chief blood-making tissues—spleen, lymphatic glands, marrow of the bones —causing defective elaboration of the blood, it seems proper to select some name that will indicate this fact, as *anæmatosis*.

4. The changes in the blood consist of great reduction in its mass, with extreme diminution in the proportion of red globules, without increase in the white corpuscles. There are probably also changes in the vital properties both of the red and white corpuscles.

5. The other lesions, chiefly fatty degeneration of the heart and other organs, passive effusions and hemorrhages, are secondary, and depend upon the blood changes.

6. The symptoms are explicable, in great part, by the state of the blood and the condition of the heart.

7. The disease, when once fully established, appears to be invariably fatal.

8. The remedies which afford most prospect of relief are cod-liver oil, arsenic, and phosphorus.

9. Transfusion is only capable of doing temporary good.

10. The operation is not free from grave danger, owing to the feebleness of the heart and the small amount of blood in the vessels; and, in order to be safely employed, the amount of blood injected must be very small (f℥iij), it must be introduced very slowly, and the operation must be repeated at suitable intervals. It adds to the safety of the operation to inject the blood into a small artery instead of a vein.

ART. II.—*Angina Pectoris*. By HAMILTON OSGOOD, M.D., of Philadelphia.

IN looking through the literature of this agonizing disorder, one is struck by the great, indeed confusing, variety of opinions which have been put forth concerning its cause and nature. Its terminology is not less embarrassing. The disease was first described by Heberden in 1768. He gave it the name of "angina pectoris." Since his day various authors have affixed to this lesion names which express, to a greater or less degree, the opinion of each writer as to its etiology and pathology.

Thus, we have "sternalgia" (Good); "syncope anginosa" (Parry); "stenocardia" (Brera); "strernodynia syncopalis" (Sluis); "neuralgia cardiaca and hyperæsthesia plexus cardiaci" (Romberg); "hypercinesis with hyperæsthesia" (Bamberger); Bouillaud terms the affection "neuralgia of the phrenic nerve," and finally we find the common German term

"Brustbraüne," literally "breast quinsy," a poor substitute for better names.

These terms are descriptive and for the most part unsatisfactory. The disease has no common name, although, in English and French works, "angina pectoris," which is too diffuse, is the term commonly used. The Germans generally prefer "stenocardia," which, while it localizes the disease and is undonbtedly a better descriptive title than angina pectoris, does not cover the probable nature of the lesion so fully as the "cardiac neuralgia" of Romberg; which term upon the whole appears to me to be the most intelligent and satisfactory. But, if one were to use this term, he would at once be reqnested to explain himself, which he would do by substituting the term angina pectoris; which name, while it answers every purpose so far as the sensations of the patient are concerned, neither strictly locates, nor in any way snggests the nature of the complaint. Even the term cardiac neuralgia (which may be understood as including a derangement of the cardiac plexus) becomes poverty stricken when we ask how it happens that during an attack of neuralgia of the heart, the fourth finger and ulnar side of the ring finger of the left hand become numb, cold, and painful. Why not the whole hand, when the whole left arm aches? And, if these symptoms indicate that only the ulnar nerve is engaged, who can explain why it is that other nerves coming down the arm from the brachial plexus are not also affected? That nerves of another sort are involved, is shown by a symptom which I do not find mentioned in any article on angina pectoris to which I have had access, but which came to my notice in the first case of this lesion I ever saw in my own practice, and which I had abundant opportunity to study during the six months of its intermitting attacks. The symptom to which I refer is a decided difference between the radial pulses; a difference which makes them so entirely dissimilar, that, while the right pulse is comparatively full and strong, the left is almost or quite imperceptible. This symptom suggests that, in the affection under consideration, the vaso-motor nerves of the left arm are also subjected to the same influence which affects the ulnar nerve. How does this curious selective influence do its work? If by the route of the spinal cord, as some suggest, why is the left arm alone affected? Why not the right? It is but very rarely that this arm is involved in the attack. And, even in the left arm; since the median nerve receives half its fibres from the inner cord of the brachial plexus, whence also arises the ulnar, why is the latter nerve subjected to effects which, save in rare cases, spare the median? And if the pain sometimes involves the median, why not always? By what process can pain, extending from the cardiac to the brachial plexus, travel over this plexus, run down the inner cord, and be so accurately, and almost invariably, switched off upon the ulnar nerve alone? The fact, too, that the vaso-motor nerves of the left arm are also engaged, increases the general cloudi-

ness of the subject. "As in many other neuralgic affections, so in this, too, is found much that is inexplainable." (Von Dusch.[1])

