

is a case on record of a man who was sentenced to be bled to death. He was blindfolded, the sham operation was performed, and water allowed to run down his arm in order to convey the impression of blood. Thinking he was about to die, he did actually die. Imagination had the same effect as the reality. But it is impossible to say how much fear had to do with it; probably a good deal, as in the instance of the man reprieved after his head had been laid on the block and the fatal axe was about to fall. The reprieve came too late. The anticipation of death had arrested the action of the heart."

John Hunter had an attack of gout when he was forty-one years old, and during the last fifteen years of his life he suffered and finally died from angina pectoris. He tells us that in his later life he became subject to violent disturbances of his heart when he was annoyed or anxious about any event. He says ("Works of John Hunter," edited by Mr. Palmer, 1838, vol. ii. p. 336), "At my country-box I have bees, which I am very fond of, and I was once anxious about their swarming, lest it should happen before I set off for town; this brought it on [an attack]. The cats tease me very much by destroying my tame pheasants, partridges, etc., and rooting up my plants. I saw a large cat sitting at the root of a tree, and was going into the house for a gun, when I became anxious lest she should get away before my return; this likewise brought on a spasm; other states, when my mind is much more affected, will not bring it on." Anger and anxiety had a very pernicious influence on his heart, and, knowing this, he said, "My life is at the mercy of any scoundrel who chooses to put me into a passion." And, indeed, his apprehensions were realized, for in a dispute concerning the admission of certain students to St. George's Hospital he was flatly contradicted by one of the governors. This so enraged him that he "immediately ceased speaking, retired from the table, and, struggling to suppress the tumult of his passion, hurried into the adjoining room, which he had scarcely reached when, with a deep groan, he fell lifeless into the arms of Dr. Robertson." An examination after death showed that his heart was small and strongly contracted. The coronary arteries had been changed into bony tubes, the aorta was dilated, and both the aortic and mitral valves were diseased.

#### SYPHILIS AS A CAUSE OF ANGINA PECTORIS.

The possibility that syphilis may produce angina pectoris is a subject of grave importance, and one to which sufficient attention has not as yet been directed. From our knowledge of the hydra-headed mani-

festations of this disease it would seem strange, however, if the heart escaped its contaminating influence. I have seen a number of cases of organic heart-disease and heart-pain the origin of which I attributed to a syphilitic source, but a true syphilitic angina pectoris I do not remember as having occurred in my experience. Other observers have been more fortunate in this respect, and during the last few years a number of such cases have been reported, and on account of their intrinsic value I shall abstract the histories of a few from an article by Professor G. A. Sacharjin, of Moscow, on "Die Lues des Herzens von der klinische Seite betrachtet" (*Deutsches Archiv für klinische Medizin*, 1890, Bd. xlvi. S. 388).

Male, aged forty, syphilitic, with frequent attacks of angina pectoris, which were followed by pulmonary oedema, and for which digitalis and other agents were employed to no purpose. Under an energetic course of sodium iodide treatment the attacks disappeared entirely, but returned later, although in so light a form that they were readily set aside by digitalis.

Male, aged fifty-five, infected with syphilis, although at an earlier period he passed through an attack of acute articular rheumatism, and was also addicted to the excessive use of alcohol. For more than a year he had suffered from severe attacks of angina pectoris, which were but indifferently relieved with nitro-glycerin. There was evidence of arterio sclerosis and aortic insufficiency, but aside from the stenocardiac paroxysms the patient was apparently well. Sodium iodide internally and inunctions of gray mercury ointment cured the attacks of angina pectoris, but had no effect on the aortic regurgitation or the chronic arteritis.

Male, aged —, is analogous to the last case. The attacks of angina pectoris were likewise entirely relieved by the same treatment.

Male, aged thirty-seven, addicted to alcoholic abuse for a number of years, and two years before he came under observation he acquired syphilis, which was followed by the customary secondary symptoms. Eight months previously he was suddenly attacked one night with angina pectoris. Two months ago he had his second paroxysm. Under the influence of sodium iodide and inunctions of mercury he was fully restored.

