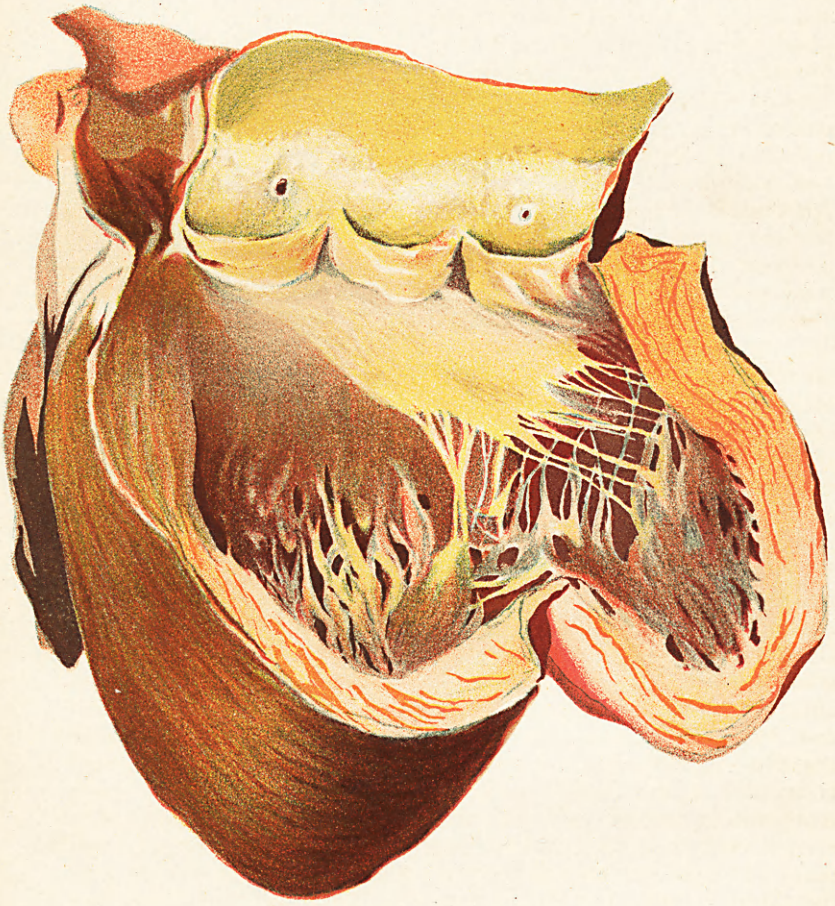

R. S. Reid.

J. Bartholomew, Edinr.

Part First.

ORIGINAL COMMUNICATIONS.

ARTICLE I.—*Upon Paroxysmal Angina Pectoris and other forms of Cardiac Pain, with some Remarks on the Diagnosis of Fatty Heart.* By GEORGE W. BALFOUR, M.D., F.R.S.E., Physician to the Royal Infirmary, Edinburgh, Consulting Physician to the Royal Hospital for Sick Children, etc.

GENTLEMEN,—Pain in the cardiac region is not only a common symptom in all diseases of the heart, but is often enough complained of when no cardiac disease is present; it is also an important indication of substernal aneurism. Pain below the left mamma is well known to be an almost constant complaint in chlorotic and debilitated—anæmic—individuals, whether male or female; and many neurotic individuals, whose nervous system is developed at the expense of their muscular, who are often said to be gouty, and frequently are so, complain of various uneasy sensations in the cardiac region, often amounting to a positive soreness of the heart. In some cases of infra-mammary pain the pain is occasionally wholly external, a form of intercostal myalgia, only a sign of muscular weakness, and in itself of no serious import. In most, however, the pain is truly cardiac in character, and though, strictly speaking, myalgic also, yet as the debilitated muscle whose action is painful is the central organ of the circulation, it is of much more serious import; it is still perfectly curable, but probably only differs in degree from the suffocative breast pang which has so many sad and fearful memories attached to it. Cardiac pain of this kind is obviously associated with imperfect nutrition, and when this imperfect nutrition is concomitant with an organic cause, the pain is apt to be more permanent and severe; and if to any physical cause of mal-nutrition we have superadded some physical source of obstruction necessitating a more than usually powerful action to overcome it, then we have a strain thrown upon the cardiac muscle which it is physically unfit for and which it resents, or, to speak more correctly, indicates by the pain which attends its action. Hence there are few cases of cardiac disease which in their uncompensated or imperfectly compensated stages are unattended by pain. This pain is more or less constant; it

may vary in degree, and it does so vary, but it is seldom absent, and its cessation is a tolerably certain sign that compensation is restored wholly, or, at all events, partially.