The singular partiality for certain widely deviating paths, which is[2] exhibited by the pain of angina pectoris, is well shown by Von Dusch:—

"The neuralgia which arises in the cardiac plexus, radiates over the track of the vagus and sympathetic to the cervical and brachial plexuses, with which these nerves are so intimately united. The pain also involves the major and minor occipital nerves (which arise from the first and from the posterior branch of the second cervical nerves respectively), as well as the supra-clavicular nerves which supply the integument of the upper chest as low down as the fourth rib. The anterior thoracic nerves mentioned by Lussana, and which arise from the brachial plexus, are purely motor; and yet, it is over the internal cutaneous nerves on the inner side of the upper arm, and over the ulnar on the posterior and outer side of the forearm, down to the integument of the fourth and fifth fingers, and in rare cases over the terminal branches of the median in the remaining fingers, that the pain radiates; and these nerves arise from the same plexus as the anterior thoracic. Through the cervical plexus the phrenic nerve is also brought into sympathy, and hence arises singultus; while the œsophageal symptoms (suffocation, difficulty in swallowing, and globus), and the gastric symptom (vomiting), indicate the participation of the pneumogastric filaments."

It is these eccentric routes of the pain which mystify Von Dusch and many another physiologist.

Romberg[3] theorizes as follows:—

"So far as the rootlets of the sensitive fibres of the cardiac tissue are concerned, the seat of sympathetic pain is of physiological interest. Müller has already called attention to the fact that the prevertebral cord of the sympathetic is only an *apparent* connective between the upper cervical and the coccygeal ganglia, and that the root-fibres from the spinal cord, after they have entered the prevertebral cord, run only for a certain distance in the same, and then, leaving the cord, lose themselves in the peripheries of the organs, etc. Consequently, sympathetic fibres which emerge from the prevertebral cord are neighbours of cerebro-spinal nerves which supply the upper portion of the trunk. Hence, when pain radiates from organs either of the abdomen or thorax, the sensation necessarily appears in the upper extremities, in the neck, and still higher, in the head. (Henle.) The radiation of cardiac neuralgia, then, allows us to suppose the anatomical contiguity, in the spinal cord, of the sensitive cardiac fibres of the sympathetic and of the sensitive elements of the cervical nerves. That a similar group of symptoms also makes its appearance in primary affections of the cervical district of the spinal cord, cannot be surprising. This was quite overlooked by observers, who, following in the footsteps of Parry and Jenner, suppose that cardiac neuralgia, or, as Heberden first entitled it, angina pectoris, has merely a peripheral origin, and connect the symptoms with a definite organic change, viz., incrustation of the coronary arteries. Others claim that this neuralgia may find its cause in a great variety of morbid conditions of the heart; but with what misjudgment is proven by more recent observers. Whoever doubts this may find enlightenment in Laennec's experience."

This suggests the widely divergent opinions as to the etiology and pathology of angina pectoris. But, while there exists this opposition of opinion as to cause, there seems to be among physicians a general belief that angina pectoris *invariably* includes organic disease of the heart. I

[1] Lehrbuch der Herzkrankheiten.

[2] Op. cit. pp. 336.

[3] Nervekrankheiten, s. 145.

think this belief an error. It may be that those who thus believe, class that form of angina pectoris, in which the heart is apparently sound, under the head of "pseudo" or "simulating" angina. For example, Flint, in the earlier editions of his work on "Diseases of the Heart," described a form of angina pectoris which he termed "pseudo"; but in later editions he omits the term "pseudo" and speaks of angina pectoris *with* and *without* disease of the heart. This modification, so far as it goes, I am convinced is correct. There can be no such affection as *pseudo* angina pectoris, even though the attack may have an hysterical basis, for, in the hysterical cases in which I have met with angina pectoris, the symptoms, varying of course in their severity, invariably included the prominent landmarks of the disease. They were too real in their effects to admit of being considered simulative; and since hysteria, as Romberg acknowledges, can run into real neuroses, angina pectoris may be considered one of them. If a seizure cause, for example, a very marked contraction of the radial artery, or severe pain in a locality so far removed from the heart as is the hand, these symptoms are significant of the real nature of the attack. But if in such case the heart were found to be normal, believers in the invariable connection of angina pectoris with organic disease of the heart would therein find their chief, indeed only, reason for considering the attack of pseudo nature and the cause hysteria. If there were no tangible effects this view might be admitted, but when, setting pain aside, symptoms so sharply marked as numbness, local loss of temperature, unsymmetrical contraction of arteries are observed, there certainly is neither voluntary nor involuntary simulation. But I feel impressed that the exact truth would carry us even further than this. When the attack seizes a person whose heart is sound, I believe the difficulty to be a functional derangement of some nerve or nerves which more or less directly influence the heart, and are capable from their connections of propagating pain elsewhere. As Von Dusch remarks, there must be an "especial cause," but what this cause is, and why the pain seeks such eccentric routes, seems unanswerable. On the other hand, if the spasm complicate some lesion of the heart, I am unable to see how it can be other than a *coincidence*, not dependent upon the cardiac affection. Otherwise, why is it that out of the multitude of cases of disease of the heart and vessels, there are so few which include angina pectoris?