The author ascribes these attacks to the noxious action of syphilis on the heart and nervous system. What special portion of the nervous system he believes to have been affected is not intimated. May the vagi have been involved? That syphilis affects these nerves is attested by Vierordt, who relates a case (*Archiv f. Psychiatrie*, etc., Bd.

xiv. S. 678) in which syphilitic infection produced multiple neuritis, great acceleration of the heart's action, exhaustion, and death from phthisis, after which it was found that the pneumogastric and sciatic nerves were disintegrated. If, as we have reason for believing, the vagi are affected in such cases, then the manner in which syphilis brings about angina pectoris becomes perfectly obvious, and also indicates a possible similar effect in the case of alcohol and other poisons, which have a like deteriorating influence on the nervous system. Indeed, Dr. Bean (*Gazette des Hôpitaux*, 1862, xxxv. 326, 329) relates eight cases of angina pectoris which were induced by tobacco-smoke, and which disappeared when tobacco was avoided.

#### CAUSE OF THE PAIN IN ANGINA PECTORIS.

Pain in the region of the heart is one of the constant symptoms of angina pectoris, and it is therefore of interest to inquire into the origin of this phenomenon. In considering this part of the subject we must bear in mind that the heart and large blood-vessels possess both sensory and motor nerves, which are supplied by the vagi and sympathetic. The sensory impulses conducted by the former reach the brain through the medulla oblongata, while those which are conducted by the latter pass through the spinal cord.

What, then, is the cause of this pain? In an exceedingly interesting paper "On Cardiac Pain in Angina Pectoris," in the *Practitioner* for October, 1891, Dr. Brunton expresses the belief that the cardiac pain in this disease "is generally due to weakness of the heart in proportion to the resistance which it has to overcome." In common with Dr. Grainger Stewart, he compares the heart in this disease to the bladder when over-distended with urine. The latter contracts against the increased resistance due to over-distention, and produces pain of the most excruciating character; while in the case of the heart it is not always a condition of over-distention, but one which practically leads to the same results. Its muscular fibres are thrown into a state of spasm, are made to contract in their weakness against a resistance which is difficult to overpower, and the pressure which ensues gives rise to the excessive pain.

Such a view of the production of pain in this disease is certainly very reasonable, and seems to be in perfect accord with the practical experience which we have on this subject. It is a well-known fact that persons who are affected with heart-disease—especially with ventricular dilatation—are very susceptible to attacks of pain in the region of the heart when they overtax this organ by exercise, or by

ascending mountain elevations. In other words, the resistance which the heart is to overcome by pumping a larger amount of blood into the blood-vessels, in these instances, is greater than its weakened capacity permits, and hence there is still greater distention, or at least a tendency thereto, and pain in consequence.

As instances of the injurious effects of excessive exercise on the heart, and in producing angina pectoris, I take the liberty of quoting the following histories, which are reported by Dr. V. D. Harris in *St. Bartholomew's Hospital Reports*, vol. xv., 1879, p. 86:

"Male, aged thirty, came under my care at Victoria Park Hospital at the beginning of March, 1879. He was a fair-nourished although anæmic man. He came complaining of attacks of dyspnoea and pain in the chest, indicating angina pectoris. He had been perfectly well, according to his account, up to the preceding boxing-day, when suddenly, whilst singing a solo in some musical entertainment at a large East-End theatre, he felt something give way in his chest, and had a spasm of dyspnoea, from which he occasionally suffered since. He did not know he had heart-disease. There was no history of gout, rheumatism, or syphilis in the patient or in his family. Examination of the heart showed a double murmur, probably in the aortic valves. The history and the physical signs appeared to indicate rupture of the aortic valves.

"Male, aged thirty-one, came to Victoria Park Hospital in June, 1879. Was quite well five years before; a great athlete, and was in the habit of lifting very heavy weights. One day he was lifting two fifty-six-pound weights, and felt something give way in his left side. Three weeks after he had the first attack of angina, after which time they always came on after exertion of some kind. He said the fit comes on suddenly, with intense pain in the chest, running down the inside of the left arm, and in the latter place it was as though the flesh were being torn off the bone with pincers. When the attack comes on he feels as though he could not breathe any more, but has to pull at the clothes about his neck. Sometimes the attacks are simply dyspnoea, but in these there is not so much pain, neither do they appear to him to be serious; the great seizures, occurring more rarely, have come on even from the exertion of getting up in the morning, and they appear to him to endanger life. Examination shows a lesion in the aortic valves; and from the history of the sudden onset of the heart-disease after tremendous exertion, the youth of the patient, and the absence of gout, rheumatism, syphilis, and atheroma, I am induced to believe it to be a case of ruptured aortic valve."

