

all occur from wounds in the area of distribution of the fifth nerve, the hypothesis of a reflex paralysis has been invoked, but it explains nothing. Tetanus is an infectious disease, caused by a microbe. This microbe gains entrance in these cases at a point where it can exert a direct action on the seventh nerves, and its effect is to paralyze one set of nerve fibers (the seventh) while it causes tetanic spasm in neighboring nerves (the motor branches of the fifth). It might well be called *tetanus paradoxus*. Some observers have claimed that the trismus also has a tendency to be unilateral, or at least more marked on the side of the wound.

Vaillard (quoted by Bourgeois) denied that the facial nerve is truly paralyzed in these cases; he seemed to think that it was in tetanic spasm. It has been suggested that the asymmetry of the face is not due to paralysis, but to excessive tetanic spasm in the muscles of the opposite side.¹⁷ Such an explanation, however, falls completely to the ground in those cases in which both facial nerves are paralyzed. Moreover, it can not explain the typical inability to close the eye and to wrinkle the brow and all the other classical symptoms of Bell's palsy. It has also been suggested (by Bernhardt and by Guterbrock) that the paralyzed muscles are also contracted; but this does not appear to be so on close examination. The passive raising of the lips by the breath on expiration, and the lifting of the flaccid lips by the fingers in attempts at speaking (a characteristic act) are clearly indicative of paralysis.

Some of the clinical observations here recorded have been confirmed by experimental work, especially with reference to the seat of the wound. Brunner¹⁸ injected the tetanus toxin into guinea-pigs and obtained symptoms on the same side into which the toxin was injected; and if it was injected into the median line, the symptoms were bilateral.

The pathology of this facial paralysis in cephalic tetanus has not been satisfactorily determined, although a number of autopsies have been made. The results, however, are negative. The reactions of degeneration have not been obtained. This was so in Willard's case, in which I made the tests. In the case reported in this paper the opportunity to make an electrical study was not favorable.

There has been no attempt in this paper to go into an exhaustive review of the literature. Willard's paper, already quoted, gives a long list of references and it is easily accessible. The attempt has been made here, however, to collect all the cases in which the facial paralysis was bilateral. Willard, in a review of 75 cases of cephalic tetanus, found but 2 in which both seventh nerves were involved. With the assistance of Dr. C. D. Camp, I have studied about 25 additional cases of *kopftetanus* reported in the last ten years, or not included in Willard's list, and have found a total of 7 cases of facial diplegia, as abstracted above, the present case being the eighth.

Important papers on the subject of cephalic tetanus are by Villar,¹⁹ Bernhardt,²⁰ Dard,²¹ and Sapincourt,²² the last two of which have not been accessible to me.

17. Gosselin: *Gaz. d. Hôp.*, 1880, ix, p. 65.

18. *Deut. Zeit. f. Chir.*, xxx, p. 574; also *Beiträg f. klin. Chir.*, vol. 9, p. 269.

19. *Gaz. de Hôp.*, 1888, p. 1357.

20. *Zeit. f. klin. Med.*, 1884, p. 410.

21. *Thesis*, 1896.

22. *Rev. Med. de Normand*, 1904, iv, 333.

ATAXIA OF CENTRAL ORIGIN APPEARING IN CHILDHOOD.*

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This paper is a brief consideration of a form of central nervous disease previously described under the titles of hereditary ataxia, cerebellar ataxia, and not infrequently under the heading of Friedreich's disease. It varies, in certain particulars, however, as we shall see, from the usual conception of what should constitute the affection bearing Friedreich's name. In view of its relative rarity, the number of cases of this affection coming under the observation of any single observer is naturally small.

The clinical characteristics of this affection stated briefly are: Muscular inco-ordination commencing usually in childhood, and, as a rule, first affecting the muscles of the lower extremities, thence extending to the upper extremities, and to the muscles of the trunk, head, larynx, tongue and eyes; slowness of muscular response to volitional impulse; swaying, unsteady gait; slow, hesitating or deliberate speech, sometimes of nasal character and occasionally explosive; nystagmus; oscillation or tremor of head, body or extremities; and certain involuntary movements, which may or may not be of choreiform character, and which may occur independently or may be associated with volitional intended movement of some other part of the body; lateral curvature of the spine; deficient energy in carrying out voluntary movements; and, in an advanced period of the disease, paralysis, muscular spasm and contractures.