It should be remarked that the pain in the left arm does not invariably extend to the fingers, for I have seen one case in which the numbness and neuralgia stopped at the elbow. But, even in this case, there was the contracted radial at the wrist, indicating that the vaso-motor nerves of the forearm were under the influence of the attack. It may be supposed that other arteries of this arm also become contracted, this condition contributing, perhaps, to the pain which, according to the statements of patients whose forearms were suffering, was not confined to the track of

the ulnar, but involved the whole member. Yet in these cases only the third and fourth fingers of the left hand were affected, thus suggesting that out of the motor nerves of the arm the pain had chosen only the ulnar.

Another point is, that, although the third and fourth fingers of the left hand have a lower temperature than other fingers of the same hand, the entire member has less warmth than the right. The same remark may be applied to the temperature of the whole left arm as compared with that of the right.

To return now to the different opinions of writers on angina pectoris as to the cause of the attacks. The passage from Laennec to which Romberg, as above mentioned, refers, is the following :—[1]

"The majority of physicians in England, Germany, and, above all, in Italy, are not the less convinced that angina pectoris is always connected with some organic disease of the heart, that the attacks are very serious, and that the greater number of patients who are attacked die suddenly. These ideas are far from being exact. Angina pectoris of moderate severity is an extremely common affection, and very often exists in individuals who have no organic disease whatever, either of the heart or of the great vessels. I have seen many individuals who have experienced only a few attacks of great severity, but of short duration, and who subsequently became entirely freed from them." And further: "On the contrary, it is true that angina pectoris is frequently coincident with organic affections of the heart, but even in such cases nothing proves that the angina is dependent upon the cardiac affection, for it can arise when the heart is normal. I have made *post-mortem* examinations of the bodies of subjects who were simultaneously subjected to hypertrophy and dilatation of the heart and angina pectoris. In not one instance did I find ossification of the coronary arteries. One of these patients died suddenly during a violent attack of angina pectoris. Any one will admit that the union of so intense a nervous affection with enormous hypertrophy of the heart (which existed in this case) might sometimes produce this result."

Laennec considered the disease a neuralgia, and says: "I believe that its seat may vary, or rather, that a neuralgia which originates in different nerves may give rise to the same symptoms." Hence he argues that the pneumogastric, or the cardiac portion of the sympathetic, may either of them be the point of origin, and that the brachial plexus becomes sympathetically implicated.

Heberden[2] thought that angina pectoris originates in cardiac cramp or spasm, and mentions the autopsy of a man who died suddenly of this disease. "A very skilful anatomist," says Heberden, "could discover no fault in the heart, in the valves, in the arteries or neighbouring veins, excepting some rudiments of ossification in the aorta."

Friedreich[3] divides the disease into *dynamic* and *organic*, and says: "The dynamic (idiopathic, functional, nervous) form arises without complication with any organic affection whatsoever. The organic form is united with organic change in the heart or large vessels." He believes

[1] Traité de l'Auscultation, 3d edition, pp. 350, 351.

[2] Heberden's Commentaries, pp. 296. 297.

[3] Herzkrankheiten, Virchow's Handbuch, v. Band.

angina pectoris to be a neuralgia of the cardiac plexus, that dynamic angina is a hyperæsthesia and primary neuralgia of this plexus, and that "organic angina pectoris depends upon calcification, obliteration, and degeneration of the coronary arteries." Bamberger[1] writes: "That the affection is neuralgic can be positively asserted. Whether the real point of origin is to be sought in the sympathetic, the spinal cord, or in the cardiac ganglia cannot be determined. The greatest probability, however, declares in favour of the latter, or, in a larger sense, in favour of the cardiac plexus." He believes the affection to be most frequently dependent upon some material disturbance of the heart, but adds:—

"One must protect himself from the one-sided view, that one particular anatomical condition of the heart is the cause of stenocardia. Hence the old view which makes calcification of the coronary arteries the cause of the lesion is certainly as incorrect as the more modern belief which exclusively accuses fatty metamorphosis and flaccidity of the heart." And finally: "One is inclined to assert that stenocardia is essentially grounded in a clonic spasm of the heart with hyperæsthesia, which condition, however, extremely seldom (perhaps never) depends upon a purely nervous disturbance, but as a rule is the result of a diseased condition of the heart through which an abnormal irritation acts upon the cardiac ganglia or cardiac plexus."