One of the ablest clinical articles on heart-disease in relation to pain, which has come under my notice recently, is that by Dr. James J. Levick, on "Heart-Strain and Weak Hearts" (*Transactions of the American Climatological Association*, 1888). After discussing the various phases of "heartache," he classifies the immediate exciting causes of sudden death in persons who suffered from heart-weakness, so far as his experience goes, as follows: "First, walking on slippery, icy pavements on a cold day. (The patient had walked three or four street blocks to church, and died soon after taking his seat there.) Second, hurrying to railway station immediately after eating a hearty meal. Third, driving for some miles a hard-mouthed horse. Fourth, riding a hard-mouthed horse. (The patient had been helped by gentle horseback riding.) Fifth, sawing off the limb of a tree in his own park. The limb required some effort to reach; the position was a constrained one. This gentleman had had frequent attacks of this disease (heart-pain), with a feeble heart. A violent paroxysm followed this exertion, and he died before medical aid could be obtained. Sixth, hurrying from one steamboat to another, carrying at the same time a heavy bag. Seventh, assisting to carry a trunk from the railway van to the station. Eighth, shovelling coal into the furnace in the cellar. Ninth, the act of sexual intercourse. Three cases of this kind have recently come under my notice. In the first, a married man, aged sixty-five, had a violent paroxysm of cardiac pain immediately following this act. The patient lived for more than six months, was liable to severe paroxysms of dyspnoea,—which he never had before,—and died suddenly as he arose from his tea-table. A post-mortem examination showed the absence of valvular disease, but the existence of a firm clot in the ventricle, which was evidently ante-mortem, and which doubtless was formed coincidentally with the first severe paroxysm six months before. The second case was that of a gentleman, aged seventy-two, single, and remarkably hale and vigorous for his years, but who had at long intervals attacks of heart-pain. After a morning drive, his coachman driving, he visited (I use his own words) a lady and committed venery. He was almost immediately seized with an intense pain near the heart, but managed to walk home, a short distance, and I found him there with a cold skin, very feeble pulse, although he walked forward to receive me. He was immediately put under treatment, but death supervened rapidly. A somewhat similar case is reported of a judge of the Nottingham assizes, who was induced to go home with a young woman of the town, who testified before the coroner that immediately after having had intercourse with her he turned

on his side, gave a groan, and died. I have recently seen in consultation a fatal case of heart-failure in an elderly man, where the history pointed to this as the cause of death."

Dr. Alfred L. Loomis contributes a very interesting paper on "The Effects of High Altitude on Cardiac Diseases" to the *Transactions of the American Climatological Association* (1888), in which he details the histories of six cases of heart-disease which were affected very deleteriously by ascending mountain elevations of from one to four thousand feet. The heart-symptoms became aggravated in each case by the ascent, although in none were there any characteristics of angina pectoris developed. Five died, and one recovered after being brought nearer the sea-level.

In my own experience I met with at least one person with heart-disease in whom a severe attack of angina pectoris was provoked, and whose death was finally brought about, by ascending a mountain elevation about twelve hundred feet higher than his own home. It was that of a young man, aged twenty-two, who, when he was fourteen years old, suffered from chorea for nine months, directly after which he had an attack of articular rheumatism, which gave rise to some heart-trouble. At the age of twenty he had some difficulty with his heart, which was followed by profuse œdema of the lower extremities. This yielded to the judicious treatment of his physician, and from this time up to the time when he visited his aunt on the mountain elevation, and where he came under my care, he had been comparatively free from disease, and was able to do some work. Immediately after arriving at his aunt's house he was taken with a most excruciating pain in the cardiac region, and his sufferings here and along the course of the large arteries of the neck and arms were intense. Physical examination showed lesions of both the aortic and mitral valves. Œdema of the lower extremities supervened, but under the use of heart-tonics he rallied sufficiently to be able to return to his own home in the course of two weeks, where he died from copious hæmoptysis, probably due to rupture of a pulmonary blood-vessel, the day after he reached it. The autopsy showed an enormously hypertrophied and dilated heart, weighing a fraction over three pounds, with the aortic and mitral valves almost entirely obliterated. I think there is reason to believe that the attack was precipitated in this case by the relatively greater rarefaction of the mountain atmosphere, causing a disturbance in the already supersensitive equilibrium between the inside pressure of the blood and that on the outside of the body, and the resultant strain was too great for the previously weakened heart and arteries to endure.

