematous or hemorrhagic effusion. In a few patients who have been seen in consultation a mild euphoric silliness has been present. This has been combined with slight memory defects, tendency to punning, and mild anxiety states, difficulty in controlling the bladder and increased bowel activity. These have been mistaken at times for "hysteria," but they had none of the psychogenic conversion features which are essential for this diagnosis. The symptoms were not mental conversion symbols. They were direct results of a focalized lesion in the first or second frontal lobes, chiefly the left side—enlarged pupil, usually, and pilomotor and vasomotor anomalies of the same side—and their usually favorable prognosis is in no manner to be regarded as indicating a so called hysteria. Thus Grasset's and Rauzier's reported case has been cited in litera-

ture as hysterical or hysteroid. The essential symbolic features of this purely psychogenic psycho-neurosis were absent. It is better considered to be a multifocal meningoencephalitis with flexed contracted limbs, involuntary urination, hemianesthesia, analgesia, etc. A certain emphasis is laid upon this point because there is such a prevalent trend among the laity as well as among physicians to name a peculiar, bizarre and noisily inconsistent set of symptoms, especially when occurring in women, as hysteria. This a great mistake. A great many of the lethargic encephalitis patients which were also frequent in the epidemic of 1890 and called "Nona" or living death patients, have been called hysterical coma and have been foolishly converted into pincushions by over zealous and under informed investigators. To stick pins in an individual and when he says he does not feel it, or gives no evidence of feeling it, and then say—hysteria—is bumble puppy and not diagnosis.

Certain cataleptic encephalic states are occasionally observable in hospital practice. In very severe frontal involvement permanent impairment may result. These show as various dementing states, occurring in individuals between 60-70; losses of memory and other indicia of the sudden onset of a syndrome clinically indistinguishable from senile dementing types.

When the lesion involves the Rolandic areas various forms of monoplegia or hemiplegia result. These may be temporary or permanent. The general prognosis in influenzal monoplegias and hemiplegias is fair. Involvement of Broca's convolution of the left side produces a motor aphasia and implications of other speech zones may induce other aphasia types. I have seen several instances of these aphasias in the past ten years. Not only have the arteriosclerotics suffered but in a number of the patients with aphasias, which have been deemed influenzal, there has been no evident arteriosclerosis

as registered by eye ground, palpable arteries, kidney lesion or high blood pressure. This latter group has had an invariably better prognosis than the former.

I have seen no hemianopsias or instances of cortical blindness from occipital lobe encephalitis. They are known however. Harris has reported a patient with complete blindness of cortical origin which cleared up in two weeks.

The numerous complicated midbrain and medullary encephalitides giving rise to the nuclear palsies of the cranial nerves have already been discussed. From a topographical point of view the central types belong in this section.

Mental involvements. There is probably no other acute infectious disease which gives rise to, or results in so many diversified types of mental disturbance, ranging from the simplest fatigue states of a transitory nature to some of the severest defect mental conditions which may wipe away at a blow the entire mental life. Fortunately the tendency is towards the mildest and milder involvements, but the gamut of possibilities is indeed kaleidoscopic. This great diversity in syndromy is worthy of the closest scrutiny for it affords a very important research background bearing upon the complex dynamic interdependence of the health of the bodily organs and interference with the energy receptors, transformers and effectors. As has already been observed, there is a special affinity for the grip toxins whatever they may be chemically, for nervous structures. The special nervous structures which apparently handle the poisons with the greatest difficulty seem to be the sympathetic division of the vegetative neurons. As is well known functional balance of the metabolism is chiefly if not exclusively maintained by the vegetative nervous system. The functional metabolism of nervous structures themselves is likewise affected and fatigue is a preliminary warning in consciousness of threatened faulty adjustment. The fatigue threshold is

dangerously near consciousness because of the most universal of all affective goals, indolence. Indolence is ever ready to camouflage its real desires and by means of a conversion mechanism fatigue states arise from our conflict with indolence, which varies with every individual. Hence in those, and perhaps they are the majority, a slightly added weight by means at times of a minimal amount of metabolic imbalance from intoxication which throws up the danger semaphore (instinctive sense of wellbeing) the sense of fatigue is doubled or redoubled. Flight now is the psychological alternative as a protection mechanism. The robust and healthy stand up and fight and the victory is won. This robustness applies to mental rather than to physical robustness. Many of the muscularly most robust of mankind are worshippers at the shrine of Narcissus. They are strong for self aggrandizement. Hence they are mentally not healthy for mental health means the direction of one's aims towards socially valuable rather than individual goals. Right here one may see a partial answer to the problem which has disturbed the medical mind why so many of the apparently healthiest are so readily laid low by the influenza organism. Parenthetically, also some light may here be directed towards the valuable psychological attitude of the value of a universal muscular training for the preservation of one's nation, rather than the advantages to be gained by individual athletic gymnasium work for the limited, hence more Narcissistic and infantile glory of this school or that university or other exhibitionistic aim.

Those less healthy minded then unconsciously run away and the flight into a protective psychoneurosis or to a psychosis ensues. Right here may be seen I believe in its proper setting the whole vexed question which has been stated in so many different ways as to the influence of heredity, of neuropathic character, of the background, in short, of the individual.

Everybody—bar none—is by necessity, as a bit of living matter, constantly engaged in struggle.

