

from the labor of their inmates, having been built from monies received from a tax imposed on liquor dealers, or a license fund, and be independent of the tax payer or of State support.

These places would receive the classes who now are sent to jail, and that other class who are neglected until they have passed into the chronic stage and have become inmates of prisons and insane asylums.

A very large proportion of these several classes could be made self-supporting while under treatment, and in many cases be an actual source of revenue. The hospitals would naturally be divided into two classes. The first would receive the better, or less chronic cases; the second would have the incurables, and those whose recovery was deemed more or less doubtful. In one case the surroundings and discipline would be more adapted for the special inmates than in the other, but the same general restraint would be followed in each.

In both recoveries would follow. A large class would be restored to society and become producers. In the second, cases would be housed and made to take care of themselves, which would be an immense gain to society in economy and safety.

Private enterprise should be encouraged by legislation to provide smaller hospitals for the better class and those who would be unwilling, or whom it would be undesirable to compel to enter public asylums. Here the commitments should be both forced and voluntary, and the restraint combined with the fullest and latest appliances of science for the end to be accomplished, blending seclusion and good surroundings to build up and make recovery possible.

The first step is to recognize the fact that the inebriate, whether continuous or periodic, has to a greater or less degree, forfeited his personal liberty, become a public nuisance and an obstacle to social progress and civilization. Second, that he is suffering from a disease which affects society and every member of the community in which he lives, and from which he cannot recover without aid from other sources, making it absolutely necessary that he should be forced into quarantine on the same principle as the small pox or yellow fever patient. This is simply carrying out the primitive law of self-preservation. Naturally, the money to accomplish this shall come from the license revenue, on the principle that every business should provide for the accidents and injuries which follow from it. Railroad companies and other corporations are required to pay damages for the accidents which follow their business, and this is conceded to be justice. But to-day the tax on the liquor traffic is used to support courts and jails where the inebriate, by fines and imprisonment, is only made worse or more incurable. Thus, literally, the business of selling spirits is increased by the almost barbaric efforts of courts and jails, and every person so punished is made a permanent patron of that business. Against this all the teachings of science and all practical study utter loud protest.

The practical success of workhouse hospitals for inebriates is demonstrated in every self-supporting jail and State's prison in the country where the obstacles are greater and the possibilities of accomplishing this end more remote. This can also be seen in asylums for both insane and inebriates, in the various sanatoria and hospitals through the country where the capacity for self-support and the curability of these cases are established facts.

More than that, these hospitals would relieve society of great burdens, of loss and suffering, the diminution of the number of the inebriates indeed become a practical certainty, the extent of which we can have no conception of at present.

It is impossible at the present time to estimate the beneficial results that would follow a systematized plan of thus housing and treating the inebriate, but there are positive indications that its effect would be felt in all circles. One of the great fountain heads of insanity, criminality and pauperism would be closed, and a new era would dawn in the evolution of science.

HINTS UPON THE PATHOLOGY OF SO-CALLED FRIEDREICH'S DISEASE, BASED UPON THE STUDY OF A SERIES OF TWENTY-THREE CASES.

Read in the Section of Neurology and Medical Jurisprudence, at the Forty-third annual meeting of the American Medical Association, held in Detroit, Mich., June, 1892.

BY SANGER BROWN, M.D.,
OF CHICAGO.

Of the utmost importance in the present condition of the subject is any addition to the stock of exact clinical and pathological data in the study of degenerative diseases of the central nervous system, for it is only by the examination of such data that a durable and reliable basis of classification can be reached for the guidance of the practitioner.