We have also a paroxysmal form of cardiac pain which is not associated, or, at all events, not usually recognised as associated with any detectable form of cardiac lesion. In rare instances the victim of this affection is woke up from sleep by the first attack, which, repeatedly recurring, cuts him off in a few hours, as in the case of the late Dr Arnold of Rugby.[1] In most cases the attacks are repeated, and they often last for years, and, with our modern methods of treatment, I believe they are not so likely to be so suddenly fatal as in the case just mentioned. Generally the first attack comes on as the patient is ascending some slight acclivity, or making some trifling exertion, possibly after a meal. He is suddenly pulled up by an excruciating pain shooting through the lower part of his sternum to his back-bone, often accompanied by a constrictive feeling, as if his chest was grasped by a mailed hand; the pain may remain localized, or it may shoot towards the shoulder down the left arm by preference, or down both arms. These are its most usual courses, but it may also occasionally shoot down the abdominal and lumbar nerves. The patient is at once brought to a standstill; he fears even to breathe, but if he chooses to make the effort, he can breathe freely enough; he feels a sensation of impending death, and a ghastly paleness overspreads his countenance. The pulse may intermit, or be feeble or irregular, but it is sometimes—especially if the angina be uncomplicated by any other cardiac disease—quite regular throughout the whole of the paroxysm. After a few seconds the pain ceases as suddenly as it came on, and the patient finds himself as he was, puzzled to know what has happened to him, and terrified at the prospect of a recurrence of the attack. There is in this seizure nothing pulmonary; the air enters freely into the lungs if the patient has the courage to breathe, and full inflation of the lungs has no influence upon the attack. It has no apparent connexion with cardiac or spasmodic asthma, neither does it arise from cardiac strain, or at least from any immediate and recognisable cause of strain. At first it seems to originate in some trifling exertion, or in some emotional excitement of the heart's action, but presently not even these exciting causes are necessary, and now and then, as we have seen, even the primary attack is without any such provocative, the patient waking from sleep in a paroxysm of anginous pain. In most cases it is not till after several and often numerous attacks, brought on by trifling exertions, that the disease arrives at such a pitch as to occur when the patient is asleep or at perfect rest, but sooner or later it reaches this stage.

Neither excruciating nor commanding are words strong enough to express the character of the pain in this affection; it seems to be

[1] Latham on *Diseases of the Heart*, 1846, vol. ii. p. 373.

something appalling, it unnerves the strongest mind, and death itself seems preferable to the repetition of a similar seizure.

Should death be the result of such an attack, the heart is found loose, flabby, and uncontracted—not exactly the condition in which one would expect to find it were death due to spasm, as is supposed by Latham, Heberden, and the older writers, but very much as it ought to be were death due to inhibitory paralysis, as Parry, Stokes, and Walshe[1] usually suppose. And the history of the mode of death in this disease corresponds thereto, for in such cases death is not usually instantaneous, as would be the result of a suddenly fatal spasm of the heart, but commonly occurs from a gradual sinking of the aortic pressure, the result of an equally gradual diminution of the heart's force, the pulse getting feebler by degrees until it ceases, and never passing at once from its ordinary force to a full stop, as would be the case in sudden cardiac spasm. Moreover, the pathology of angina pectoris as a neuralgia of the cardiac nerves is in accordance with this view of the cause of death, for we know that any sudden and violent pain produces sickness, faintness, and depression of the heart's action; and we also know that whatever produces depression of function in the fibres coming from the posterior root of a spinal nerve, and as its result pain or neuralgia, produces also depression of function of the motor fibres coming from the anterior root of the same nerve, and as its result subparalysis of the parts to which they are distributed.[2] Hence we have in angina pectoris two distinct sources of depression of the cardiac action: first, we have the directly depressing influence of a pain, the most acute and severe which the human frame can experience; and second, we have the action on the cardiac motor ganglia of the same cause which, acting on the sensitive nerves, gives rise to this excruciating agony, and we cannot but suppose that as a rule the functional depression of the motor nerves is not much less than that of the sensitive ones, that is, that the subparalysis of motion must bear some proportionate relation to the acuteness of the pain, which is the index of the functional depression of the nerves of sensation. This, however, is a rule to which there must be some exceptions; even in strictly spinal nerves the pain is often very much greater than the subparalysis, and there are many cases of angina in which the pain recurs at intervals for years in a most excruciating form without producing a fatal result; still the analogy is a fair one, and to some

[1] Anstie seems also to lean to this view, *vide Neuralgia and its Counterfeits*, London, 1871, p. 73. Dr Moinet likewise adopts the idea of paralysis in contradistinction to spasm, *vide Treatise on the Causes of Heart Disease*, 1872, p. 102. Eichwald, "Ueber das wesen der Stenokardie," etc., Wurzburger *Medizinische Zeitschrift*, IV. Bd. 4 heft., 1863, gives a good historical *résumé*, and regards angina as very often a reflex neurosis which is fatal by arrest of the heart in diastole.

