adore, when sickness comes, not before. When we get well, both are alike requited; God is forgotten, and the doctor slighted." But a period in the near future shall mark the triumph of science over darkness and superstition, and the physician no longer be summoned only in emergencies to apply his art to the cure of maladies produced by a disregard of the laws of health, but he shall be an officer of state, clothed with honor and authority and salaried by the government. His mission shall be as an apostle of the goddess Hygieia to teach the people the science of health—the art of keeping well. Under his genial administration crime and disease shall be the exception, peace and health rule. Healthy parents shall behold with pride the rosy cheeks of their children, which, like olive plants, shall grow up and become fruitful. National prosperity and happiness shall be the result of a symmetrical manhood and womanhood endowed with physical strength and beauty, mental excellence and moral rectitude. Mankind shall not be hurriedly summoned from the stage of the world's drama in the innocence of childhood, the bloom of youth or the busy activities of middle life, but in a green old age at the nightfall of life, willingly yield to the influences of a gentle and peaceful euthanasia.

Original Articles.

ACUTE ASCENDING PARALYSIS AND LANDRY'S PARALYSIS.

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At the present time there is much confusion of thought, and not a little conflict of statement, as to exactly what is meant by Landry's paralysis and as to the true nature of the disease. Landry's paralysis and acute ascending paralysis are by no means always the same, and yet the terms are used synonymously. There must be a middle ground whereon they overlap to warrant this synonymous use of the two names. A clear conception of the nature of this middle ground, from both the clinical and pathologic points of view, will not only go far toward explaining the dissimilar cases lying outside of it and on either hand, but it will help to accomplish what is of far more practical importance, for it will afford greater unity, definiteness and coherency in the treatment of all cases. The purpose of this paper is to clear up a little, if possible, the confusion still surrounding the present conception of acute ascending paralysis. The following propositions, resting on clinical and laboratory observations, will be discussed, and in the end certain definite, practical conclusions will be attempted:

(a) Landry himself described not a new disease—not even a disease—but rather a symptom-complex or clinical picture, which included much less than what is now included under the term acute ascending paralysis.

(b) Both Landry's disease and acute ascending paralysis are recognized to-day as mere phases of a particular form of specific intoxication of the nervous system.

(c) The proof that this intoxication is of bacterial origin, the particular micro-organism, if, indeed, there is a particular one being still unknown, is about established.

(d) This bacterial intoxication produces functional disturbances, and later on diffuse inflammatory and degenerative changes in various parts of the central and peripheral nervous apparatus, thus accounting for the variability of the clinical phenomena.

(e) Time, virulence of the poison and resistive power of the patient are the principal factors that determine the nature of the pathologic findings and the clinical form of the trouble. Under some circumstances the post-mortem findings may be *nil*, while under other circumstances they may be highly suggestive of a diffuse myelitis, an acute anterior poliomyelitis or a peripheral multiple neuritis. It is an error, therefore, to identify Landry's paralysis, or even acute ascending paralysis, always with one or the other of these diseases.

(f) The recognition of the class of cases under discussion, as a form of diffuse bacterial intoxication, not only explains most satisfactorily their striking clinical discrepancies, but suggests a far more rational guide for their management than has any explanation hitherto offered.

If we are to limit the definition of Landry's paralysis to what Landry himself described in 1859, we must narrow it to an acute ascending paralysis, flaccid in character, rapid in progress, without disturbance of the reflexes, without muscular atrophy or electrical changes, without sensory phenomena and without implication of the vesical and rectal functions; usually fatal in a few days with respiratory involvement; and devoid of all discoverable postmortem pathologic changes. Such a definition, however, is not comprehensive enough to include the modern conception of acute ascending paralysis. Cases are on record wherein the paralysis was not of the regularly ascending type, but had descended and even skipped about from arm to leg and from side to side. In these cases, moreover, sensory symptoms appeared early; typical muscular atrophy was present; and there were well-recognized alterations in the electrical responses.

Not a few of the cases recovered. Among those that were fatal, postmortem examinations, made in accordance with the more delicate modern methods, revealed distinct inflammatory hemorrhagic and degenerative lesions. Thus, from both the clinical and pathologic points of view, the description of acute ascending paralysis has to be drawn on much more extensive lines than those adopted by Landry and his immediate followers. As we proceed with our discussion, it will be seen that Landry described merely a symptom-complex or group of cases that form a part of a much more distinct and comprehensive group.