To this symptom-complex may be added, during the course of certain cases, various other symptoms not sufficiently invariable to be considered characteristic, but nevertheless to be regarded as inherent, although less common, phenomena. Among these may be mentioned vertigo, headache, optic atrophy and impairment of pupillary action and of the movements of the ocular muscles, apathy of facial expression, a tendency to involuntary and unprovoked laughter, loss of muscular tone, sensory disturbance, peculiar deformity of the feet, trophic changes in muscles, and very rarely interference in the action of the sphincters and trophic changes in the skin.

Certain other phenomena must be regarded in the light of complications, since they are clearly due to involvement of those parts of the central nervous structure, primary affection of which would produce a clinical picture totally different from the one we are considering.

Such is the occurrence of psychical phenomena, depreciation of intellectual power, or epileptiform attacks. In this symptom-complex will be recognized many of the symptoms that characterized the well-known cases described by Friedreich in 1861, and which have since been identified with his name. Since then many cases have been reported differing so widely from the type originally described that certain observers have been led to believe that they were dealing with an affection of different nature. Several of these cases were undoubtedly not instances of Friedreich's disease, and even in the light of our present knowledge of how manifold may be its symptom-complex, bore in their clinical characteristics no resemblance whatever to the affection. The

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majority, although varying in certain features, are, for the most part, so strikingly similar that no doubt of their close relationship can be entertained. Neither Friedreich's cases nor any other of the early instances reported can be used as a standard either on the basis of their clinical or pathologic character. An adequate idea of the affection and its variations can only be obtained by a study of many cases at different stages of the process and by careful comparison of the clinical phenomena with the changes found in the central nervous structure where systematic pathologic examination has been made.

Not all the symptoms of the affection develop in its early stage. Several are late phenomena. Naturally, therefore, the number of typical symptoms presented by any given case depends, in great measure, on the duration of the affection. Then some of the later symptoms tend to mask or obscure the earlier phenomena and thus alter the clinical picture. Finally, much depends on the extent of the process in the central nervous structure. Obvious as these facts may seem, they have not been given due consideration by many observers. In this connection I venture to assert my belief that the condition of the tendon reflexes, so long a bone of contention between the adherents for and against the maintenance of Friedreich's original type, is solely a matter of the duration, location or extent of the central lesion; and that the presence or absence of the knee jerks is, in itself alone, of no differential diagnostic value in this affection, and only indicates to us whether the process has or has not involved certain definite portions of the nervous structure.

Several cases, all in the early stage, because seen in young children from 3 to 10 years of age, have come under my observation. Five concern children 4 and 5 years old, in all of whom the affection was noticed first when the child began to walk, by its failure to acquire the power of equilibration. The brevity of the time allowed unfortunately forbids the giving of these cases in detail.

In all of these five cases the gait was unsteady; in three extremely so, these children walking with feet wide spread, from time to time hesitating, halting in their forward progress to sway several seconds over their center of gravity, as if about to fall, then moving forward, often with several short, quick steps in their effort to maintain their balance, approaching their objective point in a more or less indirect course.

In three of the five, well-marked ataxia of both upper and lower extremities was present, and was in all much more marked in arms and hands than in legs. In two speech was affected, in one associated with distinct involuntary accessory movement of lower facial muscles; in two speech was not acquired; in the fifth it was not yet abnormal. None of the other symptoms have yet appeared.

These cases are mentioned together because of the early period at which they came under observation, but also because in no single instance were the knee jerks lost.

For the privilege of reporting these cases I am greatly indebted to Dr. Bullard and to Dr. Thomas, from whose clinic at the Children's Hospital the cases come.

To these I wish to add a child of 10 years, which I saw in Senator's clinic, in which the knee jerks were greatly increased, and, in addition to the above well-

marked symptoms, choreiform movements of head and arms and nystagmus were present.

The six cases reported by Friedreich in 1861 presented many, though not all, of the features of our symptom-complex. Being the first cases to be clearly recognized as a clinical entity, apart from the ataxy of Duchenne, and so reported and carefully described, they attracted wide attention and were discussed everywhere. This universal interest, fostered by several later papers by Friedreich, and increased by subsequent reports of apparently analogous cases by other observers, soon led to the adoption of Friedreich's name as the synonym of the disease. Then appeared, on the part of certain writers, an attempt to construct a rigid standard, based on the symptoms of Friedreich's cases and of those reported soon after his article appeared, to which it was required that all cases should conform. For a brief period all went well. Soon, however, as the field of observation enlarged, there began to appear instances differing materially in features of their symptom-complex from the clinical picture considered by these observers as characteristic of Friedreich's disease.