Canstatt,[2] deriving his conclusions from the autopsies of forty-five fatal cases of angina pectoris (gathered by Forbes), and of which only six were found free from affections of the heart or vessels, believes that "fatal cases of purely functional angina pectoris are rare;" and further says that—

"The common revelations of autopsies in fatal cases consist of striking changes in the cardiac structure or large vessels, especially the aorta." Mentioning a dozen varieties of these changes, he concludes thus: "None of these changes, which are often absent, and not less often present, without causing angina pectoris, contain the essential cause of the disease, although it must be admitted that they are reckoned as factors of the lesion."

According to Stokes—[3]

"It is greatly to be doubted that angina pectoris has ever occurred in a patient perfectly free from organic disease of the heart or aorta. In the present state of knowledge we must follow Dr. Latham in considering angina pectoris rather a special set of symptoms than a disease having a fixed anatomical character."

Latham believes the affection to be invariably connected with disease of the heart, and, as to its true character, says:—

"Now we are in search of something in the heart which, as a concomitant of pain, may be disabling to its natural functions and capable, according to degree, of hindering or abolishing them altogether. This we find in spasm. In its spasm of smaller degree the heart fails to close freely upon the blood and to impel it freely into the arteries. In its spasm of greater degree it fails to project it altogether. Hence we discern an adequate explanation of the chief phenomena of angina pectoris: it is spasm of the heart."

Stokes confesses that "it is difficult to understand how such a general or local spasm, bearing in mind that the heart is a hollow muscle, could

[1] Krankheiten des Herzens. [2] Pathologie u. Therapie, iii. Band, s. 88.
[3] Diseases of the Heart and Aorta, art. Angina Pectoris.

occur as would only impede and not destroy the heart's function; for a complete spasmodic closure of any one cavity ought to cause death by breaking the continuity of the circulation." In regard to this point Flint agrees with Stokes. According to Parry, whose views are also opposed to the doctrine of spasm, "the disease is an example of syncope, preceded by a notable anxiety or pain in the region of the heart, the result of organic lesion, which acts in diminishing the energy of the heart." And he holds that "the symptoms arise from retardation and accumulation of blood in the cavities of the organ." Hence Parry's name for the disease: "Syncope Anginosa."

Köhler[1] considers angina pectoris undoubtedly a neuralgia of the cardiac plexus, "complicated only in severe cases with cardiac spasm."

C. J. B. Williams[2] affirms that the disease "is commonly associated with organic lesions, especially, but not exclusively, those affecting the aorta and its valves; but," he adds, "such affections frequently exist without it. So, on the other hand, anginal or neuralgic pains sometimes occur in persons who have no organic disease; thus resembling other forms of nervous disorder, which, although occasionally excited by the irritation of bony deposits, or other permanent lesions, *may arise from an excessive sensibility developed by more transient causes.*"

Tanner[3] says: "It may be remarked that our improved means of observation have rendered it almost certain that this disease is always associated with some important organic affection."

Niemeyer[4] asserts that "angina pectoris is almost exclusively met with in individuals who are suffering from organic disease of the heart." Mentioning a variety of cardiac affections which were found in fatal cases of angina, he adds:—

"Nevertheless, angina pectoris is not to be considered as a special symptom of these anatomical changes, for none of them is constantly connected with it. It arises under the same form in the most opposed structural changes of the heart, has its paroxysms and free intervals. So that, in fact, we are obliged to consider it a nervous affection of the heart, to which by preference only organic diseases of the organ are disposed. In solitary cases, angina pectoris arises without organic cardiac lesion, especially in aged, fleshy subjects; in men oftener than in women."

Trousseau[5] wrote as follows:—

"All the incontestable histories of individuals who during life presented all the characteristic symptoms of angina pectoris, and yet, whose autopsies did not reveal the slightest anatomical change to which these symptoms could be attached, demonstrate that angina pectoris is not necessarily connected with organic diseases. From the absence of appreciable organic change and from the variability of the phenomena, we must conclude that the disease is a neurosis or more precisely a neuralgia. It ordinarily occupies the cardiac nerves which emanate from the pneumogastric, whence it radiates to the cervical and

[1] Therapie, i. Band, s. 484.
[2] Diseases of the Chest, pp. 232, 233.
[3] Practice of Med., p. 394.
[4] Pathologie u. Therapie, 8e Auflage, i. Band, s. 411.
[5] Clinique Medical, t. ii., pp. 529, etc.

brachial plexuses. With the majority of clinicians I admit that this singular neurosis may be symptomatic, but I admit it only in the sense that in such case there is a simple coincidence, and that, whatever they may be, organic affections merely afford an opportunity for the development of the nervous lesion which attaches itself to them."