Among the first to break entirely away from Landry's narrow conception of the disease were Taylor and Clark, who, in 1900, wrote: "On the basis of an acute, clinical and necessarily highly superficial pathologic observation made many years ago by Landry, subsequent observers, led largely by a name, have been attempting to force into the narrow clinical conception outlined by Landry, various symptoms, none of them fundamental and supported by no constant pathologic findings and still less by a definite etiology. The so-called disease has, with each new investigation, lost something of its coherence. We have persistently clung to a name, and demanded that symptoms which it represented should conform to our preconceived idea." These words voice the opinion of DeJérine, Schwab, Schmaus, Diller and others. To separate such groups of cases as Landry described and give them the name of a distinct disease, is

an arbitrary, useless and confusing procedure. It belongs to that sort of ultra-refining classification which makes a special disease, ataxic paraplegia, for instance, of a mere combination of lateral and posterior sclerotic degeneration, or enumerates a half dozen or more types of chorea out of a mere symptomatic form of muscular movement.

It is much more rational and practical to regard all of the groups of acute ascending paralysis as merely particular manifestations of a diffuse, specific intoxication, which intoxication is rendered more or less variable by such factors as the duration of exposure, the virulence of the poison and the particular susceptibility of the patient; and which, therefore, reveals itself in one or other of the clinical types of the disease described above. The one point in which these various types of acute ascending paralysis all seem to agree, and which justifies, probably, the recognition of it as a more or less distinct disease, is its apparent limitation to the lower or peripheral neurons. If perchance some day a specific micro-organism should be discovered as the cause and the *only* cause of Landry's paralysis, the ground for considering the latter as a distinct and uniform disease would be quite unassailable. At present no such ground exists.

Let us now consider a little more in detail the modern clinical picture of acute ascending paralysis, so as to discover, if possible, its likeness and unlikeness to other well-known forms of disease, such as multiple neuritis, acute anterior poliomyelitis, acute meningomyelitis or acute ascending myelitis. With Landry we agree that there is a group of cases in which there is a rapidly ascending paralysis, motor in type, flaccid in character, beginning usually in the lower extremities and then passing to the upper limbs, and finally causing death by involvement of the bulbar functions, especially respiration. In its upward, rapid course this paralysis creeps, as it were, from the feet to the neck. Even the muscles of the trunk and throat do not escape. Deglutition and articulation are affected. Cheyne-Stokes breathing is frequently present, and asphyxia with diaphragmatic paralysis usually closes the scene. So swift and profound is the disease that it is almost appalling at times. I have seen it terminate in death a few hours after its first manifestation. Generally it lingers a few days, death occurring, as a rule, within a week. In the exceptionally rapid cases the reflexes may be found as yet undisturbed. In the larger number, however, there is more or less loss of the deep and superficial reflexes. Only in a few prolonged cases do muscular atrophy and abnormal electrical reactions appear. The bladder and rectal functions remain normal. Sensation is rarely, if ever, disturbed. The mind continues clear. Often in the beginning there is a slight elevation of temperature, but this quickly subsides and remains at normal. This peculiar elevation of temperature, with hyperidrosis, mild delirium and other signs of a general septic infection, appears rather frequently in the more recent reports. Postmortem examinations reveal no lesions inside or outside of the nervous system, no changes of any sort, except the rather frequent enlargement of the spleen. Such, in brief, is the modern typical Landry's paralysis. It is the definition of Leyden, approved by Thomas, that is accepted nowadays.

From this type, however, we recognize many divergent forms. For instance, Mills and Spiller, after adopting the definition of Leyden, admit a descending type of the disease in which paresthesia, without severe

or persistent pain, is a prominent symptom. Clinically, the picture of Landry's paralysis undergoes some most noteworthy and suggestive changes, especially in those cases that have escaped a fatal issue, and that gradually subside after a comparatively long period of duration. Moreover, these prolonged cases, when they do die, reveal at the autopsy some very striking and suggestive pathologic changes.