All the evidence which has been adduced thus far shows that anything which disturbs the cardiac equilibrium, be this a rupture of the aortic valves, a sudden weakness induced by mental excitement, a simultaneous spasmodic contraction of its wall and of the arterial coats, or abrupt barometric transitions, will produce the same final results,—viz., an increased resistance within, and an inadequate power without, thus confirming Dr. Brunton's view of the generation of pain in this disease.

THE SIGNIFICANCE OF ATHEROMA AND OSSIFICATION OF THE CORONARY ARTERIES AND FATTY DEGENERATION OF THE HEART-MUSCLE.

There is a disposition on the part of many who have discussed this subject to attribute angina pectoris to atheroma and ossification of the coronary arteries; indeed, some have gone so far as to suggest that only those cases in which there is a morbid change of this kind, especially if this is associated with fatty degeneration of the heart, should be designated as genuine angina pectoris, and that this ear-mark should form the dividing-line between the true and the false forms of this disease. It is hard to account for the basis on which this argument rests, for atheroma and ossification, not only of the coronary arteries, but of the aorta, are quite common occurrences,—in fact, they may be regarded as different steps in the process of chronic inflammation of the intima,—yet angina pectoris is a comparatively rare disease, and is found independent of such degeneration. In the case of Leroux, quoted on page 34, the coronary arteries were open and normal; there was no fatty degeneration of the heart-muscle, but there existed an atheromatous condition of the aorta. Rothe contributes the following case (*Virchow u. Hirsch's Jahresbericht*, 1885, Bd. ii. S. 78), which shows no morbid change anywhere in the central portion of the circulatory apparatus beyond a dilatation of the arch of the aorta, and gives us reason for believing that extreme dilatation of the aorta may act as an indirect cause of death in this disease.

Male, aged fifty-three, while in his last attack of angina pectoris had an accelerated but regular pulse, unimpeded respiration, and was fully conscious. Chloroform, morphine, and amyl nitrite failed to relieve him. Death took place suddenly. On section, the thoracic and abdominal organs were found healthy, with the exception of dilatation of the aortic arch, the walls of which were hard and had lost their elasticity. The coronary arteries and heart wall were unaltered.

The same may be said of fatty degeneration of the heart as a cause

of angina pectoris. If we take for granted that this disease is fundamentally of a nervous character, it is readily seen that fatty degeneration may be a natural sequence of nerve-disorder. The experiments of Eichhorst which have been quoted on page 35 demonstrate that fatty degeneration of the heart-muscle follows section of the vagi in birds, dogs, and rabbits. We must remember, however, that fatty degeneration may also occur in muscle on account of inactivity, or on account of inanition sequential to nerve-section, and therefore these experiments are not wholly conclusive. But Wassliet's experiments, noted on page 34, establish the fact that among starving pigeons fatty degeneration occurred much earlier in those in which the vagi were cut than in those in which the vagi remained intact.

This gives us still more reason for believing that degenerations of the coronary arteries and of the heart-muscle are merely sequences and not causes of angina pectoris. Indeed, all the evidence, so far as I have been able to bring it together, goes to show that angina is essentially a disease which belongs to the spasmodic type, and that we might as well assert that bronchitis, bronchiectasis, emphysema, hypertrophy of the right ventricle, etc., are causes, and not secondary changes, of asthma, as to say that atheroma of the coronary arteries, fatty degeneration of the heart-muscle, dilatation and ossification of the aorta, etc., hold a causative relation to this disease.

INFLUENCE OF HEREDITY.

That heredity is a factor in the production of angina pectoris there can be no doubt in the mind of any one who has given thoughtful attention to this subject. It arises frequently among the members of neurotic families, who are also predisposed to asthma, migraine, epilepsy, hysteria, and pulmonary consumption. Dr. Ross states ("The Diseases of the Nervous System," vol. i. p. 577) that "hereditary predisposition can be traced in many cases of angina, and it is frequently found in members of families who manifest a tendency to other neurotic diseases, such as hysteria, insanity, and epilepsy. Attacks of angina may form a symptom of hysteria, precede or alternate with an attack of epilepsy, or constitute an intercurrent symptom of chronic mental disease." Dr. Anstie ("Neuralgia, and the Diseases that resemble it," p. 146) gives a *résumé* of three cases of angina pectoris in the families of which there existed epilepsy, asthma, softening of the brain, and other neurotic diseases.