Gerhard[1] believed that "angina pectoris depends upon a functional disease of the heart closely allied to gout, or upon various organic lesions, especially ossification of the valves."

Oppolzer[2] is undecided as to the etiology of the disease, but thinks that a certain connection exists between angina pectoris and heart disease. He refers to cases of so-called "reflex stenocardia," which were observed in coexistence with diseases of the uterus, kidneys, and liver, and mentions instances which were caused by catarrh of the stomach.

Da Costa[3] thinks it "more than highly probable that these so-called spasms of the heart are always linked to some structural change." Mentioning the variety of structural changes which may be coincident with angina pectoris, he concludes that "it is not impossible that combined with all these states is fatty degeneration, which thus would be at the root of the angina;" and that, "whether this view be correct or not, it is undoubted that fatty degeneration is more frequently conjoined with angina than in any other disease."

Flint's[4] opinion is that "fatty degeneration is sometimes observed, but by no means as a rule." He says further:—

"I will simply remark that the hypothesis which attributes the occurrence of angina pectoris to simple weakness of the heart, has no better foundation than that which attributes it to spasm. Angina pectoris is not always accompanied by evidence of lesions of the heart or aorta. There is reason to believe that it may be a purely nervous affection. Heretofore I have considered true angina as invariably incident to cardiac or aortic disease, considering all cases in which more or less of the symptomatic phenomena are manifested, without the signs of disease of the heart or aorta, as cases of simulated or pseudo-angina. I am satisfied that this doctrine is incorrect, inasmuch as the distinctive features of the affection may be as well marked without as with these signs."

He thinks the paroxysms involve, as a point of departure, neuralgia of the cardiac nerves, and that the disturbed action of the heart in certain cases is due to an affection of the pneumogastrics, but does not find this cardiac disturbance a constant element of the paroxysms. Referring to the Heberden theory of spasm, Flint says: "A spasmodic condition of the heart sufficient to occasion such prolonged as well as intense pain, would be incompatible with life."

Von Dusch[5] expresses his belief that the disease is a neuralgia of the cardiac plexus. He adopts two forms of cases, the "idiopathic or reflexive," which is not attended by changes in the cardiac structure, and the

[1] Diseases of the Chest, 4th ed., p. 427.
[2] Pathologie u. Therapie, s. 267.
[3] Medical Diagnosis.
[4] Diseases of the Heart, art. Ang. Pect.
[5] Op. cit.

"symptomatic," which he considers the most frequent, and which involves a complication of some disease of the heart.

Finally, he says: "If we follow the analogy of other paroxysms of pain, in the district of the sympathetic, *e. g.*, cases of intestinal colic, cardialgia, nephritic, and gall-stone colic, we must suppose the existence of a spastic condition of the heart, which causes the painful sensations," and he points to the small pulse as significant of this spasmodic condition of the organ.

Wolff[1] believes that the cause of angina pectoris lies neither in fatty heart nor atrophy of the organ through calcification of the coronary arteries, but finds the reason simply in an imperfect innervation of the heart.

Lancereaux finds the cause in an inflammation (neuritis) of the cardiac plexus, and cites *post-mortem* proof.

Eulenburg[2] and Guttman conclude from an analysis of cases, and from the experiments of Bezold, that abnormal action of the heart in angina pectoris is due to the influence of the sympathetic on the ganglia of the heart, and, as all the sympathetic fibres of the heart meet in the cardiac plexus, that this plexus is the medium through which the abnormal action is produced.

J. Lockhart Clarke (quoted by Knight, loc. cit.) thinks Laennec's view in regard to the proximate cause of the disease has been materially supported by recent inquiries.

Loupias[3] believes in an "essential and symptomatic" form of angina pectoris.

Moinet[4] strictly divides the disease into functional and organic. The first form he confines to young persons; finds it independent of heart disease, thinks it a neurosis due to lack of cardiac innervation, and curable. The organic form, he thinks, is due to a serious affection of the heart, which paralyzes the organ. The paroxysms of the organic form he cannot explain.