The modern clinical divergent types of the disease are to be found anywhere within the extremes of a multiple neuritis on the one hand, and an acute anterior poliomyelitis on the other. So completely, indeed, have we faced about, that Diller, in his exhaustive study of Landry's paralysis, now assigns the first position, both in regard to time and importance, to the sensory symptoms.

Including these divergent forms of the disease, then, Landry's paralysis, in the most modern sense, must conform to the following conception:

It does not always regularly ascend. It skips about. Occasionally it appears first in one leg and then in the opposite arm, to continue in the other leg and finally in the other arm. One leg and both arms, or both legs and one arm, may represent its distribution. Fagge states that the great naturalist, Cuvier, succumbed in a few days to an acute descending paralysis which bore all the signs of Landry's disease; while Oppenheim reports a case in which only bulbar symptoms were present. Rowden's case was of the descending type, and in Howard's case bulbar symptoms showed themselves first, and then in order involvement of the following muscles: hip, thigh, shoulders, arms, legs, hands, feet, lips and diaphragm. I recently saw a case in which bulbar symptoms appeared early, but diminished as the paralysis attacked other muscles of the body. The cranial nerves are sometimes involved, as shown by the facial paralysis, diplopia and paralysis of accommodation, all of which have been reported as a part of the picture of Landry's disease.

A certain degree of muscular atrophy has been observed in some cases, so much so that they quite resembled an extensive form of acute anterior poliomyelitis. The electrical reactions have also been seen to undergo the well-known changes characteristic of degeneration in the lower motor neurons. In Burghart's case, quoted by Knapp and Thomas, there were typical electrical reactions of degeneration. In a case of Oppenheim's, the nerves and muscles reacted to weak electrical stimulation, but an increase in the current strength did not increase the intensity of the contractions. Even the strongest current failed to secure a pronounced contraction. Curiously enough, on several occasions, a strong current produced no contraction at all or a weaker one than that evoked by a weaker current. Oppenheim says that a piece of the muscle showing these peculiar electrical phenomena revealed on examination a waxy-like degeneration. The symptoms disappeared with the recovery of the patient.

So far as I know, very few cases have been observed or reported in which the sphincters were involved. Oppenheim, however, is authority for the statement that complete sphincter paralysis does occur. In Goebel's case the vesical sphincter was weak, and late in the disease there was diminished sensation. Sphincter troubles were noted in Knapp and Thomas' three cases.

More remarkable than all this is the frequent association in the modern cases of Landry's paralysis of pronounced sensory with the motor phenomena. The sen-

sory disturbances have been of every variety, but chiefly in the pain, temperature and touch spheres. From hyperesthesia to anesthesia, hyperalgesia to analgesia, all along the line of the various paresthesiæ, have the sensory manifestations been observed. These cases have been reported by such competent observers as Robinson, Taylor and Clark, Mills and Spiller, Worcester, Marie and Marinesco, Hirtz and Lesné, Oettinger and Marinesco, Diller and others. Early sensory symptoms, in some cases before the onset of the paralysis even, have been seen in cases reported by Schwab, Wappenschmidt, Griffith, Kapper, Maxwell, Stockton, Diller and others. In the words of Diller: "In the symptomatology of Landry's paralysis, sensory symptoms are almost constantly present and generally precede the motor paralysis by several hours or a day, occasionally by much longer periods." Pains are found in many of the cases. They are of the nature of local tenderness and neuralgic pains, and in this respect are highly suggestive of peripheral neuritis. They are elicited by pressure on the muscles and nerves and by passive movement. Though frequently complained of, these pains after all are, as a rule, absent even in modern Landry's paralysis. A complete loss of sensation is so rare that anesthesia falls short of being an important symptom. Fugitive and variable paresthesiæ are occasionally noted. After all, it is true, as Oppenheim says, that, as a rule, gross sensory disturbances are absent in Landry's disease. When they are present it is at times practically impossible to differentiate the case from one of polyneuritis. And yet it is a mistake, as I will endeavor to show later on, to identify Landry's paralysis with peripheral multiple neuritis, as Ross and his followers have done, and to declare that they are one and the same disease.