[2] Anstie, *op. cit.*, p. 6; Van der Kolk *On the Spinal Cord*, etc., New Syd. Soc. Trans., 1859, p. 7.

extent must hold good. And there are many things in pathology which enable us to understand that a depressing cause may at one time act with greater intensity on the sensitive fibres, at another on the motor fibres, and at still another upon both alike. Take for instance an attack of peritonitis; the action on the heart in such a case is wonderfully analogous to that of angina pectoris, yet in one case we may have excruciating agony and but little depression of the heart's action, in another the agony and cardiac depression may proceed *pari passu*, and in still a third case we may have no pain at all, and yet the cardiac depression may prove rapidly fatal.

We must never forget that angina is sometimes feigned, and is occasionally of a purely hysterical character. Mrs Chisholm, whose case was formerly commented on,[1] was an instance of the former character, and a very clever imposter she was, giving a great deal of trouble before she was detected. Last winter we had in the case of the girl L.[2] a well-marked example of the hysterical variety. L. suffered from aortic regurgitation, from which she subsequently died, and the true character of her attacks was not at first suspected; but happening to find her in the fit at visit one day, I at once recognised its hysterical character, and took measures to prevent its recurrence, which were happily successful. I have seen several other cases of hysterical angina, but none so well marked as this, and, let me add, never any one in which there was less cause for suspecting hysteria, or more apparent reason for believing the paroxysms to be real.

We must never forget that pain in the heart, however caused, is, if not angina, at least very closely allied to it. The infra-mammary pain of chlorotic girls is almost invariably associated with pain in the heart itself, neurotic in character and depending on mal-nutrition. So also, in all other forms of spanæmia, pain in the heart, often radiating more or less along the nerves after the fashion of true paroxysmal angina, but less severe in character and more constant—less distinctly paroxysmal—is a very common event. I presume it is cases of this kind, probably of an unusually severe character, that Beau has dignified with the name of angina arising from the abuse of tobacco. The tobacco heart, however, as a rule, is simply a spanæmic heart, weak, irritable, and somewhat dilated, with an auricular murmur and an accentuated pulmonary second. In all spanæmic cases the pain or uneasiness—for it is seldom more than this—is persistent, with occasional exacerbations following exertion, a distended stomach, or some other exciting cause, and it is readily cured by improving the general health. The first step towards this is, of course, the giving up whatever has deteriorated the health, whether that be excess in tobacco, tea,

[1] Balfour *On Diseases of the Heart and Aorta*, 1876, p. 279.

[2] M. L., admitted to Ward XXXIII. on 28th January 1880; died from sudden asystole, 31st May 1880.

whisky, work, or anything else; and the next, so to regulate the diet, nature and hours of work and of rest, as to promote the due nutrition of the body, to enable it to live within its income, and to accumulate that margin of health which is our only safeguard from a thousand ailments. We can generally aid the attainment of this end by the exhibition of appropriate tonics; these, however, require to be specialized for each case. Along with general remedies, it is often advantageous in such cases to apply sedatives locally to the cutaneous nerves, either by putting a belladonna plaster over the pained part, or by rubbing the side and shoulder with an opiate liniment, and these soothing means are often, in slight cases, attended with such happy results as to lead to the neglect of the central cause. But whenever we find an accentuated pulmonary second, or an auricular murmur, associated with uneasy or painful sensation in the left breast, we must never forget that we have to do with a cardiac lesion which requires special treatment to cure it quickly and well. Between the slighter forms of mere cardiac uneasiness and the distinctly paroxysmal variety attended by severe precordial pain, shooting in various directions, but most commonly up the chest and down one or both arms, and accompanied by an overwhelming sense of impending dissolution and all the signs of serious nervous shock, there are many intermediate grades of severity. And though we might theoretically desire to exclude from the category cases of tumultuous and forcible heart-beat with lancinating pain, as apparently inconsistent with the idea of the subparalytic character of angina, yet this disease may begin in this way. Though I myself would not therefore apply the term angina to cases presenting these symptoms, yet true angina may develop out of them, as the following most interesting case abundantly proves:—