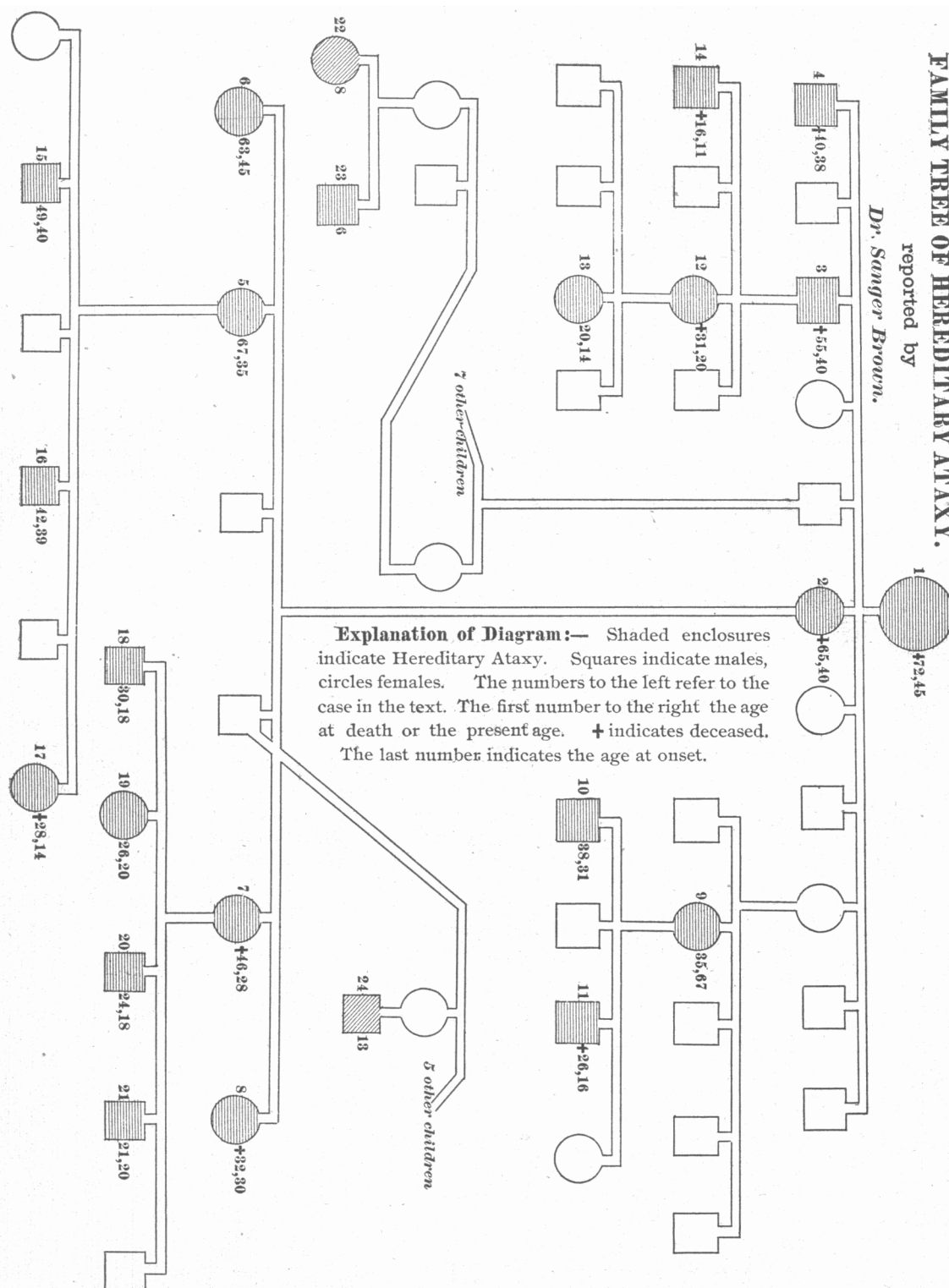
There has been such a large accumulation of recorded facts bearing upon the subject within the past few years, that the conclusions previously reached from the data then at hand, are no longer tenable.

Two broad divisions, however, may so far be safely made, one in which heredity can be demonstrated to play a prominent part, and one in which this influence is not apparent. Of the former, the so-called Friedreich's disease may be taken as a type, and of the latter the ordinary form of tabes, and unless it should be demonstrated that heredity plays an important part in the progressive form of spinal muscular atrophy, and disseminated sclerosis, the pathological process giving rise to the symptom complex, commonly described as Friedreich's disease, must, I think, at the present time, be regarded as constituting the sole instance of hereditary degenerative disease of the central nervous system. In this disease the essential pathological features are, that certain tissues have derived such a deficient vital endowment from the parent, that they undergo a more or less premature, rapid and extensive degeneration, and further, the effect of this process is mainly, if not exclusively, confined to the so-called upper nerve segment, and to the conducting filamentary process, and not the body of the cell of this segment; different cases and series of cases presenting considerable variation both in the extent, degree and period of onset of the pathological process.

That in the future there may be discovered certain laws determining the significance of certain variations, such, for instance, as whether or not the knee jerk is lost, retained or exaggerated, or whether or not there is nystagmus, optic nerve atrophy or integrity of the sphincter, is quite possible; but existing data do not, I think, render an attempt to found a classification upon these differences at present feasible.