In summing up, then, as Buzzard so well does, the clinical conception of modern Landry's paralysis, we may classify the cases under three heads. The first class includes those that are marked by motor palsy, absent reflexes, absent electrical changes, slight paresthesiæ and absence of muscular atrophy. These are the cases in which no postmortem findings can be demonstrated by the use of osmic acid and the Nissl method. In the second group of cases there are more advanced motor and sensory symptoms, some slight electrical reactions and trophic phenomena. In these cases there is in all probability a degeneration of the motor and sensory neurons, with beginning chromatolytic changes in certain ganglion cells. In the third class of cases are to be placed those that exhibit still more pronounced motor and sensory symptoms, sphincter disturbances, typical muscular atrophy and electrical changes. These cases are probably characterized by advanced changes in both the neuron bodies and their processes.

A careful study of the pathology and pathogenesis of acute ascending paralysis, so far as we have data at the present day for such a study, leads almost conclusively to the opinion that the trouble is merely a manifestation of a profound form of toxemia, in which the toxin expends its force chiefly on the lower neurons. In the earlier descriptions of Landry's paralysis, there were no postmortem findings. Westphal confirmed this, and both he and Landry, therefore, adopted the toxic theory of the disease. Others who have failed likewise to find any postmortem changes in the nervous system include such careful investigators as Kahler, Pick, Greppin, Ranvier, Vulpain, Bernhardt, Déjérine and Goetz, and Hun. It may be that in some of these cases the early and faulty technic should be blamed for

the inability of the examiners to find any pathologic alterations, while in others the lack of thoroughness in the examination must bear the responsibility. For instance, in one of Westphal's cases, only the cervical part of the cord was examined. As later observations seem to show that both the cervical and lumbar parts of the cord are equally important centers of attack, it is possible that changes might have been discovered in the lumbar region. Again, it is not improbable that in long-running cases, such as another of Westphal's, the lesions may have disappeared and the exudates have been absorbed before death finally took place. All of this may reduce the number of cases with negative findings. Nevertheless, it must be admitted that cases do come to the autopsy that do not reveal the slightest sort of pathologic change. The more recent reports of Ormerod and Prince and of Giraudeau and Levi confirm this. The number of such cases, under the more accurate and painstaking methods of modern times and the use of the osmic and Nissl staining technic, has been gradually decreasing, until now they constitute quite a small minority. It may well be imagined that in these minority cases the virulence of the poison or the special susceptibility of the patient brought about the fatal termination before any histopathologic changes were possible.

Landry, Westphal and the other early investigators were supported in their belief that acute ascending paralysis was due to a profound toxemia by the frequency with which they found on postmortem examination enlargement of the spleen, old hemorrhagic remains in the lungs and bowels, inflammation of the lymph glands and occasionally albuminuria. Gowers long ago made the shrewd suggestion that the not infrequent paralysis of accommodation seen in cases of Landry's disease confirms its toxic origin, because the isolated acute paralysis of a functional center, not anatomically separate from others, is known to be caused only by a toxic influence. The more recent work of special observers has almost absolutely established the toxic origin of the disease. For instance, Baumgarten found that in all probability anthrax was present in a case seen by him. The bacilli were discovered in the blood and tissues. In a case reported by Curschmann, though there were no clinical manifestations of typhoid fever, the characteristic intestinal lesions were present, and pure cultures were obtained from the typhoid bacilli discovered in the spinal cord. Bacilli were seen by Centanni in the endoneural lymph spaces of a case in which there was an interstitial neuritis. According to Eisenlohr, there were found in some cases various kinds of bacilli in the central nervous system, especially staphylococci. This author is inclined to attribute Landry's disease to a mixed infection. He does not believe that the micro-organisms are necessarily constant. Indeed, they may be entirely absent or the spinal cord alone may be affected by them. Among the other micro-organisms that have been found in different cases may be mentioned the *Streptococcus longus* by Remmlinger, a diplococcus by Marinesco, the anthrax bacillus, or one much like it, by Marinesco and Marie, and the so-called Fraenkel-Weichselbaum bacterium by Seitz. Bacteria have also been discovered by Burghardt, Friedlander and Giese, Mouravieff and Montourine, Oettinger and Marinesco and others. Even more direct evidence than all this of the bacterial origin of the disease has been furnished by the cases of Courmont and Bonne, Piccinino, Roger and Josué and others. Very recently Buzzard detailed

a typical case of Gowers, in which he discovered a diplococcus from which pure cultures were made, and from the injection of which into rabbits he obtained in these animals a symptom-complex identical with Landry's paralysis. As this micro-organism was found in the dura mater, Buzzard suggests that it be called the *Diplococcus thecalis*. He says that it does not conform with any previously discovered organism. In addition to Buzzard, Thoinot and Maselli have produced in animals the typical clinical picture of Landry's paralysis by the injection of micro-organisms into the blood and spinal cord (Oppenheim).