Speaking mentally what we call neurotic, neuropathic psychopathic or what not, is only a vague way of attempting to embody the externally observable behavioristic features of that struggle by some diagnostic label. Because of the great diversity and complexity of the observable phenomena there results a great range in attempt to restrict these phenomena by static definition. Those whose conduct varies more from the average than others, to the good or bad, it may be mentioned it seems the differentiation is rarely made by the usual observer, are stigmatized as neurotic, neuropathic, etc. A stigmatization it might be observed which has much of the Pharisee attitude of self laudation about it. Neurotics, neuropathics, even some psychotics are capable and alone are the capable it may be added, to add to the store of the world's most precious possessions. The creative artists of the world are among those usually stigmatized neurotics, etc., but they are the ones who have successfully struggled with universal indolence and made something new. Other neurotics have laid down on the job and become the hoboes, the prostitutes, male and female, and the failures.

A static definition of neurotic means nothing; a dynamic definition of neurotic means increased or diminished capacity for new adaptations—which it is going to be, plus or minus, is always a question of fact for the individual and for the moment and for the particular situation.

So to return to our mutttons—the manner in which each individual is going to react to the grip virus is going to be determined by his dose and the way in which he has handled, or is handling, his conflicts. As these are two, or more, independent variables, the results, speaking mentally, are legion.

The most frequent of these are the various neurasthenic forms which may show as simple fatigue, involving attention, or myasthenic states, or a host of neurotic or fatigue medleys in the viscera. These influenzal neurasthenias occur with either severe

or with mild systemic signs of infection. There is for most patients an extraordinary myasthenia with great depression of spirits. In the majority of instances this clears up in from one to two weeks—in some after two or three days. But in a still strikingly large number of patients the residual neurasthenic fatigue is severe.

By neurasthenia is here meant the pure fatigue syndrome due solely to the toxemia alone or toxemia plus the emotional conflicts to which attention has already been directed.

Some mention has already been made of headaches. The persisting localized ones may be the results of serous meningitides as has been said. They may also be protective devices of the unconscious to prevent further disturbance to the individual forcing him to pay attention to his state of well being. The somatic instinctive sense of well being—in the healthy minded of our previous definition—is an excellent guide for conduct, and here the protracted headaches say "stop, look and listen." Such individuals are advised to rest and feed.

Of the other neurasthenic syndromes much may be said. There are many in which the fatigue is not the only symptom but in which various visceral neurotic disturbances persist. Thus in the skin localized or more or less generalized areas may persistently gooseflesh, or formication may come and go with every grinding noise, or sudden jar, or unaccustomed sight. A hair trigger localized vegetative unrest of the skin structures causes such minor accompaniments of the fatigue state. Or a similar mechanism in the blood vessels will bring about great chilliness, or marked cyanotic blueness—at times almost passing over into a Raynaud's syndrome, thus lending a certain support to the hypothesis that the vegetative nuclei in the cord may have been involved. Again there may be mild persistent edemas, or reddish mottlings of the skin, irregular erythemas, etc. Other visceral signs may be present such as digestive upsets, diarrheal at-

tacks, polyurias, icterides, etc. One might box the compass of the various viscera of the body and find one or two or a host of such mild disturbances of function in the influenzal aftermaths. The precise pathology of these we hope to touch upon before closing this review.

Psychoses. By almost insensible gradations, mild or profound depressed states develop on a basis of the neurasthenic toxic condition plus a greater individual unconscious conflict. The flight into the psychosis may become an overcompensatory one in those, by no means rare cases, in which suicide is effected or attempted. Less severe depressions are the rule and are very frequent. It has seemed not only my own experience but apparently from the many reports of others, quoted in part in the bibliography to have seen depressed states very frequently, so that they may be termed the most frequent of the grip psychotic conditions. At times the depression may be accompanied by delusional ideas. These are not specific. They have no relation to the influenza per se but are the symbolized products of the individual's own conditioned reflexes, or complexes, using a physiological (Bechterew, Pavlov) or a psychoanalytic term (Freud, Jung). They tell of the patient's conflicts which existed long before the influenza came along, but which by reason of what for lack of a better concept we call the "reduction in resistance" or "lowering of the psychological level" because of the toxemia and the attending worries, financial or in the love life, permits the conflict to break through under various camouflaged forms.

Thus one of my patients who had come to a fairly satisfactory compromise with her difficulties by means of a compulsion neurosis in which religious and social cleansing symbols—much praying with beads and much hand washings—are the chief hampering activities, has had two or three rather sharp influenzal attacks during the years I have known her. She came for treatment comparatively

late in life and gets along with a minimum of compulsions now that their function is somewhat understood. Following each of these attacks she has been much depressed and has heard hallucinatory voices which have referred very plainly to her anal erotic complexes. Intense constipation which has required frequent enemas or mucous diarrheas have been also present. From a psychoanalytic view point it is apparent what important function the prayers and the hand cleansing serve. These protective devices, however, break down as substitute carriers for the unconscious affective conflicts under the added stress of the grip situation, when these affects are now handled partly by means of the direct satisfaction (unconscious) of the anal areas, constipation or diarrhea, and partly by a projection of the unconscious preoccupation through the hallucinatory voices which invariably deal with anal and erotic images. (K—m—A—s) (S—t) are the most frequently heard expressions. Usually they are male voices, often heard from passersby in the street, or occasionally the belief comes to consciousness that a group of men standing on a street corner are talking about the patient and are discussing the question of giving her an enema. In such a patient the nature of the conflict is readily recognized because of the intense work already done with the analysis of the compulsion neurosis. It is worthy perhaps of more than passing comment to note that an earlier attack of grip with a similar depression and similar voice projections was also reacted to by suicidal ideas and a nearly successful suicidal attempt because of the ideas of great sinfulness re—the character of the nasty voices. A severe increase in the neurosis took up the period of recovery from the grip. Fifteen years later, however, with marked lessening of the compulsion the hallucinatory attack almost gave the finish to the neurosis for the patient now saw for the first time that the hallucinatory voices were her own unconscious preoccupations projected upon an outside

source in order to be the more readily camouflaged. She not only did not pass into a depressed state but made a distinct step towards freedom from her unconscious sadistic difficulties.