Nothnagel[5] thinks the "so-called angina pectoris" is in many cases due to a spasm of the arteries, and terms it "angina pectoris vasomotoria," deeming the cardiac spasm secondary to a wide-spread arterial spasm.

Bouillaud[6] thought angina pectoris a neuralgia of the phrenic nerve.

Handfield Jones thinks the disease a neuralgia.

Bellingham called it "dyspnœa of the heart."

Dickson[7] thought it a functional affection of the heart itself, being depen-

[1] Schmidt's Jahrbücher, 1866, ii. Band.

[2] Knight's Article on Ang. Pect. in Boston Med. and Surg. Journal for May 21, 1874.

[3] Schmidt's Jahrbücher, 1866, ii. Band.

[4] Schmidt's Jahrbücher, 1870, ii. Band.

[5] Schmidt's Jahrbücher, 1867, ii. Band.

[6] Maladies du Cœur.

[7] Elements of Medicine, p. 366.

dent neither upon a gouty diathesis nor upon disordered state of the stomach (as Butler, McQueen, and Chapman have taught), nor necessarily connected with organic disease of the heart; but admits the possibility of cardiac spasm.

A comparison of these conflicting views, which it were useless to classify, will indicate the confusion which exists among medical writers in relation to angina pectoris. The subject is still enveloped in a haze of uncertainty. Opinions are kaleidoscopic. Every writer has his own view. A search in books for satisfactory information as to the true nature of this neuralgia reminds one of the vexed subject of tubercle. But Laennec's opinions, amalgamated with Romberg's title for the disease, seem the most reliable. It will be noticed that the doctrine of the invariable connection of angina pectoris with heart disease meets with strong opposition, —opposition which is yearly gaining new disciples. That the paroxysms do not depend upon cardiac lesions has been proved to my satisfaction in four of the five cases which I have treated. In the fifth case I believe the attack to have been a coincidence and not a result of the condition of the heart.

CASE I. The first of these cases was that of a young lady who had suffered from hysterical neuralgia for several years. Two years ago, upon one occasion, I greeted her by taking her left hand, and was at once struck by the very great coldness of the little finger. Testing the sensibility of this finger, I found that it lacked ordinary sensation, likewise the ulnar side of the ring finger. Upon questioning the patient, I learned that the entire left arm was numb and somewhat painful, and that a heavy feeling existed about the heart. A few days subsequently the first attack of angina appeared.

Nothnagel might claim these prodromic symptoms as testimony to his theory of primary spasm of the arteries.

The first attack in this case was attended by instructive features. When I reached the patient, I found her unconscious, in a recumbent posture; face somewhat flushed; pulse 140; respiration laboured; left hand relatively cold. Arousing her with questions, she replied in a bewildered manner, invariably using the last word of my question, saying nothing more, and even that much with difficulty. Directing her attendants to remove her clothing, I saw her again after she was placed in bed. The case puzzled me. I gave her brandy. To my surprise the pulse fell within a few minutes from 140 to 90. This furnished the first leading clue as to the nature of the difficulty. Somewhat revived, the patient alternately grasped her left arm, and clasped her hand spasmodically over the heart. I then decided the case to be angina pectoris. Still more revived, patient, using signs, called for pencil and paper, and wrote: "I cannot speak, but feel better." A few minutes later when I questioned her, she pointed to her larynx and shook her head, still unable to speak (thus indicating the implication of the laryngeal nerves), but expression and gesture told me of the pain she was suffering. At intervals I administered tinct. op. in large doses, brandy and laudanum, ammonia and brandy, meanwhile applying mustard over the heart and bottles of hot water to hands and feet. The first attack passed by, but within an hour

came a second. This was followed during the next thirty-six hours by several additional attacks, the patient gradually weakening. Under increased doses of opium and brandy and of brandy and tinct. of valerian she however revived, and during the next two months attacks were mild in character. Then came a paroxysm to which I thought the patient would succumb. The same treatment, supplemented by galvanism (Duchenne), and applications of ice over the heart (Romberg) finally conquered. During the intervals tinct. of valerian was used with very comforting effect, apparently acting as a prophylactic. A new attack appeared, and for the first time I resorted to the nitrite of amyl. Beginning with two drops, I was obliged to increase the dose (given by inhalation) until I used nearly a teaspoonful. The effect was happy. The attack was cut short. In the subsequent three or four attacks the pain was less severe, and teaspoonful doses of nitrite of amyl invariably conquered, and eventually relieved the patient of further annoyance. During the past eighteen months no paroxysms have appeared. These attacks occurred at intervals which comprised a period of six months, during which time the left arm was not only constantly numb, but lost in strength as well as in size as compared with the right.