Whether the relationship is etiologic or not, it is a most suggestive fact that Landry's paralysis has been seen to develop after malaria, diphtheria, typhoid, variola, anthrax, influenza, pneumonia, pertussis, septicemia, gonorrhea and the puerperium. Once it occurred after the Pasteur treatment for rabies, and sometimes it has been attributed to syphilis and alcohol.

In a considerable number of cases, diagnosed as Landry's paralysis, changes were found in the peripheral nerves indicative of an interstitial and parenchymatous neuritis. Since Ross' exhaustive and valuable study of multiple neuritis, wherein he classifies Landry's disease as a form of polyneuritis, many have adopted the view that acute ascending paralysis of the Landry type is but a phase or manifestation of peripheral multiple neuritis. Ross based his conclusions, which in the main agree with those of Nauwerck and Barth, on a tabulated series of 93 cases. Among those who follow the teachings of Ross may be mentioned Déjérine and Goetz, Klumpke, Centanni, Kirilzew and Mamurovsky, Dydyski, Krewer, Walton, Putnam and others.

In another group of cases evidences of the disease were found only in the spinal nerve roots; and according to Buzzard, such changes are more frequent in the anterior than in the posterior roots. Oppenheim records that in one case merely a degeneration of the anterior roots was discovered. In Hun's case the results of the autopsy were negative, except for a slight cerebral and spinal meningitis and a degenerative neuritis of some of the fibers of the anterior roots of the cauda equina. Cultures were made, but no bacteria were found.

A third class of cases were characterized especially by spinal-cord involvement. Such cases have been reported by Eisenlohr, Schultze, Ketli, Hlava, Immerman and others. Disseminated foci of inflammation, exudates and capillary hemorrhages in the medulla and cord, swelling of the axis cylinders in the white matter of the anterolateral tracts, poliomyelitic remains, etc., have been the usual findings. Iwanow's two cases were highly instructive. The cervical and lumbar enlargements showed a concentration of the disease process, and at these points it was more or less limited to the central canal with extension into the anterior horns. It consisted of an exudation of lymphoid bodies, fibrinous exudates around the vessels and nerve fibers, the result of an inflammation, disintegration of the axons, swelling and indistinctness of the neuron bodies. An edema surrounded the vessels. The nerve roots and meninges were normal; bacteria were absent. The cases of Eisenlohr and Krewer showed both neuritic and myelitic processes. In Kirilzew and Mamurovsky's case there was marked acute primary parenchymatous neuritis of the nerves of the feet, legs and forearms, and to a lesser degree of the roots of the cervical and lumbar enlargements. The cord itself was hyperemic, with cloudiness of the gray substance, slight atrophy of the

anterior ganglion cells and an accumulation of cortical cells in and about the central canal. Clark has described a fluid exudation about the central canal on a level with the lumbar nerves. Baumgarten, Eisenlohr and others have noted similar findings. In Kleb's case there was pericardial tuberculous and widely distributed hyaline thrombi in the branches of the central artery of the cord. They were most numerous in the lumbar region. Secondary phenomena were minute hemorrhages and exudations, especially in the ganglion cells. The peripheral nerves were intact. The thrombi were probably of toxic origin. From the symptoms of a case which ultimately recovered, Sanger Brown believed that the chief disturbance was in the lumbar and cervical enlargements. The reflexes were exaggerated and there was an ataxic paraplegia. Touch and temperature sense were diminished somewhat. Carter's case, which also recovered, exhibited a similar picture. Both of these cases bore a singular resemblance to syringomyelia, with implication of the tissues immediately surrounding the central canal. In nearly all of these myelitic cases, a predilection is shown by the disease process for the immediate neighborhood of the central canal in the cervical and lumbar enlargements.