Cases appeared, the apparent origin of which was not at puberty, but much earlier; others also appeared of later origin. In some the knee jerks were not absent, but present, and even increased. Impairment of vision, disturbance in action of the pupils and external ocular muscles and other evidence of cranial nerve involvement occurred, while the not very infrequent presence of disturbances of sense gave evidence of occasional participation on the part of the sensory sphere.

Among these instances and possessing nearly all of the phenomena of our symptom-complex in most pronounced degree are the cases reported by Everett Smith, Menzel, Nonne and Sanger Brown. These cases are of exceptional interest on account of the extreme degree the affection attained and the frequent occurrence of the more unusual symptoms, and especially bulbar phenomena. Smith's cases are of special interest to us, because of five children affected in a family of thirteen the affection began in four before the tenth year. Of the greatest importance, however, is the fact that we have, in certain cases of each series, the records of the pathologic changes at the basis of this symptom-complex.

Six autopsies are presented for our consideration. Nonne's case showed general smallness of the entire nervous system without signs of degeneration. This case as reported is an anomaly. If this was, as Nonne thinks, a congenital condition, it can not be explained why the symptoms did not become marked until well along in adult life, and then were markedly progressive up to time of death.

In all of the other cases extreme degeneration of the posterior columns of the cord was found, together with marked involvement of the direct cerebellar tract (if we assume, as seems to me probable, and as I think the plate of the cross-section of the cord indicates, that a part of the degenerated fibers, in the lateral columns of Smith's case, were fibers of this tract). The condition of Clark's column is also unfortunately not mentioned in this case. It is, however, specifically stated that cells of the gray matter were far less numerous than usual, especially in the posterior portion, and in some places only a scattered few were to be seen. This, taken into consideration with the fact that the cells and fibers of

this column of the gray matter of the cord have been affected in nearly every instance where a pathologic examination has been made, make it exceedingly probable that Clarke's column was involved.

If, therefore, we may include this case, we find this column also affected in every one of these five cases. In every case the posterior nerve roots were more or less degenerated. The central canal is usually blocked with cells and its epithelial lining lost. In addition to these well-defined lesions, slight changes were found in individual cases in the rest of the gray matter and in the anterior roots. In two instances the crossed, in one the direct, pyramidal tracts were involved in the degenerative change. In all but one of these cases there were found abnormal conditions of the medulla and cerebellum, consisting of a reduction in size as a whole, atrophy of medullary fibers belonging to the cerebello-spinal system, and in two instances fibers of the cerebello-cerebral tract in the brachia conjunctiva, and atrophic changes and reduction in size and number of the ganglion cells of the gray matter, including in Menzel's case some of the cranial nerve nuclei, the spinal accessory, hypoglossal, facial, and the motor nucleus of the fifth.

The finer changes are characterized by a loss of the medullary sheaths, a shrinking and granular degeneration of the ganglia cells, by a proliferation of the neuroglia and an increase in the glia cells. In other words, by a parenchymatous degeneration and an interstitial proliferation.

While it is not my intention, in this brief paper, to discuss the etiology of this affection, it is nevertheless of the greatest interest to note that that portion of the cord, at least, which is the seat of the greatest intensity of the process is that part which is formed last, and which is nearest in point of time, and perhaps in condition, to the embryonic state, namely, the columns of Goll, Clarke and Burdach. Of the elements composing their structure, the matrix, or neuroglia, remains the nearest to the embryonal state. The tendency of embryonic or immature tissue to proliferate, in response to trauma or other irritant, is well known. It may be that the primary step in this affection is a proliferation of the neuroglia, followed by a degeneration of the medullary fibers, which are, perhaps, through their immaturity or inherent lack of vitality, only too ready to degenerate.

The sclerosis of the direct cerebellar tract is, I think, to be regarded as purely secondary to the atrophy and disappearance of the cells of the column of Clarke, and that, whatever part the neuroglia may elsewhere play in the process, it is here increased only as a result of the loss of the medullary fibers.

Just why the pyramidal tracts should degenerate as they have in certain cases is much more difficult to answer. It seems possible to me that, in cases where there is extreme proliferation of the neuroglia in the region of the transverse fibers of the pons, the pyramidal tracts may be injured as they pass through these transverse bundles from the middle cerebellar peduncle. This does not explain, however, those instances where the pyramidal fibers in the cord alone are implicated, with no involvement above their decussation. Interesting, however, is the occurrence of ascending degeneration of these tracts in certain cases of gliosis and cavity formation in the cord. Here it is, also, of interest to note the singular proneness of Clarke's column to degenerate in these cases of syringomyelia, and the much greater frequency of involvement of the posterior columns in the gliosis and secondary degeneration in this affection.