It was this case which revealed to me the marked difference, in the course of an attack, between the radial pulses. Indeed, I finally found that the contracted left radial preceded an attack, and that the patient simultaneously felt the sensation of an oncoming paroxysm. Guided by this symptom, I frequently administered remedies before any other symptom appeared, and found it invariably the case that so soon as the left pulse resumed its normal size, painful sensation ceased. During a paroxysm the right pulse assumed a size smaller than was normal, but while it was always perceptible, the left frequently disappeared. This condition of things led me to suspect vaso-motor spasm in the left arm. It is possible that the theory of Von Dusch, which attributes the small radial pulse of a paroxysm to spasmodic contractions of the heart, which allow only an incomplete diastole, and hence an imperfect supply of blood to the arteries, is correct. But this occurs during an attack, and does not account for the narrow left radial preceding a paroxysm. Nor does it explain the notable difference in size between the right and left pulses in height of an attack.

In the case just detailed, the routes taken by the pain were sharply outlined. They ran to the left ear, to the left side of the lower jaw, to the œsophagus (causing globus), to the larynx (producing aphonia); involved the phrenic (as shown by singultus); also the brachial plexus and the ulnar to the fingers. The case gave me opportunity to test the whole repertoire of remedies for angina pectoris, which are praised by various writers. Stimulants, opium, and counter-irritants were the most serviceable, yet did their work indolently, and with each new attack gave me a wearying round of action.

Romberg's ice treatment and galvanism, recommended by Duchenne and others, merely increased the discomfort of my patient. My experience

has taught me that nitrite of amyl is the best known remedy for angina pectoris. There are cases on record (Fagge's, etc.) in which it failed. At my hands it has never done so, and although in my fifth case it did not preserve life, it was of great use, as will be seen.

I find that many physicians lack the courage to use so powerful a remedy. This fear, I believe, to be ungrounded. I have never yet seen the slightest ill results from its use, notwithstanding the enormous doses required in my first case. If my patient be a novice to the remedy, I begin with one or two drops, and, watching the effect, increase the quantity until it gives relief. With the exception of the one case in which I used the liquid in nearly drachm doses, I have never been obliged to exceed eight drops at an inhalation. My habit during the administration is to keep my finger on the pulse, and watch the *nose* of the patient. The pulse commonly runs up to 120, 130, 140, and at the same time the nose and afterwards the face becomes deeply flushed. So soon as the nasal flush appears, I at once remove the handkerchief, even though the pulse be not greatly accelerated. Following this course, I have seen nothing but relieving effects. Headache of considerable intensity sometimes temporarily sets in, but should not be considered (unless unbearable) until the heart becomes relieved. Under the effects of the remedy the patient occasionally suffers a general but transient discomfort. Those who wish detailed information concerning the physiological action of the nitrite of amyl, may find it in the valuable and interesting paper by Prof. H. C. Wood, Jr., in the number of this Journal, for July, 1871.

CASE II. This case is that of a young lady who suffers mild attacks of angina pectoris, which cause pain and a heavy feeling about the heart, pain which shoots up the left neck, and pain in the left arm, involving the third and fourth fingers, and producing a contracted left radial.

In this case I have used no other remedy than the nitrite of amyl, to the effect of which the patient is exceedingly sensitive. I am unable to use more than three-drop doses, and generally during the second inhalation, with a spasmodic shudder, the patient flings herself away from the handkerchief. By this time she is, however, invariably relieved. The effect of the remedy upon her is unusually profound. She remains in a semi-unconscious state one to three minutes following the final inhalation. I mention her case in the present tense because, although much better, she continues to send for me at intervals of two or three months, in order to gain the relief which she never fails to find in a single inhalation.

CASE III. was that of a young lady who received the attack while in church. I found her in a convulsive condition as to limbs and fingers, and unable to respond, being apparently unconscious of the sound of my voice. The right hand was occasionally clutched over the heart. There was the very small left radial pulse as compared with the right. Removing the patient to fresher air I gave her brandy with aqua ammoniæ, meanwhile sending for nitrite of amyl. No change had taken place when it arrived. I administered two drops. With the first inhalation the patient, evidently startled, began to shriek wildly, in spite of which I continued the inhalation, and was rewarded by the return of her ability to

speak. She felt relieved of pain, which had been "frightful." After two additional inhalations, which relieved her still more, the patient was removed to her residence, where, after one more administration of the remedy, I left her in comfort. On the following day she complained of a sore and heavy feeling in the cardiac region, for which I did nothing. The next morning she came to my office to ask relief from this discomfort. A single inhalation of the nitrite of amyl relieved her completely, and up to the present time (a lapse of thirteen months) she has suffered no return of the paroxysms.