Case I.—A. S., a male, æt. 24, admitted to Ward V. 6th October 1877, complaining of palpitation and pain in the precordial region. The patient stated that he had suffered in this way for fourteen weeks, and that the pain was sometimes a mere uneasiness, and at others more acute. On admission, the patient presented a somewhat anxious expression, and was found to be well nourished, all his organs being healthy with the exception of the heart, which beat rapidly, 120 per minute, and in a somewhat tumultuous manner; the radial pulse was quite regular. On auscultation, the first sound at the apex was impure, the pulmonary second markedly accentuated, and a faint diastolic murmur was audible over the aorta at midsternum. The case was at once recognised as a serious inflammatory affection of the heart or ascending aorta, but whether it was endocarditis, myocarditis, or endarteritis, the symptoms were not distinctive enough to decide. The treatment consisted in the administration of full doses (15 grains) of iodide of potass in a bitter infusion three times a day, with perfect rest in bed, and an unstimulating diet. Under this treatment his heart quieted

down, but the pain increased, and became localized as a constant pain in the scrobiculus cordis, unaffected by pressure. This extension of the pain to the epigastrium was at first regarded as due to the iodide; but finding that it recurred in a distinctly paroxysmal manner, and that the pulse became rapid and feeble both during the paroxysm and for some little time after, the diagnosis was at once completed, and the affection stated in all probability to be an acute endarteritis of the cardiac end of the aorta, implicating the openings of the coronary arteries, with consecutive (febrile) dilatation of the heart. At first inhalations of nitrite of amyl gave great relief, the paroxysms lasting from five to fifteen minutes. But in the final attack, which commenced about four o'clock on the morning of the 24th of October, and lasted for about two hours, the amyl was of no use, and the only relief obtained from the intense agony was from chloroform inhalations.

At the autopsy on 25th October the body was found to be well formed and fairly muscular. Rigor mortis and post-mortem lividity well marked. Thorax: about six ounces of clear serum in each pleural sac, and about two ounces of similar fluid in the pericardium. The blood was remarkably fluid. The heart weighed thirteen ounces, the ventricular cavities were slightly dilated, and their walls slightly hypertrophied. The mitral orifice was enlarged, admitting four fingers; cone diameter 1·6. The cusps were natural. The tricuspid orifice was also enlarged, admitting six fingers; cone diameter 1·9. In the wall of the aorta, immediately beyond the aortic cusps, there was a ring of atheromatous thickening which involved the whole circumference of the vessel at and a little beyond the sinuses of Valsalva. In this situation the tunica intima had grown to twice or thrice its natural thickness, and presented a clear, translucent aspect, being only here and there affected with points of fatty degeneration. The openings of the two coronary arteries lay in the midst of this atheromatous area, and were both so extremely contracted as barely to admit the point of an ordinary surgical probe. The aortic valve allowed water to leak through it slightly, but it might be said to be practically competent. The cusps were slightly thickened at their free margins and above the *corpora aurantii*. The muscular substance of the heart was everywhere of good colour and consistence, and on microscopic examination presented no abnormality except the presence of a considerable number of reddish-brown pigment granules in some of the fibres. All the other organs of the body were perfectly healthy, but somewhat congested.[1] In this most interesting and probably unique case you will observe that we have a fatal cardiac lesion coincident with a practically healthy heart. The slight dilatation, slight hypertrophy, and trifling leakage through two orifices were nothing more than we may find in any

[1] Condensed from the Pathological Records of the Edinburgh Royal Infirmary. A chromo-lithograph of this heart is prefixed to this paper.

spanæmic heart, especially when febrile excitement coexists. Similar conditions are to be found in hundreds who make perfectly good recoveries. The fatal lesion was evidently the blocking of the coronaries; this was what was found, and this was precisely the lesion which had been predicted to be the most probable one. This opinion was based on the obviously causal connexion between the acute attack and the angina in this case, and on the fact that in by far the larger number of cases angina seems to depend upon some interference with the blood supply to the walls of the heart itself, and consequently to the intra-cardiac nerves. In a very large experience of angina, including over a dozen cases known to have been fatal, I have never failed to detect indications of defective blood supply to the heart, and in the only three dissections I have had this view has been abundantly confirmed. The case just narrated supports this opinion, as indeed almost all those recorded also do, even the celebrated case of Dr Arnold being no exception. For though his coronary artery—he had but one—was neither diseased nor obstructed, it is stated that, "considering the size of the heart, it appeared to be of small dimensions, and with some difficulty admitted a small director" (Latham); while the thin, soft, flabby texture of the heart sufficiently testified to the inadequacy of its feeding power. That is the true cause of angina; so long as the coronary arteries are able to feed the heart, it matters not what their structure is, we have no angina. Whenever from any cause the blood supply to the heart is insufficient, then we are liable to have angina, and in all such cases we have more or less of it. Pressure on the cardiac nerves in some part of their intra-thoracic course is an occasional cause of angina, but there is every reason to believe that even in this case it is produced by direct or reflex interference with the intra-cardiac circulation. The sequence of events in ordinary cases of angina seems to be, first of all imperfect nutrition of the cardiac muscle, associated with various uneasy or painful sensations, and generally (always?[1]) accompanied by the early physical signs of dilatation, usually with some hypertrophy. Next we have paroxysmal attacks of pain occurring when the heart is called upon for extra exertion, especially when a weak pneumogastric nerve is irritated by a distended stomach. At first these attacks only occur when the patient is debilitated from any cause, and his cardiac power thus temporarily impaired; in these circumstances improvement in health is followed by cessation of the angina, temporarily or permanently. By-and-by,