Proliferation of glia tissue, in cases associated with structural defects, either congenital or acquired, in the cord, is, of course, well known. Of the greatest interest it seems to me, in this connection, is the fact that structural abnormalities of the cord have been found several times in Friedreich's disease; in fact, in one of the cases reported by himself two symmetrical canals were found in the lower dorsal region nearly corresponding to the columns of Clarke on each side; and in a case reported by Mackay, a picture of which I hope to show you later, marked structural anomaly was present. In our series, in Smith's case, an abnormal canal was present in the lumbar region. These defects lend strong support to the view that we have to do with, a process supervening on a developmental insufficiency, either in structure or inherent vitality, of the nervous system. This view is still further strengthened by the frequent strong family tendency to the disease, by the occasional history of its occurrence in other generations, and by the not infrequent presence in other members of the family, not affected by the disease, of marked anatomic, physiologic or psychological defects. Marie, in 1893, attempted to establish a differentiation of certain cases from Friedreich's disease on the basis of a difference in the clinical symptoms, laying especial stress on the age of onset and the condition of the knee jerks. A study of a large number of instances of the symptom-complex does not seem to support the contention.

The variations in the phenomena are seen to be merely a question of degree, the variations so merging into one another as to allow of no abrupt distinction. Neither do the results of pathologic examination permit of any sharp differentiation. Even in one of the first cases ever reported we find invasion of the lower portion of the medulla; in others the upper part as well; in still others implication of the pons and cerebellum. Moreover, some of the symptoms of Friedreich's disease are distinctly cerebellar in character, and, while I do not believe it essential that the cerebellum be affected for the production of these phenomena, they being also possible in disease of its connections with the rest of the nervous system, I believe it would be found affected much more frequently if microscopic examination of its structure was made.

Moreover, many of the phenomena of our symptom-complex have occurred in cerebellar atrophy, hemorrhage and other lesions, in the human being, and have been features of the phenomena following atrophy or experimental lesions of the cerebellum in animals. I regret that the time does not permit giving the details of these instances.

I may mention, however, among many other interesting instances, the results of the experiments of Luciani, in Italy, in which partial or complete extirpation of the cerebellum, in monkeys and dogs, was followed by muscular inco-ordination, loss of muscular energy, loss of muscular tone, tremor and oscillations of different parts of the body, curvature of the spine, forward inclination of the body, slowness of voluntary movements, nystagmus and muscular contractures; all of which symptoms are frequently met with in Friedreich's disease, and the most of which are almost invariably present.

DISCUSSION.

DR. C. F. WAHRER, Fort Madison, Iowa, asked whether the choreic movements mentioned by Dr. Fairbanks included the movements of the so-called *tic convulsif*; also whether there were any athetoid movements and if diminished nutritive change was noticed in the nerve tracts.

DR. H. E. GARRISON, Dixon, Ill., asked if Dr. Fairbanks has ever noticed any consumptive tendency in connection with

these children. The only case Dr. Garrison has seen which resembled it was a 5-years-old child whom she was called to see. A consultation had been held and the child was to be operated on for tubercular peritonitis. Dr. Garrison found the child completely paralyzed except in the left hand. She objected to the operation, for she saw no signs of tubercular peritonitis, and asked the physician to examine for tubercular organisms in the sputum and a few bacillus were found. The child, three months afterward, visited Dr. Garrison and remained for three weeks. When she came she was able to creep around. By judicious manipulation the development of the atrophied muscles was aided and the child is now apparently in perfect health and has since been able to enter school.

DR. A. W. FAIRBANKS said that the movements noted were not those of *tic convulsif*. The athetoid movements did occur, but so rarely that he did not include them in his summary of the signs. He is inclined to think that they were not athetoid movements, but rather choreic movements of slow character, simulating athetoid movements. There was extreme atrophy and degeneration of the peripheral nerves. Dr. Fairbanks thinks that there is no ground for the assumption that tuberculosis is an etiologic or pathologic factor. Tubercular processes are sometimes terminal factors of the disease, but do not form an integral part. He should not consider that the case mentioned by Dr. Garrison belongs to this class of disease because of its marked improvement. Instances do occur in which the disease does not progress for a time, but sooner or later it progresses and the patient dies. He believes that the more these cases are studied the more it will be found that they originate in childhood. The very slowly progressive symptoms in the early stages are overlooked by the parents.