A great variety of acute hallucinatory and confusional syndromes may be described. The content of the hallucinations is always of value in casting light on the conflicts of the individual and thus later may be of great service, should the opportunity arise, in showing the patient what has determined their "neuropathic" make up, not in terms of their grandmother or other equally elusive ancestral shade, but for themselves and right now. It may be very fascinating to know what Mendelian laws are being verified in the light of heredity in traits mental and otherwise, but that is all passed and been rendered static, it is of no service in the actual alleviation of the patient and really casts not the slightest scintilla of light upon the present difficulty in the working of the individual machine. It is perfectly true that a two armed juggler can probably toss more balls than a one armed one, but the actual problem is, no matter how many arms the patient has been fortunate enough to get from his ancestors, what is he doing with those he has. He is what he is. How he is going to handle the situation is the practical problem. A careful study of the content of the psychosis is then of inestimable value in further helping the patient to a more healthy adjustment of his internal difficulties when he recovers from his psychosis, which latter is the rule.

At times extremely severe post influenzal psychotic states are observed, Ruju's case of a catatonic syndrome being a case in point. These are rare but a careful study of similar cases, and they do occur, is well worth while as throwing some light on the extremely important problem of dementia precox, that most widespread and devastating of all the psychoses. Acute infectious deliria, sometimes fatal, have been described. To epitomize the entire

literature of the psychotic possibilities let loose as it were by the influenza toxemia would need a volume.

Some General Observations. In closing a few general reflections are tempting. Bacteriologically the influenza bacillus is probably a specific entity, so far as species in bacteria go. Like other plants the products of their metabolism yields complex substances chiefly protein in their character, which may or may not be prejudicial to other organisms. There are some products of the Pfeiffer bacillus which have a definite action upon certain parts of the nervous structure.

Indeed, from the very beginning, earlier students of the disease have been struck by the high incidence of nervous symptoms, and from the eighth century to the present, there have been those who have accented this aspect of the situation. Some have gone so far as to claim that the influenza is essentially one in which nervous structures are primarily involved. That there are certain valid reasons for this generalization, we shall point out; or rather phrasing it slightly differently, we shall say that influenza is a disease of microbic origin, the poisonous products of which have a specific action upon the vegetative nervous system. The part of the vegetative nervous system bearing the brunt of the toxemia is the sympathetic. This leads to a host of physical upsets, chiefly mediated through impaired balance of the vagus sympathetic adjustment with pronounced vagotonic predominance, causing vessel paresis, and the exudative phenomena which form so essential a feature of the disease. Vowart of Bordeaux called it a pneumogastric neurosis in 1881 and many others have reached for a conception of the neurological features which were so prominent. These exudative phenomena, depending upon their location cause the various symptoms, localized vagotonias. If cephalic, they give rise to the cephalalgia, which is universal and in a small number of cases when severe, and when infection is added, as not

infrequently occurs, gives rise to a serous or non-purulent meningitis with either a maniacal coloring (rarer) or mild stuporous states, or milder, neurasthenic or hypochondriacal conditions. Epilepsy and chronic serous meningitis are among the rare results which have been discussed.

When the exudations caused by the failure of control of sympathetic tonus and hence, overaction of autonomic impulses involve the cranial nerves they result in disturbances of smell, optic neuritis, ocular palsies, trigeminal neuralgias, facial palsies, deafness, vertigo, modifications of taste, pharyngeal and laryngeal palsies. When the peripheral spinal vegetative arcs are involved, various neuralgias and neuritides result. These result chiefly from the exudation phenomena taking place in the nervi vasorum of the nerve sheaths, brachial, intercostal, and particularly sciatic. Herpes zoster is an indication of direct implication of the vegetative ganglia themselves more often an exudate rather than an infection, since the influenzal zoster, in my limited experience, have been benign. Its incidence runs high in certain epidemics. The most striking cervical sympathetic involvement is that of the pneumogastric and sympathetic adjustment. The vagus itself, which is autonomic, sometimes shows its overcompensations by bradycardia, but as a rule the sympathetic paresis or paralysis permits an overaction of the autonomic and causes the edematous flooding which characterizes the pure grip pneumonitis. This peculiar exudative character of the lung manifestation has been noted for many centuries although its fundamental pathology is still to be more adequately elucidated. The pneumonia is not to be spoken of as a complication, but as a primary disturbance of the vegetative nervous system control of pulmonary vessels, with edema and bloody infiltration resembling in its fundamental characters, the exudative phenomena of asthma or spasmodic croup, angioneurotic edema, acute edematous arthritis, hay fever, horse serums, protein

poisoning, anaphylaxis, or exudative phenomena of various origins in which there may be a generalized or localized vagotonia. Implication of the thoracic and lumbar sympathetic arcs is responsible for many of the gastrointestinal vagotonic symptoms; here the exudative phenomena are as striking as they are in the pulmonic areas, gastric, diarrhea, etc.