## THERAPEUTICS OF ANGINA PECTORIS.

The therapeutics of this disease resolves itself into those measures which give instantaneous relief and those which prevent a recurrence; and only such means will be referred to here as have been found serviceable in my own experience. We have seen that the essential nature of angina pectoris is a spasmodic convulsion of the heart and blood-vessels, and in order to meet the first indication we are compelled to resort to the administration of agents which possess the power of relieving this spasm. To the scientific acumen of Dr. Brunton the profession is indebted for its possession in amyl nitrite of an agent which is capable of dilating the heart and blood-vessels, of lowering the blood-pressure, and of relieving angina pectoris. This discovery he made in 1866; amyl nitrite has been employed by inhalation in the great extremity of this disease ever since. It is certainly one of the most valuable additions that have been made to the therapeutics of internal diseases for a long time, yet in some cases it fails to act favorably, chiefly, I think, on account of its application to doubtful pathological conditions. Such is the impression which I have been able to gather from my somewhat limited experience with this drug, and I can offer no better guide to its proper administration than that of correctly diagnosing the disease. The rule which is generally laid down, that amyl nitrite is applicable in those cases only in which there is no flushing of the face, does not seem to apply universally; and when we take into consideration that a spasmodic condition may exist in one part of the circulation and dilatation in another part at the same time, it is obvious that the absence or the presence of flushing of the face may be a very spurious index as to the state of the circulation in the heart and large arteries, at least in some instances, although as a rule there is present paleness of the face in a paroxysm of angina pectoris.

This opinion also seems to be confirmed by the personal experience of Dr. Madden, of Torquay, as he relates it in *The Practitioner* (vol. ix. p. 331), and from which, on account of its intense practical interest and of its strong bearing on this subject, I take leave to make a copious quotation. After announcing the remarkable benefit which he obtained from the use of the amyl nitrite, he states that on one point he certainly increased his wisdom. From his reading he had formed the opinion that it was suitable only in those cases in which the face was pallid during the paroxysm, and since his was always flushed he had given up the idea of even trying it, "and paid the penalty of hasty conclusions in the shape of a large amount of acute suffering."

At the age of twenty-four he suffered from the effects of overwork, at which time there was also discovered some obscure disease of his heart and lungs. A short time before, his father died of angina pectoris,—the organic condition of the heart in his case being "atheromatous obstruction of the coronary arteries." Continuing to suffer from his heart- and lung-affection, he left Scotland the same year (1839) and went to Torquay, and in a few years he recovered sufficiently, resumed his practice in that place, and led an active life up to the time of writing. "On July 8, 1872, being fifty-seven years of age, feeling perfectly well, I was suddenly, and without the slightest warning, arrested by a severe attack of angina pectoris, the pain extending across the front of the chest, along the inside of the left arm, and across the chin." A medical friend was called in, and diagnosed a systolic mitral murmur, and counselled rest, which Dr. Madden did not take, but persevered in his ordinary work for ten days longer, when the severity and the frequency of the attacks compelled him to stop. "At first it seemed as if the quiet would prove curative, but in the afternoon of the fourth day, after taking to bed, I woke out of a doze with the severest and most prolonged spasm I had yet experienced. From this time the disease appeared to acquire increased violence. The attacks lasted, for the most part, for a quarter of an hour or twenty minutes, and recurred frequently at intervals of about three hours. Various remedies were tried, with little or no benefit. Morphia given hypodermically was the most useful, but it was impossible to employ it often enough without producing dangerous narcosis. At this time, when I was getting thoroughly worn out by the constantly-recurring pain, a friend, who happened to have in his possession a specimen of the amyl nitrite, suggested to one of my kind attendants the desirableness of giving it a trial, and furnished him with a small quantity. He consulted with his colleagues, and they unanimously advised me to make the experiment. I was willing enough to do so, and that night I was roused out of my first sleep by a sharp attack. I at once inhaled five drops, and the effect was truly wonderful. The spasm was, as it were, strangled at its birth. It certainly did not last two minutes, instead of the old weary twenty; and so it continued. The frequency of the paroxysms was not diminished for some time, but then they were mere bagatelles as compared with their predecessors, and consequently the drain upon the vital energies was greatly reduced. Under these improved circumstances, strength gradually returned; the attacks became less and less frequent, and finally ceased. At the time of writing these lines (October, 1872), I have not had an attack for five

weeks, and have resumed my ordinary duties, of course with care." In a private communication to Professor Gairdner ("Reynolds's System of Medicine," vol. iv. p. 590), in August, 1875, Dr. Madden states "that his confidence in the remedy continues unabated, but he has not required to use it for a considerable time."