Finally, a fourth class of cases has been reported, in which all parts of the lower motor neuron, peripheral nerve, spinal root and ganglion cell were involved in a scattered and diffuse pathologic process. In a rapidly fatal case, Spiller found changes in the peripheral nerves and similar alterations in the cell bodies of the anterior cornua. Thomas has reported two cases with similar findings. In fact, Spiller, Mills and Osler all define Landry's disease as an acute poisoning of the lower motor neuron.

With all this pathologic evidence in hand, one can not escape the conclusion of Buzzard that Landry's paralysis, in the modern sense, is the result of "a local infection, a general pyemia, or lastly and most frequently of an acute intoxication"; and that, in the words of Taylor and Clark, it exhibits "no essential constancy in the clinical symptoms nor in the pathologic findings and the etiology is wholly vague."

In view of this widely varied picture of the clinical and pathologic presentations of so-called Landry's paralysis, the exact meaning of the name and the advisability of its retention both press for answer. It is the steady aim of science to simplify and generalize, as far as known data will warrant, all knowledge. At one time the term Landry's paralysis seems to have stood for a certain definite clinical entity; now it is applied to conditions as unlike those originally classified under it as day is unlike night. In behalf of scientific accuracy, one of the following reforms is imperatively demanded; the term itself should be irrevocably dropped, or a new and greatly expanded conception of its meaning should be adopted, or the symptom-complex which it represents should frankly be recognized and so treated in the text-books as one of the many possible phases of a particular, well-known disease process. There are advocates for all three ways out of the difficulty. Personally, I incline to the third or last way.

Diller suggests that the name Landry's paralysis, defective as it is, to define our modern conception of the trouble, is nevertheless convenient for practical purposes, much as the term apoplexy is which, in itself, means nothing, but yet covers a certain set of clinical phenomena that may be caused by a number of more or less obscure pathologic changes. If the term

Landry's paralysis were only as indefinite as the term apoplexy, Diller's suggestion might be commended, but unfortunately the term Landry's paralysis does stand for a distinct, well-marked clinical picture, and therefore can not justly be made to cover all the varied groups of cases that it is made to cover to-day. Furthermore, it is a question whether the art and science of medicine are not greatly hampered by the retention of names for the mere sake of practical convenience, when those names are so positively misleading as the name Landry's paralysis is when applied to some of the grossly divergent forms of the trouble reported nowadays.

The suggestion of Diller virtually amounts to an absolute denial of such a disease as Landry's paralysis, in which denial most of us will probably acquiesce. The distinct clinical manifestation, however, described by Landry in 1859 and since known by his name, can not be denied. It is a clearly defined entity, whose relationship to some more extensive disease process we may later on discover, but whose name we can not justly or wisely apply to manifestations which Landry, perhaps, never dreamed of when describing his particular symptom-complex.

Those who still cling to the name, recognizing the definiteness of the clinical picture which it was originally made to cover and who are loath to give it up for any reason whatsoever, adopt a curious mode of scientific logic. They eliminate from consideration all reported cases that do not absolutely conform to Landry's original description. Some very recent writers, notably Goebel, have adopted this procedure, while Bailey and Ewing, in 1896, rejected from their 75 collected cases, 31; namely, the cases of Bernhardt, Pitres and Vaillard, Hayem, Pribytkow and Brochain, because there were marked sensory phenomena present; the cases of Vierordt, Strümpell, Eichhorst, Diller and Meyer, because their course was chronic or prolonged; and the case of Roth, because it was not ascending in type. As a collection of cases strictly illustrating what Landry meant by his paralysis, the retained list of Bailey and Ewing is a happy hit; but such a method of classification, it seems to me, is of no value whatever, unless it can be clearly shown that the rejected cases were absolutely unrelated, clinically as well as pathologically, to those of the retained list. In other words, any man may describe any sort of a symptom-complex, and the chances are ten to one that a tolerably good list of cases that will correspond may be gathered together. The real question is not as to the number of cases that correspond merely with Landry's definition, but as to the reliability of Landry's description as a definition of a unique and distinct disease, and of the relationship of it, if any, to more extensive disease processes or of the divergent, rejected cases to it.