The spleen, liver and kidney disturbances also show a somewhat similar pathology which has not been thoroughly elucidated. Joint exudations are early and frequent. They occur suddenly and the character of the disturbance is directly indicative of the disturbed vegetative balance. The joint and muscle pains are likewise corroborative of this same general viewpoint. The various eruptive phenomena on the skin and mucous membranes speak in the same general way. Erythematous, petechial, urticarial types all permit their alignment with similar eruptive phenomena known to occur in the vegetative nerve disturbances which accompany the vagotonic trends. In many respects the striking analogies to anaphylactic reactions afford a clue to the inner vegetative mechanisms. Smith has elucidated these in a striking manner, following Roncoroni's classic exposition.

It would make a most alluring hypothesis to attempt to show that a more or less widespread and constant though unperceived involvement of the thyroid might serve as a starting point for this disturbance of sympathetic balance, the thyroid hormone containing type constituting the chief reservoirs for sympathetic upkeep, which is not confined solely to the thyroid, and therefore when involved itself adding its own disturbance to further unsettle the physicochemical balances of the body fluids. The observations thus far recorded, however, are still too scanty or too scattered to permit this generalization. At the same time attention may be called to the more or less universal adenopathy, the frequent occurrence of an acute, mild or severe thyroiditis, and the frequent overcompensatory

character of the adrenal system activity, the acute sthenic fight put up followed by the great myasthenia and other signs clearly indicative of adrenal exhaustion, Sergent white line, etc., already noted here.

We cannot carry these suggestions further in this place. There are abundant sources with pathological protocols to show the probable pathogenic affinities, say to such sympathetic paralysants, or autonomic stimulants, such as nicotine, pilocarpine, physostigmine, or muscarine. And the time is almost ripe for a true dynamic pathology of visceral disease to be written in terms of the reciprocal activities of the autonomic and the sympathetic regulatory mechanisms.

Every single organ of the body is under the balanced control of these two sets of opposing mechanisms. Inhibition is a problem of a resultant of positive forces—there are no negative ones in a transmitter—for the human body is a mechanism for the capture, transformation and release of energy. The physicochemical work for metabolism is regulated by the vegetative nervous system chiefly, and any disturbance in one branch of that system is bound to cause overactivity in the other. Whether the influenza toxins not only paralyze the sympathetics but stimulate the autonomies as well, thus causing an excessive autonomic swing with the unusual vagotonic predominance is a matter of fact to be determined only when the poisonous substances are isolated, their internal structural composition analyzed, and pharmacodynamically proved out. Until such time arrives more attention should be focussed upon the neurological problems of influenza, for herein may lie a key to the control of its many complicated symptoms from a cold in the nose to cold toes.

BIBLIOGRAPHY.

The literature available for the review of the nervous and mental disturbances of influenza is already voluminous. Only a brief summary of these can be given here. The interested student will find the available literature summarized to 1901 in the *Index Catalogue of the Surgeon General's Library, II Series*. The later

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literature may be found in the *Jahresberich f. Neurologie u. Psychiatrie* and in the valuable indices of the *Revue Neurologique* more particularly, and those of the *Neurologisches Centralblatt* and the *Journal of Nervous and Mental Diseases* and the *Revista di Patologie Nervose e Mentale*. The indices of the *Journal A. M. A.* afford a valuable source, as well as the most complete of all the *Index Medicus*.

Vegetative Disturbances: Endocrinous glands, etc.—BAHRI: *Rev. hebd. d. Laryng.*, 1, 129. BENON: *Gaz. d. Hop.*, 85, 1727. CRESPOLANI: *Gior. int. d. s. med.*, 1900, 865. ECKLEY: *Tr. Med. Soc. Iowa*, X, 1892, 93. FONTANIER: *These de Paris*, 1910. GHEDINI: *Il Policlin.*, 18, 26. GHEDINI: *Cent. f. Bact. T.*, 57, 567, 0. GHEDINI: *Gazzetti Osp.*, 31, 1041. GHEDINI: *Clin. med. Ital.*, 49, 1910, 152. GHEDINI: *Clin. med. Ital.*, 49, 439. GOMEZ: *Riforma med.*, January 23, 1900. LECLERC: *Bull. e. mem. d. Soc. d. hop.*, 31, 739. MILLS: *Maryl. M. J.*, 55, 96. MOREL-LAVAILLE: *Méd. Orient.*, 1898, 73. PAGANELLI: *Clin. med. Ital.*, 53, 197. PATRONE: *Gazz. d. Osped.*, 31, 1513. PETER: *Gaz. des Hop.*, 1891, 1054. ROSENWALD: *Greifwald These*, 1890. TESTI: *Giorn. med. d. essercito*, 1890, p. 1890.

CRANIAL NERVES.