[1] I have never seen a case of angina in which these signs were not present. At present I am inclined to lay some stress upon the presence or absence of these signs of cardiac dilatation, as an indication that the angina is purely cardiac in character, or merely dependent upon the existence of substernal aneurism. But my experience, as yet, is insufficient to enable me to assign more than a moderate probability to this assumption.

as the nutrition of the heart becomes more impaired, the attacks are more readily brought on; the most trifling excitement of the heart's action, whether induced by exertion or by irritation of the terminal branches of the pneumogastric in the stomach, suffices to induce a paroxysm of pain; and in some cases at last the ordinary action of the heart is painful, aggravated by continually recurring paroxysms of greater severity, which wax and wane in that inscrutable fashion so common in other neuralgias.[1] Death occurs from asystole or rupture, sometimes during an attack, more often, perhaps, during a painless interval.

Such seems to be the history of all ordinary forms of angina. In traumatic angina the case is different. Here we have an individual apparently in the most perfect health, who meets with some trifling accident, and is at once plunged into a series of recurrent paroxysms of the most severe cardiac pain, which ere long terminate in death. The only case of this kind which has occurred to me is the following:—

CASE II.—J. L., a married woman, aged 50, stout, healthy, and who had passed through her life without an ache or a pain except those incident to maternity, slipped and fell on the street in the beginning of January. Being rather heavy, she was considerably shaken, but apparently not otherwise injured. By-and-by, however, anginal paroxysms set in and continued gradually to increase in severity. There was nothing abnormal to be detected about her heart, but she died suddenly in a paroxysm about the middle of March of the same year. Unfortunately I was unable to obtain a post-mortem examination; but from the sudden onset of the angina, its obvious dependence on the fall, and the resemblance of the most prominent symptoms to those of substernal aneurism, there is every reason to believe that the middle coat of the aorta was fissured transversely just above its cardiac origin at the time of the fall, that the angina was caused by pressure on some of the branches of the cardiac plexus by the gradually increasing aneurism thus formed, and that death was caused by this aneurism bursting into the pericardium.

The two cases just narrated have been the only cases in my own experience who have died during a paroxysm, and I am inclined to believe that with improved methods of treatment this will be an increasingly rare mode of death. All the others have died from asystole brought on in various ways during the painless interval. One, after many years' freedom from pain, died quietly after suffering for some time from gradually increasing dropsy and other signs of a dilated heart. Another, who during several years suffered from many comparatively slight attacks of angina,

[1] In regard to the periodicity frequently presented by neuralgias depending upon persistent grave organic lesions, *vide* Trousseau's *Clinical Medicine*, New Sydenham Society's edition, vol. I. p. 598.

lunched cheerfully with some friends, walked with apparent ease to a railway station but a short distance off, sat down, and died. Others, after suffering more or less intensely at intervals for years, died more or less suddenly without giving any indication of suffering. And one well-known literary man I myself saw die from ingravescent asystole about a week after his last paroxysm. He had for long suffered from angina, with all the signs of a weak, dilated heart due to arterial atherosis, and I had brought him safely through two most severe attacks with a comparatively painless interval of a year between them. Subsequent to the last attack he had been confined to bed with symptoms of pulmonary œdema, which is so common a result of a severe paroxysm. At last, when apparently fairly convalescent, he obtained permission from his medical attendant to rise from bed, and while dressing his weak heart failed and ingravescent asystole set in. There was no pain. When I reached his apartment he said, "Doctor, this is very different from anything I have had before," and he died quietly after drinking about half a glass of brandy given him in the hope of stimulating the heart to more vigorous contraction. The whole act of dying occupied about half an hour. At the post-mortem examination "both coronaries were found atherosed and obstructed, and in the substance of the left ventricle there was an elongated patch of advanced fatty degeneration. The limits of the patch were well defined, and the appearance presented bore a considerable resemblance to that of a hæmorrhagic infarction which had undergone fatty degeneration."[1] The heart itself was somewhat dilated.