With this view of the subject, the symptom complex marked out by Friedreich in 1861, may be taken as the first link in the chain, and up to the present time the report by Tooth, of London, in 1891, of four brothers affected with spastic paraplegia without ataxy, but with affection of the voice and sphincters,

affection of the voice, would strongly suggest, if not indeed, positively demonstrate that they properly belong in the same category, and this being admitted the cases reported by Tooth should also be included, for there is as much difference almost between my series and Friedreich's, as Tooth's series and mine.



and no other important symptoms, as the last link; all other cases, including those of this series, forming intermediate links. For notwithstanding the differences existing between this series and that described by Friedreich, the heredity, the ataxy, the marked

The comparative absence of the various neuroses among the relatives of the individuals composing this and other series, and among the individuals themselves, has suggested to my mind the probability that the primary defect might be rather in the

accessory than in the essential nerve elements, for it is a matter of general belief among neurologists that if hereditary defect of the nervous elements is proven to exist, this is apt to be manifested by the appearance of various neuroses in the family so affected. Here we have, indeed, marked evidence of an hereditary degenerative process affecting the nerve elements, but if the above considerations are valid, not commencing in them. This view is rather supported, also, by the consideration that the defect being transmitted in and confined to a protoplasmic cell, the whole cell ought reasonably to suffer to some extent at least, and furthermore, this view is harmonious with the assumption that the inherited defect is confined to the connective tissue cell, which is effected throughout, but inconsistent with the theory that the primary defect is in the nerve cell, for in the latter case we have to conceive of the inherited defect as being arbitrarily restricted to a mere part of a cell, to wit: the axis cylinder, and that in the face of the generally accepted belief that the nerve cell and its processes are continuous and homogeneous. Then, too, the frequency with which there is considerable jerkiness in the various movements, including nystagmus, suggests a pathological analogy between this disease, and disseminated sclerosis, in which the primary pathological process almost certainly commences in the connective tissue element; in the former the process being more general and less intense than in the latter. In disseminated sclerosis it is easily conceived how a motor nerve current may be suddenly brought up in full career, so to speak, against a sclerosed patch, and with momentary interference force its way through, and thus on to its destination with a corresponding affection of movement. In Friedreich's disease a similar obstruction, though less concentrated and arresting the current more gradually, may be hypothecated with a corresponding motor result. From the foregoing considerations I deduce the corollary that motor defects in this disease are mainly due to interference with the centrifugal paths of the upper nervous segment.

The accompanying diagram represents a series of cases of hereditary degenerative disease of the central nervous system considerably more extensive both in regard to the number of individuals affected, and the number of generations through which it has extended than any that has hitherto come under my observation. I presented essentially the same series a few months since to the Chicago Medical Society, but was then unable to present any of the cases, as I had hoped to do. I am very glad to be able to present two of the cases now, because I am well aware that where a report so extensive is made, so circumstantial, and withal, departing so widely from previous reports, as to rudely disturb, perhaps, existing theories, the skeptical might naturally be expected to mistrust either the capacity or honesty of the reporter.

I have had four of these cases (18, 19, 20 and 21) under observation over a year, and two more (9 and 10) for about nine months, and still another (22) I examined thoroughly three months since, that is since reporting the series to the Chicago Medical Society, and since that time also two cases (5 and 9) have died (and unfortunately, I was unable to get an autopsy). Two others (5 and 15) living at a distance, were thoroughly examined for me by Dr. Norman Bridge, so that in nine of the cases the exam-

ination may be regarded as fairly satisfactory.

In all the other cases I have been able to get a definite enough account of the symptoms to enable me to make a diagnosis, and this is not such a difficult matter, because when the disease is well developed it has so many prominent diagnostic features, and because the families among whom it has been distributed have been composed of people of good social position, of good education and superior intelligence.

Case No. 18.—Male, single, thirty years of age, business man of correct habits, with an excellent family history aside from this disease, the hereditary relations of which are readily seen by reference to the chart. With the exception of the effects of the disease now under consideration, the general health has always been good, and the patient, from his childhood up, showed more than an ordinary degree of bodily and mental vigor, but these characteristics were judiciously directed, so that they do not appear to have had any etiological bearing upon his case, and I merely state them so definitely in order to make it clear that in his case, at least, development was normal.

In this case, as in many others of the same kind, the symptoms developed so insidiously, that it is impossible to fix upon a very exact period as marking the onset of the disease. The patient thinks, and this opinion is shared by some members of his family, who, like himself, have made a close study of the subject, that at the age of puberty there was a greater affection of the voice than could fairly be attributed to that physiological phenomenon alone; and it is quite certain that at eighteen he could not walk steadily when fatigued, and would communicate a perceptible movement to any moveable object against which he might happen to support himself.