Olfactory.—BARDT. BOSSERS: *Dissert.* DIPPE. ZWAARDENMAKER: *Tidsh. f. Genesek.*, 1891, 495.
Optic.—ANTONELLI: Paris, 1903, 19. ANTONELLI: *Annali di Ottal.*, 21, 1892, 119. BADAL et FAGE: *Arch. d'Ophth.*, 1890, 418. BICHELONNE: *Ann. d'Oc.*, October, 1904. BONCHUT: *Gaz. d. Hop.*, 1878, No. 1. BRAUNSTEIN: *Jahresb. f. Oph.*, 1890, 447. BURNETT: *Arch. of Ophth.*, 1893, 286. CROSS: *Tr. Ophth. Soc.*, xii, 1893, 70. DENTI: *Annali di Ottal.*, 19, 771. DESCHAMPS: *Ann. d'Oc.*, 122, 436. DE SCHWEINITZ: *Ophth. Record*, 1901, 41. EPERON: *Prog. méd.*, 1890, 471. GAZIS: *Rec. d'Oph.*, 1890, 586. GALEZOWSKI: *Recueil d'Oph.*, 1897, 177. GALLEMAERTS: *Policl. d. Bruxelles*, June 1, 1901. GORECKI: *Recueil d'Ophth.*, 1890, 46. GOWERS: *Lancet*, July 1, 1893. HANSEN: *Nord. Oph. Tidsk.*, iii, 1890, 2. HARTRIDGE: *Ophth. Rev.*, 1892, 345. HILLEMANS: *Bonn Dissert.*, 1890. HIRSCHMANN: *Festsch.* 50 Jahres, Dusseldorf, 1894, 340. A. KNAPP: *Arch. Ophth.*, 45, 1916, 247. KOENIGSTEIN: *Wien. med. Bl.*, 13, 1890. KOPFF: *Cong. Int. de Med.*, Paris, August 4, 1900. LEBEAU: *Ann. d'oculiste*, 1890. LEBEAU: *Ann. d'ocul.*, 1890. LEE: *Liverpool Med. J.*, January, 1892. MCHARDY: *Lancet*, 23, 1892. MACNAMARA: *Ann. d'oc.*, ii, 189, 208. MASCHOWZEWA: *Jahresb. f. Oph.*, 1891, 463. METAXAS: *Ann. d'ocul.*, 107, 343. MEURER: *Prog. méd.*, 1891, 111. NATANSOHN: *Klin. monats. f. Augenb.*, 39, 811. NATANSOHN: *Verh. Mosk. augen. Gesell.*, 1902. NOVELLI: *Boll. d. oc.*, 14, 189, 5. PFLUGER: *Ber. Kl. W.*, 1890, 603. PROTHON: *These de Lyon*, 1901. PUCCIONI: *Boll. Ac. Med. Rom.*, 1906, 32. RAMPOLDI: *Annali d. ottal.*, 19, 70. REMAK: *Cent. f. Augenb.*, 1890, 201. SAMEH BEY: *Clinique ophth.*, 1899, No. 15. SCHIRMER: *Sömmn. zu H. Aug.*, 1896, 1. SCHMIDT-RIMPLER: 455. V. SCHROEDER: *Petersb. med. Woch.*, 1892. SNELL: *Ophth. Rev.*, 1892, 219. STOEWER: *Münch. m. Woch.*, 1890, 465. THIELE: *Charité Ann.*, xx, 1895. UHTHOFF: *XXVII Versamm. d. Ophth. Ges. zu Heidelb.*, 1900, 28. VIGNES: *Recueil d'Oph.*, 1890, 402. WEEKS: *N. Y. M. J.*, August 8, 1891. WINGEROTH-GELPE: *Kl. Mon. f. Augen.*, 37, 85.
Oculomotor Palsies: Peripheral and Nuclear.—ALDRICH: *Clevel. Med. J.*, 1901, 325. BRAUNSTEIN: *Jahresb. f. Oph.*, 1890, 447. FAYE: *Arch. d'Ophth.*, x, 1890, 136. FAYE: *J. de med. de Bordeaux*, 1890, 423. FUKULU: *Ther. Monats.*, 1890. FUKULU: *Neur. Ctb.*, x, 532. GAYET: *Recueil d'Oph.*, iii, 1876, 172. GILLET: *Prog. Med.*, 1890, 29. GOLDFLAM: *Neur. Ctb.*, x, 162. GUIDICELLI: *Bordeaux*, 1912. GUTMAN: *Ber. Kl. W.*, 1890, No. 48. OPPENHEIM: *Die Encephalitis*, 1897, p. 45. PFLUGER: *B. kl. W.*, 1890, 638. PFLUGER: *Arch. f. Aug.*, 37, 71. SATTLER: *Prag. M. Woch.*, 1890, No. 13. SCALINCI:

Jelliffe: Nervous and Mental Disturbances of Influenza.

- Riv. Ital. ott., 7, 188. SCHIRMER: *Kl. Mon. f. Aug.*, 1890, 312. SINNINGER: *Br. M. J.*, July, 15, 1899. STOEWER: *Kl. Mon. f. Aug.*, 1890, 418. THOMSON: *Manhattan Eye and Ea. Rep.*, xii, 9. UHTHOFF: *Deut. m. Woch.*, 1890, No. 70. VAN DER BERGH: *Jahr. f. Oph.*, 1890, 446. VASCHIDE: *Soc. d'Oph. d. Paris*, 1890. WILLBRAND and SAENGER: Vol. 1, p. 252. WILLIAMSON: *B. M. J.*, May, 1898, 48.
- Trigeminus*.—BRAUNSTEIN: l. c. FRANKL HOCHWART: *Zt. f. Kl. Med.*, 1890, 361. FREY: *Deut. m. Woch.*, 1891, 429. SCHIRMER: l. c.
- Facial*.—LAACHE: *Norsk. m. f. Laeg.*, 1890, 11. SCHIRMER: l. c. SINNINGER: l. c. THUE: *Norsk. mag. f. Laeg.*, 1890, 11.
- Auditory and Vestibular*.—GRADENIGRO: *Allg. Wien. Zeit.*, 183, 38. NAGER: *Int. Congr.*, xvi, *Otol.*, pt. 2, 687. NAGER: *Verh. d. Otol. Gess.*, 22, 291.
- Glossopharyngeal*.—BONNET: These de Lyon, 1893. GOLD-FLAM: *Neur. Ctbl.*, x, 1890, 162. HEYMANN: *B. Kl. Woch.*, 1890, 899. KRAKAUER: *B. Kl. W.*, 1890, 899. OPPENHEIM: *Die Encephalitis*, 1897. UHTHOFF: *D. m. Woch.*, 1890, No. 70. WYLIE: *Proc. Roy. Soc. M.*, 1911, 12.
- Vagus*.—BELL: *Indiana M. J.*, 1890, 221. MEMMI: *Rev. d. Med.*, 18, 189. OPPENHEIM: *Die Encephalitis*, 1897, p. 45. STRANGE: *B. M. J.*, ii, 1890, 629. YEO: *Bull. Med.*, 1892, 732.
- Hypoglossal*.—LAACHE: *Norsk. M. f. Laeg.*, 1890, 842. SCHIRMER: *Kl. Mon. f. Aug.*, 1890, 312.