Of course any deficiency in the blood supply to the cardiac walls must injuriously affect the intra-cardiac motor ganglia, and thus we have the element of danger introduced. When an anginous heart retains a fair amount of reserve power, and the pain is of short duration, the danger may not be great, and, as occasionally happens, especially in cases complicated with aortic regurgitation, the sufferer can sometimes by a voluntary effort call upon this reserve power, flush his heart with blood, and overcome his breast pang. I can recall several such cases; but the experiment is dangerous, and failure is sudden death.[2] If, however, the reserve power is feeble, then the danger is probably commensurate with the severity and duration of the pain, and it is a matter of paramount necessity to relieve this as speedily as possible.

Our forefathers had nothing to trust to but the external application of cutaneous irritants, and the exhibition of stimulants and narcotics by the mouth; a vain hope, when moments are precious,

[1] I quote from a letter received from Dr Wyllie, at that time pathologist to the Royal Infirmary, who made the dissection, the full report having been unfortunately lost. The specimen is in the University museum.

[2] Forbes, *Cyclopædia of Practical Medicine*, vol. i. p. 94, also mentions similar cases.

and to the time needful for absorption—twenty minutes in the most favourable circumstances—was superadded the further delay occasioned by a failing circulation. Modern discoveries have changed all this; we can now by inhalation thoroughly narcotize a patient in a few seconds, and by means of hypodermic injection secure in ten minutes a painless unconsciousness which will last for many hours.

Foremost among all our modern appliances for the relief of this dreadful breast pang we must place the nitrite of amyl; it is perfectly safe, and may be entrusted to the patient with the certainty that he will not injure himself by its use; in all slighter attacks it serves to give perfect relief, and in more severe paroxysms it alleviates even when it cannot completely remove the pain. It flushes the face, quickens the heart-beat, and has been experimentally found to lower the blood-pressure in animals to whom it has been administered. It was originally employed by Dr Lauder Brunton in the treatment of angina, on the supposition that this depends upon increased intra arterial blood-pressure. I quite agree that in all cases of angina the blood-pressure is probably always above the normal, but that it is abnormally increased at the moment of seizure has not yet been proved, and, so far as I know, is incapable of proof. The supposition of Dr Brunton that the case is so rests solely on a single sphygmographic tracing from the radial artery; but a pulse-trace only represents the local movement of the arterial wall, and for many obvious reasons can never be accepted as a correct indication of the intra-arterial blood-pressure. Further, if we accept the face-flushing as a proof of lowering of the blood-pressure, then I am in a position to state that two specimens of nitrite of amyl will flush the face in apparently the same degree, yet only one of these will relieve the pain. The specimen which relieves the pain is one which has been freshly prepared, or which has been kept in a hermetically sealed capsule. The other specimen, which does not relieve pain, has been kept for some time in an ordinary stoppered bottle. I make these statements from a large experience of the use of the nitrite of amyl, and chiefly base them on two cases, both of whom suffered for years from terrible angina. One of these cases had a loud, musical, diastolic aortic murmur, and he never felt well unless his wife could hear this murmur across the dinner-table, a condition indicating, of course, a much greater blood-pressure than when the murmur ceased to be audible and his sufferings began. During the last few years of his life he used many pounds of the nitrite of amyl, having it constantly with him, and inhaling it when required. His face was always fully flushed, and a certain amount of relief obtained, but this relief was only rapid and complete when the specimen employed was perfectly freshly prepared. The second case was somewhat similar. The sufferer was a medical man, and he used to soak his pocket handkerchief in the amyl

and go to sleep with it on his face. The conclusion I have arrived at from these facts is, that the relief to the pain of angina is obtained, not from lowering of the blood-pressure, but from the action of a volatile narcotic, which gradually escapes from the amyl when kept, unless it is enclosed in hermetically sealed glass capsules. As these are now readily obtained, we possess a remedy which can be safely entrusted to the patient, and which is certain to give relief in all ordinary attacks.