Weakness did not appear in the legs until several years later, and it has always been distinctly subordinate to the ataxy. Ataxy did not appear in the arms until about three years after making its appearance in the legs. An increase in the knee jerk was an early symptom no doubt, because quite early in its course the disease was pronounced spastic paraplegia by experienced and eminent physicians. A troublesome tendency to choke was a comparatively early symptom, and this has continued down to the present time. There have been no sensory symptoms of any kind, no affection of the sphincters, and no muscular atrophy or trophic disturbance, and no disorder of the sexual functions. For the past year the body weight has been about stationary, but for several years prior to that there had been a gradual decline amounting to about twenty pounds.

The ataxy, impairment of vision (of such a character that patient could read best in a dim light), difficulty in articulation and weakness have gradually increased, sometimes more and sometimes less rapidly, for the past thirteen or fourteen years, until the patient has reached his present condition.

The patient cannot walk without the assistance of another person, and all the time has a marked subjective feeling of insecurity, as if his head must certainly fall violently to the ground. There is distinct weakness of the legs, but I know of no good way of exactly estimating its degree. The knee jerk is much exaggerated, and equal on the two sides, and there is ankle clonus. The skin reflexes appear normal, excepting the cremasteric, which is perhaps subnormal. There is marked ataxy in all voluntary movements, and there are associated movements extensive in range and distribution. Thus, for instance, a voluntary movement of the hand and arm often sets up associated movements in the opposite hand, the head and face. There is ataxic disturbance in the muscles concerned in articulation, including the tongue, with corresponding defect of speech.

There is marked impairment of vision, due to optic nerve atrophy, vision being 20-200. There has been diplopia at times of a few days' duration. There is ptosis when the patient is at rest, but the lids are often voluntarily raised so high as to show the sclerotic above the iris. There is lagging of the right external rectus to the extreme right, but no incoordination in the external ocular muscles, and no nystagmus. The pupils respond to light, accommodation and stimulation of the skin of the neck, but more slowly than normal. There is not much peripheral limitation of the visual field, but there is almost complete color blindness, red only being distinguished with any degree of cer-

tainty. Closure of the eyes has no material effect upon the ataxy.*

Case 20.—Brother of 18. Age 25, business man of correct habits, and during boyhood a frequent winner of prizes in athletic contests. Excepting that the disease appeared later, has advanced less rapidly, and that the range of tissues involved is more limited, the history of Case 18 may be used for this one. The tendency to choke is absent, and though the first symptom appeared at 18, the arms are not yet much affected.

Here there is marked ataxy in the legs, as shown in the gait, but it would be difficult to demonstrate weakness. The ataxy is not much increased by closure of the eyes, and as already stated, the arms are not yet much involved, and neither have associated movements appeared to any considerable extent. The vision is reduced considerably, and is much better in a dim light than in a bright one, but one would hardly make a diagnosis of optic nerve atrophy from an examination of the disc alone. There is ptosis at rest, and the lid is often raised too high by voluntary effort, but there is no nystagmus. There is obvious, but not great, impairment in articulation.

In *Cases 10 and 17* there appears to have been something like lightning pain, not severe, however; and in 10 there is considerable insufficiency of the rectal and vesical sphincters, though in every other respect the case is practically the same as 18. I might add, too, that in Case 18, during the last months of life, there was marked melancholia, with great emaciation.

Case 22 is a rather backward girl of 8, whose parents at the age of 5 first noticed a tendency to walk upon the toes, which has steadily increased, so that at the date of my examination, there was observed some permanent spastic contracture of the right leg at the knee, with ankle clonus on that side, and greatly exaggerated knee jerks on both sides. No atrophy, ataxy or apparent weakness, and no disturbance of sensation.

For a more complete report of the whole series I must refer to the February number of this year of the *North American Practitioner* and the *Chicago Medical Recorder*, from which it will appear, I think, that Case 18, as here described, with the exceptions here noted, is fairly typical of the series.

Discussion.

Dr. C. K. Mills, Philadelphia, Pa.:—These cases are of great interest, and particularly the part of the paper of Dr. Brown which gives us his view as to the pathology of the disease. Friedreich's ataxia, and a series of cases of a different sort have, I think, a certain bond in common. It is one of the most interesting matters in connection with the discussion of these cases to recognize this bond of union. We have in the wards at the Philadelphia Hospital cases of Friedreich's ataxia, and half a dozen other forms of disease which, I believe, are all hereditary; for instance, the so-called hereditary chorea, idiopathic muscular atrophy, and the association of muscular atrophy with pseudo-hypertrophy, etc. We have had, in my own personal experience also, a few cases of ordinary spastic paraplegia, and of the ordinary type of locomotor ataxia which were, in so far as the history is concerned, hereditary. A most interesting case is a young man, 26 years old, who presents a typical case of the ordinary form of locomotor ataxia, with lancinating pains, disorders of sensation, with ocular and bladder symptoms, etc. In this case the disease began at the age of 14 years. Cases of combined sclerosis or ataxic paraplegia occasionally begin early in life. We should learn to take a more philosophical view of these cases, and while we differentiate more and more the nervous diseases into types, we should be careful not to lose sight of the bond which connects them. A large number of them are due intrinsically to the same tendency to arrest of development. To many of the forms of hemiplegia and spasmodic affections of children these same remarks may apply.