CASE IV. was that of a fourth young lady, who for several months had experienced a dull pain in the cardiac region, no remedy which she had received having relieved her. I tried the effect of valerianate of zinc and Quevenne's iron. A slight relief was the result. Within three weeks of her first visit, I was one day called to her house. Found her in a paroxysm of angina pectoris. I went prepared with the nitrite of amyl. She could not bear more than one drop. Five inhalations of one drop each relieved her, not only of the paroxysm, but also of all traces of the pain to which she had for months been the victim. Heretofore unable to move quickly, to go up stairs, or drive, without additional pain, she subsequently was, and has ever since been, able to take long walks, dance, run up stairs, and ride horseback, without the slightest sensation of discomfort.

The four patients, whose cases I have cited, were of ages between 19 and 25 years, and the closest examination revealed no hint of any affection of the heart.

CASE V. was a female, æt. 57. The attack for which I treated her had been preceded during the past seven years by four others of a milder type. I found the patient almost in a state of collapse; face livid; surface of the body bathed in perspiration; pulses small, especially the left radial; there was agonizing pain about the heart extending to the occiput, left shoulder, and upper left arm; singultus, globus, great dyspnœa, and difficulty of speech. The heart sounds were weak but normal. The organ was acting irregularly. I administered nitrite of amyl, by inhalation, in five drop doses. The patient experienced immediate relief, and in the course of thirty minutes resumed an almost natural look, colour returning to the cheeks, breathing becoming much freer, and the ability to speak being restored. Attacks of lesser degree appeared during the next hour, which I vainly endeavoured to relieve by hypodermic injections of morphia and a variety of powerful stimulants. The patient was growing rapidly weaker from the effects of the first paroxysm. Under the circumstances I felt unwilling to continue the use of the nitrite of amyl, but nothing else gave her the slightest relief, hence I again administered it in smaller quantity. It quenched the pain, and permitted the patient to assume a horizontal position. I then began to give her strong doses of brandy and aqua ammon. every quarter or half hour, but she gradually and quietly sank into a new collapse, which ended in death four hours after the first attack.

Here was a case in which I found the patient in the very throes of death, out of which I am confident that nothing but the nitrite of amyl could have delivered her. She became sufficiently restored to receive the ministrations of her pastor, speak with her family, and be prepared for the end. Besides this she was freed from pain, and died calmly. The

shock and effect of the first spasm were more than her heart could bear with impunity, as will be seen by the details of the post-mortem.

The autopsy revealed a heart well covered with fat; valves normal; slight atheroma of the aorta ascendens; right ventricle extremely thin and flabby; left ventricle in an apparently fair condition, but rather light in colour; papillary muscles shortened and thickened; liver markedly fatty; no other organs examined. The microscope showed that the muscular tissue of the heart was in a state of decided fatty degeneration. It is probable that this condition of the heart would be accused as the cause of the paroxysms by those who believe that angina pectoris *depends* upon a diseased state of the organ.

Wood, in his *Therapeutics*, thinks the administration of nitrite of amyl, when the heart is in an advanced stage of fatty degeneration, is attended with danger, because of its effects upon the cardiac muscle. In the case last mentioned, however, death would have ensued in a few minutes after I reached the patient but for the quick effects of the nitrite; other remedies were perfectly impotent.

I have presented many conflicting opinions as to the nature of angina pectoris, but, supported by the judgment of acute and reliable observers, have endeavoured to show that the disease neither necessarily depends upon cardiac affections, nor is confined to middle and advanced age, as is asserted by various influential writers; that there may be a functional as well as an organic form of angina pectoris, although I prefer to believe, with Laennec and Trousseau, that even when a paroxysm occurs as a complication of heart disease, it is rather a coincidence than a symptom, for the majority of cases of disease of the heart do not experience attacks of the angina; further, that no attacks are of pseudo or simulative nature.

Finally, I wish to urge upon physicians the great value, in this malady, of the nitrite of amyl. It is nearly invariably certain in its relieving effects, acts almost at once, is quickly, easily, and, with proper caution, safely given, and spares both patient and physician the tiresome, often ineffectual, use of the scores of remedies which are mentioned in the books.

In my hands this remedy has proved equally effectual in asthma (except when patients have been in extreme old age), as well as in the spasm of colic, in which I have used it with success in two cases. I would resort to it in any form of spasm, and I may add that since my paper was put in type, I have met with the happiest results in treating a case of obstinate spasm of the diaphragm by the same means.