When the attack is a severe one, the amyl fails to give relief, however freshly it may have been prepared; of this I have been assured by many sufferers, and have myself repeatedly had occasion to observe it. Then our only resource lies in chloroform, which can only exceptionally be entrusted to the patient, but ought always, if possible, to be given by a medical man. It must be given freely, so as completely to narcotize the patient; and, when so given, I myself have not yet seen any case which was not relieved, though I have seen several in which the relief was not permanent enough to place the patient in safety. In these cases I have had recourse to the subcutaneous injection of morphia, using by preference Squire's solution of the bimeconate, of which I have injected half a drachm into each arm, without removing the clothes or in any way disturbing the patient. This, as yet, has never failed me: the chloroform sleep has passed into the morphia sleep, from which the patient has woke up some hours subsequently, free from pain, but exhausted, as we can readily suppose, and usually with some œdema of the lungs. You see, then, that I have no dread of chloroform in these cases, and by no means homologate Anstie's statement, that "the only kind of chloroform inhalation which would be useful in such cases would be that in which a carefully measured small dose of a weakly impregnated atmosphere should be inhaled, and without large experience in the administration of chloroform the practitioner will be unable to secure this effect with certainty; and the effect of a *powerfully* charged atmosphere, breathed only once or twice even, would be instantaneously fatal."[1] You have seldom an opportunity of seeing the usefulness of chloroform in angina pectoris in the wards, but you have all at least occasional opportunities of seeing its beneficial action in other kinds of cardiac pain; and what is safe enough in aortic regurgitation, for instance, cannot be dangerous in angina pectoris. Many years ago I knew an individual who was forbidden to take chloroform on account of a cardiac valvular lesion under which she laboured, and yet for long she secretly indulged in chloroform intoxication without any fatal result. So far from being unsafe in cardiac disease, it is often of the greatest use in these cases; it not only relieves pain, but regulates the circulation, now and then bringing the pulse back to the wrist, whence it had apparently fled for ever. In peritonitis this effect is occasionally quite remarkable.

[1] *Op. cit.*, p. 80.

I well remember one case of extreme cardiac pain and dyspnœa in a patient almost moribund from cardiac disease, and who did die only a few days subsequently, yet in her the immediate effect of chloroform inhalation was to restore the pulse to her wrist, to enable her to breathe more freely, and in few seconds, instead of being black in the face, pulseless, and gasping partly from pain, and partly from extreme dyspnœa, she became quiet, natural in appearance, and in a short time was able to lie down and rest. But, you may say, in angina the heart is almost universally flabby and fatty; is chloroform not dangerous when we have a fatty heart? The next case which I shall relate will be the best reply to this question. First, let me say that I doubt the possibility of diagnosing a fatty heart. We may suspect its existence, because the physical signs seem to warrant the supposition, while the conditions present are apparently favourable to its development. Thus there may have been a long-persistent spanæmic condition of the blood, or there may be a state of general (pernicious) anæmia, or there may be reasons for suspecting a purely cardiac anæmia from local causes, such as an atherosed and obstructed state of the coronary arteries, or an overgrown hypertrophy of the cardiac muscle which has got beyond the feeding powers of these vessels. For fatty degeneration of the cardiac muscle is the result of mal-nutrition, and seems never to be found apart from one or other of these conditions. But little acquaintance with pathology is, however, requisite to teach us that these conditions are not all of them easily and certainly recognisable, and that even when present fatty degeneration is not an invariable concomitant of any of them. Though, therefore, our suspicions may occasionally be right, they may more often be wrong. The signs of cardiac debility upon which we base our suspicions are much more commonly due to dilatation than to fatty degeneration, in spite of the possible co-existence of an arcus senilis. Besides, there are many cases of actual fatty degeneration in which there have been no faintings, cardiac asthma, feebleness of the pulse or of the cardiac impulse, no yellowness or pastiness of the complexion, and no arcus senilis—in fact, all the symptoms and signs connected with the heart have either been those of perfect health, or at all events they have not been such as are generally supposed to indicate fatty heart, yet the heart has been markedly fatty.[1] I may refer to the case of Bridget Henry, who died from chloroform in the Cincinnati Hospital, U.S., 13th October 1870,[2] as a well-known example of the conjunction of a normal impulse with a fatty heart; but indeed of this we could scarcely have a more

[1] While this is passing through the press this statement has received a most forcible illustration in the death, from rupture of a fatty heart, of one of our best known and most esteemed medical practitioners, whose healthy appearance and great vigour both of body and mind were entirely opposed to all the more generally received ideas in regard to this form of degeneration.

[2] *Chloroform Deaths*, by W. W. Dawson, M.D., Surgeon to the Cincinnati Hospital, 1871. Printed by Robert Clarke & Co., Cin.

striking instance than the case of Mrs Tait, which I will presently relate to you. No doubt the rapid and powerful action of chloroform renders it a very dangerous agent in incautious hands, and some diseased states of the heart, of which an enfeebled and anæmic condition are the chief, render it more sensitive to the action of chloroform, and more liable to be fatally overpowered by an overdose; but I know of no diseased condition which should deter us from its cautious employment when that is otherwise indicated, as I hold it imperatively to be in certain cases of angina pectoris, for what we desire to do in them can only be done by means of chloroform. I do not say *tuto, cito, et jucunde*, because the superlatives of these adverbs are more applicable than their simple positives. In a disease possessing such a pathological history as angina pectoris, the one great object is to free the cardiac nerves from the depressing influence of pain, which gives rise to that subparalysis in which the danger lies. This we can only do by narcotizing the nerve centres through which this action takes place, and so setting them free from all those influences which tend to depress the heart's action. The immediate result is a sensation of relief from pain, greater force and freedom of the heart's action, and a fuller pulse.[1]