PERIPHERAL NERVES.

- Neuralgia—Neuritis—Polyneuritis*.—Older lit. to 1900 in Remak. Nothnagel's Series, ii, 528, 1901-1905. Kerschesteiner in Lubarsch. Ergebnisse, 1911.
- ALLYN: *J. A. M. A.*, July 24, 1897. BIDOW: *Rev. d. Med.*, 1890, p. 661. BONNET: These de Lyon, 1890. BRORSTROM: Leipzig, 1910. BROSSET: *Lyon Med. Arch.*, 15, 1891. CANEPA DIMITRI: *Rev. Assoc. Med. Argent.*, 23, 1915, 1739. CESTAN et BOBONNELX: *Gaz. d. Hop.*, 1900, 725. DEJERINE: *Arch. d. Physiol.*, 1890, 248. DIEMER: These de Paris, 1900, 1911. DIEMER: *Gaz. hebdom.*, January, 1901. FEINBURG: *Neur. Ctbl.*, 1897, 637. FRAENKEL: *Deut. m. Woch.*, 1890, 430. GLORIEUX: *Ann. d. l. Soc. Neur. d. Belg.*, 1901. HENSCHEN: *Deut. z. f. Nerven*, 12, 1898. JACOBSON: *Hospitid.*, 1894, 1153. JOLLY: *Arch. f. Psych.*, 26, 903. LEHMANN: These de Paris, 1901. LEYDEN: *Neur. Ctbl.*, 1893, 380. LYON: *Clinique de Paris*, 7, 808. PERRIN: Monograph, 1901. PUTNAM: *Bost. M. and S. J.*, 1892, 405. RADT: *Mün. m. Woch.*, 1898, 36. RENZI: *Gazz. Osp.*, July 17, 1898. SCHWARTZ: *Prag. m. Woch.*, 1900, 627. TSCHERMOSCHWARZ: *Djetskais med. L. M. WARFIELD: Wisc. M. J.*, 14, 1915. WESTPHALEN: *St. Peter's m. Woch.*, 1890, No. 21.
- Landry's Paralysis*.—BERNHARDT: *B. Kl. W.*, April 24, 1890. EISENLOHR: *D. m. W.*, February 11, 1890. LAPEYRE: *Bull. Soc. d. m.*, p. 2, P. 1892, 278. PEILLAS: *Arch. d. Neurol.*, December, 1895. V. LEYDEN: *Neur. C.*, 1893, 380. V. LEYDEN: *Z. f. Kl. M.*, 24. WERLE: Wurzburg Dissertation, 1913.
- Discussions concerning Influenza, Poliomyelitis, Lethargic Encephalitis, Botulism, etc.*—ALFRED-KHOURY: *Bull. et mem. Soc. Med. Hop. Paris*, 1918, 35, xlii, 455. ARDIN-DELTEIL: *Bull. et mem. Soc. M. Hop. Paris*, 1918, 35, xlii, 577. A. BREINL: *Med. J. Austral.*, 1918, i, 209. E. F. BUZZARD: *Lancet*, 1918, F. 715. W. CAREY: *Lancet*, 1918, T. 716. A. CHAUFFARD and M. BERNARD: *Bull. et mem. Soc. Med. d. hop. d. Paris*, 1918, 35, xlii, 330. A. CHAUFFARD and M. BERNARD: *Bull. mem. Soc. med. Hop., Paris*, 1918, 3-5, xlii, 470. H. CLAUDE: *Bull. et mem. Soc. Hop., Paris*, 1918, 3-5, xlii, 364. F. G. CROOKSHANK: *Lancet*, T. 1918, 699. E. C. DICKSON: *Pub. H. J.*, ix, 1918, 280. C. FRANCONI: *Riv. d. clin. ped.*, xv, 1917, 505. GIOVANNI: *Boll. d. Cliniche*, June, 1897. H. A. HALL: *B. M. J.*, 1918, i, 467; *Lancet*, 1918, T. 568. W. HARRIS: *Lancet*, 1918,

Jelliffe: Nervous and Mental Disturbances of Influenza.