COLLODION AS A DRESSING AFTER INTRANASAL OPERATIONS.

A PREVENTIVE OF POST-OPERATIVE HEMORRHAGES.*

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Since the publication of my first paper on this subject,¹ some improvements have been made in my method of using collodion as a dressing after intranasal operations. These were partly evolved from my own experience and partly from the suggestions of my colleagues.

Dr. E. Rixford of San Francisco suggested the use of compound tincture of benzoin as a substitute for collodion. I found it very reliable as a preparation of the wound for collodion; it dries the tissue nicely, so that the collodion sticks to it much better.

Dr. James F. Smith of San Francisco uses pieces of gauze instead of wisps of cotton. I have followed his suggestion.

Dr. C. W. Richardson,² Washington, D. C., applies the collodion with a probe; I had tried that before I selected my present method, but discarded it.

That the usual after-treatment is not satisfactory to many was well expressed lately at two European congresses where this question was discussed.

I therefore take the liberty to describe my method. After intranasal operations, we must choose between packing the nose or risking a postoperative hemorrhage. A reliable, firm packing of the nostril causes great discomfort and often a sleepless night, while the collodion dressing allows the patient to breathe even through the nostril operated on, but covers the wound against infection from the air and prevents secondary hemorrhages.

I have now used this dressing in 243 cases with perfect results in 233 cases. I had to replace the collodion dressing after a few hours in 4 cases. In 3 of these the hemorrhage occurred on the posterior end, in 1 case of polypoid degeneration of the erectile tissue in the anterior half. In 6 cases, or less than 3 per cent., the collodion dressing was not sufficient and had to be replaced by packing. The hemorrhages occurred in 5 cases from the posterior end of the lower turbinal. One of these five patients was a bleeder, with hypertrophy of the left ventricle, high blood pressure and traces of albumin in the urine, indicating the beginning of a shrinking kidney. The sixth case was a fibrous polypus from above the posterior end of the lower turbinal on a place on which I could not properly apply the collodion dressing.

I am aware that this method is far from perfect, but I hope that if some members of this Section take the matter up and try to improve on it they will perfect it to such a degree that there will be no more postoperative hemorrhages.

TECHNIC.

After I have finished the operation and stopped the bleeding with adrenalin, I clean the field carefully, wipe over the wound and surrounding tissue compound tincture of benzoin on a cotton carrier, then cover the wound with a piece of sterilized gauze (about 1 by 2 cm.). I am particularly careful to press this around the posterior end, as from that point hemorrhages occur most frequently. These pieces of gauze I keep on hand compressed, so that they are stiffer and can be more easily introduced. I drop the collodion slowly on this gauze, beginning on the posterior end, while my assistant blows hot or cold compressed air into the nostril to quicken evaporation. The collodion and gauze form a white, firm membrane, which fulfills two purposes: it protects the wound against infection from the air, and prevents secondary hemorrhage. As an extra precaution, I instruct the patient to keep a little cotton in the entrance of the nose on the way home. This absorbs the little oozing of blood and serum which exists even when the nose is packed. The cotton may be changed as often as it is soaked; but the patient may take it out at home so that he can breathe through that nostril. This dressing I leave in the nose from four to six days; if left longer the edges become loose and too much discharge is kept back.

In order to remove the dressing, I cocaineize the nose and apply some peroxid of hydrogen, which helps to loosen the dressing, which I then take out with a pair of pincers; frequently a few drops of blood follow this procedure.

For dropping the collodion on the wound an ordinary eye dropper will not do. The collodion would flow too quickly. It must be dropped on the wound slowly and carefully, so that it does not run down into the pharynx, an accident which would make the patient gag and cough. One might use a fine Eustachian catheter, on the wider end of which a small rubber bulb is mounted, fastened airtight with a rubber band. One filling of the tube with collodion is usually enough to cover the whole wound. I use a small metal tube 9 cm long, 1 mm. thick, with a tulip tip on one end, to fasten the rubber bulb. This tube must be cleaned out thoroughly after use.

DISCUSSION.

DR. OTTO T. FREER, Chicago, regards dismissing patients after intranasal operations without tamponing as permissible after operations on the middle turbinated body, but a dan-

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1. Arch. of Otol., vol. xxxi, No. 5, 1902.

2. The Laryngoscope, vol. xiv, No. 9, 1904.