Viewing the question from all sides, then, the following conclusion may be accepted as fairly representative of the modern conception of Landry's paralysis and its relationships. There is a form of nervous intoxication, whether specific or not unknown, in which the lower motor neuron is selected to bear the brunt of the trouble. The character and distribution of the lesion in this lower motor neuron and its associated structures, together with the consequent clinical picture produced by that lesion, are dependent on many factors, chief of which are the virulence of the poison, the duration of the disease and the individual susceptibility of the patient. One phase of this intoxication

is covered by the definition of Landry; other phases are the symptom-complexes that closely resemble multiple neuritis, acute anterior poliomyelitis and acute ascending meningomyelitis.

This being so, it is worth while considering for a moment the differential diagnosis between this intoxication and the well-recognized forms of disease which it so often closely resembles.

If we believe, with Ross, Raymond and others, that a multiple neuritis is the pathologic basis of all these cases of acute ascending paralysis, we must either enlarge our symptomatology of multiple neuritis or contract that of the class of cases now under consideration. Without doubt there are cases so near to the borderline between the two classes of disease that a differential diagnosis is absolutely impossible. Such cases are not numerous enough, however, to afford ground for the belief that acute ascending paralysis is always a multiple neuritis, or to render a differential diagnosis a matter of useless effort. Multiple neuritis is something quite distinct, both pathologically and clinically, from the forms of neurotic intoxication included under the generic term acute ascending paralysis. In multiple neuritis the regular appearance early in the disease of sensory symptoms, usually before the motor manifestations, the paraplegic and more or less symmetrical distribution of the paralysis, the ataxia and other special motor phenomena, the electrical changes, the loss of the deep reflexes, the rapid muscular atrophy, the delirium and other mental accompaniments, and finally the slower course of the disease process and the more favorable prognosis are sufficient to discriminate multiple neuritis clinically. Pathologically the less generally diffuse or focal character of the lesions, the comparative absence of lesions outside of the nervous system, and the comparatively less involvement of the cerebrospinal axis proper with these scattered diffuse lesions are characteristics of polyneuritis. In this disease, moreover, there is decidedly more fever, and the bulbar symptoms are not so obtrusive. The paralysis is not so regularly of the ascending type. The age of the patient and the presence of an alcoholic history are also matters for careful consideration.

From acute anterior poliomyelitis and the various forms of acute ascending myelitis, the differentiation is still less difficult if one keeps well in mind the specially prominent characteristics of acute ascending paralysis. These are the rapidly advancing motor character of the paralysis, the comparative sensory phenomena, the unaltered electrical reactions, the absence of typical muscular atrophy, the non-involvement of the sphincters and the almost total want of fever and febrile manifestations. In infantile palsy the average age of the patient and the peculiarly localized muscular distribution of the paralysis are important aids in making a differentiation. The sensory phenomena, the involvement of the sphincters and the reflex changes distinguish quite clearly an ascending myelitis.

The frank recognition of the toxemic origin and nature of both the typical and atypical forms of Landry's paralysis, will make the way clearer for a rational mode of treatment. The two prime indications are to support the patient's vitality and to eliminate the poison as quickly as possible. For the former purpose, judicious stimulation is demanded. Alcohol, strychnia and the more diffusible stimulants may be cautiously used. For the latter purpose the skin, bowels and kidneys should be aroused into unusual activity. Diaphoresis,

provoked by the use of the hot pack, catharsis with calomel or other rapidly acting and non-exhausting purge, and diuresis with diuretin or some similar agent are all to be employed as promptly as possible. The few remedies that have been tried and recommended in this disease can not be very highly lauded. They have been tried in far too small a number of cases or else they have been given on too much of a mere empirical basis. Such remedies, however, are the salicylates, benzoate of sodium and large doses of ergotin (gr. ii every hour). As might have been expected, mercury and the iodid of potassium have been experimented with. With a history of syphilis, they are, of course, indicated. Soltman reported a case in a 11-year-old girl cured by vigorous inunctions. Less desirable, if not positively irrational, would seem to be such violent measures as strong counter-irritation to the spine with mustard and the actual cautery. Such have been tried, however, and found wanting. Special symptoms, such as the dysphagia, for instance, call for special measures, such as the rectal or nasal tube. In fine, the treatment of Landry's form of intoxication of the nervous system narrows itself down to careful stimulation, rapid, non-exhausting elimination and increased nutrition with absolute rest in bed for a more or less prolonged period.