I. 568. R. E. LORD: *Lancet*, T. 1918, 715. LORTAT-JACOB, G. L. HALLEZ: *Bull. et mem. Soc. Med. Hop. Paris*, 1918, 35, xlii, 39. P. MARIE and C. TRETINKOFF: *Bull. et mem. Soc. M. Hop. Paris*, 1918, 3-5, xlii, 475. T. H. MATHEWSON and LAWSON: *Med. J. Austr.*, ii, 1917, 352. C. H. MELLAND: *B. M. J.*, 1918, i, 559. A. NETTER: *Bull. et mem. Soc. H. d. Paris*, 1918, 35, xlii, 384. A NETTER: *Bull. et mem. Soc. m. d. H. de Paris*, 1918, 35, xlii, 307. R. M. F. PICKEN: *Lancet*, 1918, ii, 35. RAYMOND: *Nouv. Icon. d. l. Salpet.*, 1890. J. A. RICE-OXLEY: *Lancet*, 1918, ii, 15. SAINT MARTIN-I.HERMITTE: *Bull. et mem. Soc. Med. Hop. Paris*, 1918, 35, xlii, 457. SAINTON: *Bull. et mem. Soc. Med. Hop. Paris*, 1918, 35, xlii, 424. *Ib.*, 543. *Presse méd.*, September 23, 1918. J. A. SMITH: *Lancet*, 1918, T. 737. STRUMPELL: *D. E. f. N.*, December, 1900. F. TILNEY: *Neur. Bull.*, 1918, i, 7. S. K. VAIDYA: *Lancet*, 1918, ii, 322. VAN GEUCHER: *Journal de Neurologie*, 1890, S. A. K. WILSON: *Lancet*, 1918, ii, 7, 91.
Pseudo Tabes.—DEJCRINE: *l. c.* LIVIERATO: *Gaz. d. O.*, 1890, 474. PUTNAM: *Bost. M. and S. J.*, 1892, 349, 405.

SPINAL CORD.

Myelitis.—APOSTOLI-PLANET: *Rev. de Med.*, 18, 1890, 550. BERENS: *Bull. d. l. Soc. d. Med. De Gand.*, 60, 1893, 90. DE GROOTE: *Lille These.*, 1896. EULENBERG: *Deut. m. Woch.*, 1892, 38. FIESSINGER: *Gaz. med. d. Paris*, 1892, 445. FOA: *Policlin.*, 1890, 129; *Arch. Ital. de Biol.*, 1890, 57. FRIEDMANN: *Arch. f. Psych.*, 1057, 1901. GHILARDUCCI: *Rass. Sec. Med.*, 1890, 349. LUTTIG: *Kiel Dissertat.*, 1910. PONTIPPIDAN: *Hosp. Tid.*, 1898, 461. POTTS: *Univ. Med. J.*, 1890, 14. REVILLIOD: *Rev. med. d. l. Suisse Rom.*, 1890, 148. WEDEKIND: *Med. Rec.*, 1892, 418.

Brown Sequard Syndrome.—DETERMANN: *D. Z. N.*, 1891, vol. 2. EULENBERG: *D. m. W.*, 1892, 38. HERZOG v. MAILLART: *Berl. Kl. Woch.*, 1890, 35. LAVERAN-FEREO: *Soc. d. Hop.*, February 21, 1890. REVILLIOD: *l. c.*

Multiple Sclerosis Syndrome.—OPPENHEIM: *Textbook*, p. 000. ROSSI: 1900. SORGONI: *B. d. Sc. m. d. Bul.*, 1897, 847.

BRAIN.

Meningitis.—This literature is very extensive. Only a small series of articles are here quoted.

AASER: *Tidsk. f. Nord. Laeg.*, 36, 1916, 393. S. S. ADAMS: *Arch. of Ped.*, October 24, 1907. AGER and AVERY: *Arch. of Ped.*, April, 1910. ALLYN: *Med. News*, 1894, 712. F. BATTEN: *Lancet*, 1910, i, 1077. BLACH: *B. M. J.*, 1895, i, 976. BLACQUE: *These de Paris*, 40; *Steinheil*, 1911. BREM V. ZEILER: *Am. J. Dis. Chil.*, 1, 417. A. BROWN: *Canad. Med. Assoc. J.*, 5, 1915, 1076. BURY: *B. M. J.*, ii, 1900, p. 879. COHOE: *A. J. M. S.*, 137, 1909. CUMSTON: *Austr. M. J.*, January, 1912. DAVIS: *Am. J. Dis. Child.*, i, 1911, 259, bibliog. DAVIS: *Arch. Int. Med.*, iv, 323. D'HOTEL: *Union méd. d. N. E.*, 38, 1913, 193. DOUGLAS: *Lancet*, January 12, 1907. DOUGLAS: *Rep. Lab. St. George Hosp.*, 1910, 186. DUBOIS: *These de Paris*, 49, 1902. DUEROT: *Rev. m. d. la Fr. Compte*, 21, 195. R. M. EIDSELL: *Br. M. J.*, ii, 1913, 1056. FLEXNER: *J. A. M. A.*, 57, 16. F. FRANKE: *Med. Klin.*, 1909, Beihelf 10. GASKELL: *Lancet*, i, 1912, 364. GHON: *W. R. W.*, 1902, 26. GIESE: *Ugesk. v. Laeg.*, 75, 621. GRASY: *Am. J. Obst.*, 67, 1913, No. 5. HALLION and BAUER: *Rev. Neur.*, 1911, 381. HELLPACH: *Deut. m. Woch.*, 1910, 493. HYMANSON: *N. Y. Med. J.*, December 25, 1901. KLINGER: *Correspondenzbl. f. Schw. Aerzte*, 34, 1912, p. 1289. LESTRER: *Lyons*, 1904. LIEBAU: *Kiel Dissert.*, 1913. MARAGLIANO: *Cr. d. cl. med. d. Genova*, 21, 1915, 90. E. E. MOODY: *Jl. Missouri Med. Assoc.*, 13, 1916, 328. HUNTER NUTHALL: *B. M. J.*, i, 1901, p. 74. C. NYBERG: *Finsk. lak. Sal.*, 57, 1915, 1369. PHILLIPS: *Ed. Hsp. Rep.*, 1893, 276. PISEK: *Am. Med.*, April, 1912. PRASEK and ZATELLI: *Wien. Kl. Woch.*, 1911, 932. PUTNAM: *Bost. M.*

Jelliffe: Nervous and Mental Disturbances of Influenza.