As to the pathology of the disease, I think the views advanced by the author of the paper are of the greatest interest. Dr. James Hendrie Lloyd, of Philadelphia, and others maintain that there is a similarity between cases of Friedreich's ataxia and syringomyelia. Dr. Lloyd believes that if the cases were closely studied a certain percentage of them would be found to be cases of syringomyelia. In a tabulated report by Dr. Crozier Griffith of some 150 cases,

a few of the autopsies indicate the pathology to be of this character. In several cases supplementary central canals were apparently present, as well as enlargements of the original canal; or the canal itself was patulous, which, of course, is not the case as a rule.

Dr. F. X. Dercum, Philadelphia, Pa.:—I have seen a number of cases present typical Friedreich's ataxia in which there was loss of knee jerk. We have in our wards in Philadelphia a patient in whom a similar degenerative change in the cord doubtless exists, but with the ataxia there is also associated marked chorea. Whether we have at the beginning a clear understanding of the cases of adult and hereditary chorea is a question, but certainly the facts at hand are very suggestive.

A CASE OF ABDUCTOR PARALYSIS OF BOTH VOCAL CORDS.

Read before the Section of Laryngology and Otology, at the forty-third annual meeting of the American Medical Association, held at Detroit, Mich., June, 1892.

BY JONATHAN WRIGHT, M.D.,
OF BROOKLYN, N. Y.

The report of the following case is made more with a hope of helping to keep alive an interest in a puzzling subject than with a hope of adding anything entirely original to its literature.

K. T., maidservant, æt. 26, single, came for treatment to the throat class of the Out-Patient Department of Roosevelt hospital on Dec. 29, 1890.

Her family history was negative. She had a moderate alcoholic, but no specific history. Four years previously she began to have almost constant headache. She had pain in the bones and felt weak. She had shortness of breath and palpitation of the heart on exertion. She had some swelling of the feet and hands. At that time she was treated in the hospital under the diagnosis of simple anæmia. She had had similar symptoms since then but had kept at work. The previous winter she had considerable cough and expectoration. She had been run down since summer, and unable to work. For two months she had had cough and expectoration of phlegm. Five weeks before she had fallen in a faint and was totally unconscious for several minutes. A few days later her neck began to feel stiff and numb at the back. She had also numbness and dull pains in the right arm, side and leg, and she limped a little. Three weeks before admission she began to have some choking on swallowing. She felt as though there was something in her throat or behind it. There was no pain, but a deep seated numbness. A week later she began to make considerable noise at night in breathing. She had little or none of this when awake. She suffered some dyspnoea only on exertion. The feet had been a little swollen. She complained of no eyesymptoms. She had a little headache. The numbness had grown less. She had some cough. Her appetite was poor. Her bowels were regular and her menstruation was normal. She was sent to the wards of the hospital. Her condition on admission, as noted in the history books of the clinic and the hospital, was as follows:

"She is fairly well nourished; her face is rather sallow and a little pale. When awake her breathing is audible at the bedside, but not loud. It is somewhat stridulous in character. She had some difficulty in talking at times. She seems to phonate, at those times, on inspiration rather than on expiration. This is especially so after a laryngoscopic examination. P. 74, R. 22, T. 99.

"A physical examination of chest show signs of a general bronchitis, especially on the left side. There is some curvature of the spine, with the convexity to the left in the upper dorsal and lower cervical region. There are no points of tenderness. She said she had been so from a child.

"Urine, acid, 1013, albumen faint trace; a few pus cells; no sugar.

"Laryngoscopic examination. The left vocal cord is immovable in the median line. The right vocal cord moves in abduction only throughout about one-third of its normal arc, so that in greatest abduction there seems to be only about one-eighth of an inch between the two vocal processes of the arytenoids. Both cords seem tense (but this is a condition I am never absolutely sure of, the degree of tension varies so much normally in different cases)."

She had noisy breathing at night more marked on inspir-

*Dr. W. T. Montgomery and Dr. Casey A. Wood examined the eyes and made report.