After the introduction of amyl nitrite, professional attention was also directed to sodium nitrite and nitro-glycerin, which, on account of the resemblance of their action to that of amyl, were believed to be endowed with the same therapeutic properties, but this has not been borne out by practical experience. The hypodermic injection of morphine, from one-sixteenth to one-eighth of a grain, combined with one-twenty-fifth of a grain of strychnine, is useful in cutting short the attacks. So are also the inhalation of chloroform, and the application of hot poultices, mustard, and strong liniments over the anterior portion of the chest.

In the second place, what are we to do to prevent a recurrence of these attacks? First of all, our patient must avoid all exciting causes, such as fear, anger, or excitement of any kind, must do no heavy work, lift no heavy weights, and be guilty of nothing which will unduly raise the blood-pressure and thus throw an extra strain on the heart and large vessels. His food should be of easy digestion and at the same time highly nutritious, and for this reason he should live principally on milk, soups, meats of all kinds, and a fair proportion of vegetables, and drink plenty of water. In the next place, it is the physician's duty to endeavor to alleviate any organic disease of the heart that may exist, to neutralize any gouty or rheumatic diathesis that may be present, and to elevate the nutritional tone of the nervous system to the highest state of efficiency.

For the purpose of correcting any bad effects arising from organic deformity of the valves, it will be found that, as a rule, all our common cardiac stimulants—such as digitalis, caffeine, and strophanthus—are of very little value; in fact, sometimes they are positively detrimental. This may possibly be due to the fact that these agents influence the heart and the arteries too directly and too powerfully, and in this way create too great an immediate tension in the circulation; for strychnine, which I know from practical observation to be an indispensable drug in the treatment of this disease, also elevates the blood-pressure, but probably brings this about in a more gradual manner. In my experimental work I often noticed the pronounced difference with which digitalis and strychnine affected the frog's heart. Both enhanced its contractile power, but digitalis produced great irrita-

bility of the heart, and finally arrested it in systole, while strychnine did not increase its irritability, and instead of arresting the heart in systole it arrested it in diastole. This is a great difference between the action of the two agents which seemingly affect the same structure in a similar manner, and it may throw some light on the clinical experience which we have had with these agents.

Give strychnine, then, for this purpose, and in doses large enough to produce its effects. I rarely begin with less than one-thirty-second of a grain, and gradually increase to one-twentieth of a grain every four hours. The patient F., whose history is given on page 31, began with one-thirty-second of a grain of strychnine four times a day, but during the last two months it has been increased to one-twentieth of a grain four times, and he has been practically free from attacks since the increased dose has been taken. In a rebellious case I should recommend an additional daily dose of one-thirty-second of a grain of the same drug hypodermically. At the same time it may be given hypodermically once a day in one-thirty-second of a grain. Strychnine is one of our best permanent antispasmodic agents in the materia medica, not only in angina pectoris but in asthma, and it has also produced some very serviceable results in epilepsy. Phenacetin, antipyrin, antifebrin, quinine, arsenic, and atropine are also useful in elevating the nerve-tone of the arterial circulation. To counteract the rheumatic or gouty diathesis, guaiacum, salol, sodium salicylate, or colchicum must be given. The following combination is useful in many cases:

R Strychninæ sulph., gr. i;  
Atropinæ sulph., gr.  $\frac{1}{10}$ ;  
Acid. arsenic., gr.  $\frac{1}{2}$ ;  
Quininæ sulph., gr. xxxii;  
Phenacetini,  
Guaiaci res., or salol, āā gr. lxiv.—M.  
Ft. capsulæ nō. xxxii.  
Sig.—One capsule four times a day.

In addition to the above, the mineral acids must be given with each meal, and an occasional calomel purge must be employed.

The application of electricity is useful as a means of cure. Duchenne, Eulenberg, and Von Heubner speak very highly of it, and Bramwell, in his work on "Diseases of the Heart," says he has seen distinctly good results from the use of galvanism. He applies a current from about thirty elements, with the positive pole over the sternum and the negative pole over the lower cervical vertebræ. Personally, I have not had much experience with electricity here, but it is certainly as much indicated here as in any other neurotic disease.