- and S. J., 1892, 349. RHEA: *Arch. Int. Med.*, 1911, 133, Blood Cultures. RITCHIE: *J. Path. and Bact.*, 15, 1910, 615. ROSS-MOORE: *B. M. J.*, ii, 1913, 1050. SATTERTHWAITTE: *N. Y. M. J.*, 1913. SAYCE: *Austral. M. J.*, January 20, 1911. SIMON: *Monatsh. f. Kindhk.*, 1911, 549. SLAWYK: *Zeit. f. Hyg.*, 32, 1898. SPELLMAN and BENECH: *Prov. Med.*, 24, 433. SPRINGER: *Del. State J.*, v, 1. A. H. TEBBITT: *Med. J. Australia*, i, 1914, 45. THOMESCA-GRACOSKI: *Rev. Neur.*, 1905, p. 44. R. G. TORREY: *Am. J. Med. Sc.*, 142, 1916, 403. VIGOUROUX: *Bull. Soc. anat. d. Paris*, 6-5, 13, p. 288. W. C. WILLIAMS: *J. A. M. A.*, July 4, 1903. E. H. WILLIAMS: *New Zeal. M. J.*, 15, 1916, 48. M. WALLSTEIN: *J. A. M. A.*, 59, 1912, p. 148, 12. M. WALLSTEIN: *Am. J. Dis. Ch.*, i, 42. M. WALLSTEIN: *M. J. Exp. Med.*, xiv, 73. M. WALLSTEIN: *Int. Cong. Hyg.*, ii, 1913, 57. M. WALLSTEIN: *Med. Rec.*, 79, 1910, 134.
- Encephalitis.—BILHAUT: *B. et m. d. la Soc. d. Ther.*, 1890, 22. BRISTOWE: *B. M. J.*, 1891, ii, 6. DIELOW: *Lancet*, ii, 1909, 1498. DUBOIS: These de Paris, 1902. FURBRINGER: *D. m. W.*, 1892, 45. GOLDFLAM: *Neur. Ctbl.*, 1891, No. 7. HAEDKE: *Munch. med. Woch.*, 1897, 806. HARRIS: *Practitioner*, January, 1907. MARTY: *Arch. g. le med.*, 1898, 513. NAUWERK: *D. m. W.*, 1895, 393. OPPENHEIM: Die Encephalitis, 1897, second ed., 1902. PFUHL: *Zeit. f. Hyg. v. Inf.*, 26, 1897, 112. PFUHL: *B. Kl. W.*, 1892. POOLE: (Aphasia) *B. M. J.*, 1890, i, 190. PUTNAM: *Bost. M. and S. J.*, 1892, 349. STEPP: *Munch. m. W.*, 58, 2269. TYLECOTE: *Clin. J.*, 42, 622. VIGOUROUX: *Clin. Par.*, vi, 362.
- Hemiplegia.—AMANTINI: *Ann. d. Ippoc.*, v, 114. COMES-SATTI: *Rev. crit. d. med.*, 25, 360. RIA: *Riv. int. d. terap.*, iv, 259. STEPP: *Med. Klinik*, 1911, 1306.
- Epilepsy.—BOERI: *Rif. Med.*, 1894, 10, 1758. ERLNMEYER: *B. K. W.*, 1890, 295. GELINEN: *Indep. Med.*, 1900, 91.
- Cerebellum.—BERAUD: *J. d. m. d. Lyon*, 1896, 6, 181.
- Psychoneuroses and Psychoses.—AHRENS: Greifswald Diss., 1890. ALTHAUS: *A. J. M. S.*, 1892, 103, 361. BEAUDOINET CHASLIN: *Encephale*, June, 1909. BERNHEIM: *Rev. de med.*, 1909, 1911. BIDON: *Rev. de med.*, 1890, 1. BLOCQ: *Gaz. heb.*, 1890, 267. BORCHARDT: Berlin Thesis, 1890. BRIENNE: These de Paris, 1890. BRUNET et CALMETTE: *L'Encephale*, 1910, 291. CHURCH: *Chic. Med. Recorder*, 1891, p. 418. DUEROUX: These de Paris, 1902. EBERLING: 1892. FEHE: Copenhagen Dissert., 1898. HUTCHINGS: *State's Hosp. Bull.*, 1896, i, 112. JEANDIDIER: These. JONES: *Am. J. Med. Sc.*, 141, 267. JOHNSON and GOODALL: *Lancet*, August 16, 1902. JUBOINSKI: *D. m. W.*, 1891, 89. KIRN: *Allg. Z. f. Psych.*, 1891. KLENM: Warburg, 1901. KRAEPELIN: *D. m. W.*, 1890, 209. LAROUSSINIS: *Ann. Med. Psych.*, 1896, p. 472. LEHR: *D. m. W.*, 1890. MALLARDO: *Corriere Sanitorio*, January 27, 1901. MANCEL: These de Paris, 1902. MISPELBAUM: Berlin Thesis, 1890; *Allg. Z. f. Psych.*, 1890, 127. PRITCHARD: *Int. Clin.*, vol. 1, 11th series. REGIS et LAVAURE: *Congres de la Rochelle*, 1893. REGIS: *Precis*, third ed., 633. ROUGE: *Ann. med. Psy.*, April-June, 1909. SAVAGE: *Med. Press Circ.*, 96, 578. SEGLAS: *Soc. m. d. Hop.*, March 21, 1891. SHAW: *Practitioner*, January, 1907.

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