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SURGERY OF THE STOMACH.*

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We have learned from recent surgery of the upper abdominal cavity that in very many cases diagnosed chronic dyspepsia, and treated hygienically, dietetically and medicinally without permanent relief, the symptoms are not the result of functional trouble or of chronic gastritis that changes the chemistry of the gastric secretions, but are the result of pathologic conditions that interfere with the normal physics of the stomach and duodenum, because of organic and mechanical changes in these organs, caused by gastric or duodenal ulcer, with frequent perigastric inflammation, adhesions and contractions, which predispose to the development of cancer.

While our learned pathologists, in necropsies of patients dying in the large hospitals of the world, tell us that about 5 per cent. have active gastric ulcer, or the scar of an old one, and that in 50 per cent. of these there are perigastric adhesions, that from 1 per cent. to 3.5 per cent. of all deaths are caused by gastric cancer, and that approximately 40 per cent. of all cancers occur in the stomach, they are silent when we inquire, how are we to prevent this intense suffering and these untimely deaths?

It seems that this has been left to the combined surgical pathologist and clinician, who has learned more from *sectio in vivo* than from necropsies. He has learned, by timely operation for gastric ulcer and perforation, pyloric retention and obstruction, and perigastric adhesions, that he may not only cure his patient of gastric hemorrhage, perforation, intense pain and dyspepsia, but may prevent the development of cancer of the stomach, to which these irritations, by interfer-

ing with nutrition and resistance, predispose. Notwithstanding these facts, the life-saving principle of early diagnosis of organic lesions and timely surgical interference is not generally appreciated by most of our good surgeons and physicians, the work in its excellence being mainly confined to but comparatively few men (Robson, Mayo, Murphy, Ochsner, Mikulicz, Czerny, Moynihan, etc.), and even with those who are doing most to evolve what is in the interest of the immediate relief and subsequent life of the patient, there is no consensus of opinion in all instances as to when an operation should be performed, or which of the many operations devised accomplishes the most satisfactory results.

Professor von Mikulicz operates on about four times as many cases of cancer of the stomach as he does of gastric ulcer, while the reverse is practically true of Dr. Mayo; hence von Mikulicz takes this as the measure of the relative number of benign and malignant diseases of the stomach in Germany and the United States. In view of the fact that about 5 per cent. of all necropsies show active gastric ulcer, or the scar of an old one, while in the Vienna Hospital in 61,287 necropsies gastric cancer was found in but 1.5 per cent., I am inclined to the opinion that von Mikulicz's operations are not always timely, cancer having developed in an ulcer or about its cicatrix, for the cancer is usually the subsequent change in the ulcer.

Experience in gastric surgery, when the profession practically appreciates the significance of early diagnosis and timely operation, will convince us that in all countries there is about the same relative ratio in the number of cases of gastric ulcer and gastric cancer. Dr. Mayo diagnoses his cases and operates in the pre-cancerous stage, while von Mikulicz often loses the golden opportunity and operates after cancerous invasion; hence his immediate and subsequent mortality is much greater than in Mayo's cases.

Gastric and duodenal ulcer, with the numerous and varied complications, will finally sustain the same comparative relation to the surgeon and physician that appendicitis does to-day, and the same benefits will be derived from early diagnosis and timely operation. Only a few years ago, most patients with appendicitis refused to be operated on, having been told by many of our physicians and surgeons that the disease could be cured by saline purgation, opium and hot or cold applications. The reverse is true now, and in the near future the same will apply with equal emphasis to surgery of the stomach.

While gastric ulcer and cancer may occur in any part of the stomach, they are usually found in the pylorus, where there is an abundance of lymph vessels and glands, the cancer succeeding the ulcer, though often it does not do so for several years, many cases of ulcer being apparently cured. In the upper or lesser curvature of the stomach the lymph spaces and glands are abundant, and extend through the base of the gastrohepatic omentum and in the stomach tissue to the cardia, with the current from right to left, but in the lower or greater curvature, the lymphatics do not extend far to the left through the gastrosplenic omentum, and the current is probably toward the pylorus, as seems to have been demonstrated by Cunco. It is important that we should know, in pylorotomy and partial gastrectomy, how much of the stomach and the omenta to remove to destroy the cancer-bearing areas and infected lymphatics, except the isolated area of lymphatics in the dome above the gastro-

* Read before the Louisville Clinical Society.