Sulphuric ether has long been used with a similar intent; it is an admirable narcotic, and the chief objection to its use is that it is not rapid enough, taking always some minutes to bring the patient fully under its influence. Chloroform acts more quickly, even more effectually, and is perfectly safe. It is not always necessary for the medical man to administer it, though in some cases it is so. All that we require is to insure that the patient shall only have a moderate dose, and this we manage by giving him a chloroform smelling bottle, the fluid being poured over a piece of sponge, so that it cannot spill; this smelling-bottle he is told to hold to his nose, and to breathe as deeply as possible. In this way relief is obtained in a few seconds, and so soon as the narcotic influence is produced the smelling-bottle drops, and with it rolls away all risk of any overdose. Mrs Tait, long a nurse in this Infirmary, and who died in Ward XIII. on 30th March 1871,[2] was for the last few weeks of her life almost constantly under the influence of morphia or chloroform, or both, the morphia being injected hypodermically as soon as the chloroform narcosis was established, so that its soothing influence might come into play when that of the chloroform passed off. She was over eighty

[1] In his work on *The Bearings of Chronic Disease of the Heart upon Pregnancy, Parturition, and Childbed*, London, 1878, Dr Macdonald says, *apropos* of a primipara labouring under aortic insufficiency, "The patient looked pale, . . . and complained that she felt ready to faint with every pain." Though besought to give chloroform, he hesitated, but yielded at last, and then found that "under its employment the pulse became stronger and steadier, instead of feebler and more irregular, as I feared it might."—*Vide* p. 147.

[2] Margaret Tait, admitted 3d Jan., died 30th March, 1871.

years of age, and had long suffered from angina pectoris, the paroxysms of which were latterly very severe, and with but short intervals between them. She died at last, as I have said, not suddenly, but gradually, worn out by her age and sufferings. After death the aorta was found dilated, the orifice of the middle coronary artery—there were three in her heart—almost entirely blocked up by atheromatous deposit, and her heart not only thin-walled and somewhat dilated, but of a pale yellowish tint, soft, and thoroughly fatty; no muscular fibres could be more completely degenerated; yet chloroform produced in her no dangerous symptoms, and, far from shortening her days, seemed to prolong them. I may also add that her apex beat was tolerably firm to the last.

But however satisfactory our treatment of the paroxysm is, the treatment during the intermission is quite as important, and is often attended by even more striking results. From the pathology of this disease already given you will understand that during the intermission I use every endeavour to improve the patient's general health, and especially to tone up his heart. To this end he must be warned to avoid every source of excitement, to take perfect rest in a mild and equable climate, where he ought to be much in the open air, driving or sitting, but not walking. His diet must be so regulated as to consist of the blandest, most nutritious, and unstimulating foods, avoiding everything likely to prove difficult of digestion or give rise to flatulence, and being particularly careful as to stimulants, the action of which is ultimately to weaken the heart, and the more stimulating such drinks are, and the greater the amount partaken, so much more rapidly is this result attained. Of course the whole system must be carefully attended to, and acid tonics, pepsine, mild laxatives, or any other general remedies given that may seem to be required. But as all secondary functions depend for their perfect discharge very much upon the condition of the great central organ, so our best mode of improving the gastric or hepatic functions will always be to improve the heart. To that end we have as yet only two remedies of any importance: these are arsenic and digitalis. Very shortly after Fowler introduced his tasteless ague drop, arsenic was employed experimentally in a great many diseases, angina pectoris amongst the rest, and in several cases it was found to be successful.[1] Since then it has been often used in such cases. Anstie has declared it to be "an invaluable remedy in cardiac neuralgia," as well as "the most important prophylactic tonic"[2] we can employ in these cases, and my own experience is quite to the same effect. Arsenic is indispensable in all forms of weak heart accompanied by pain. It is useful in all such cases, and in many it is quite successful in putting a stop to angina. Several cases have occurred to me in

[1] Forbes, *Cyclopædia of Practical Medicine*, vol. i. p. 95.
[2] *Op. cit.*, pp. 78, 79.

which arsenic alone has removed angina after a few weeks' treatment, not only temporarily, but permanently. The ordinary dose is from three to five minims twice a day after food, but the dose may sometimes be advantageously pushed till slight physiological symptoms appear, and thereafter continued so long as desired in a dose just short of that needful to produce these effects. Its mode of action is somewhat obscure. Besides being a good general tonic, as well as a special tonic to the heart, it seems also to exercise some modifying influence on the nerves which renders them less liable to pain in spite of a continually advancing degeneration. But however it may act, arsenic is a drug well worthy of confidence in the treatment of angina, and associated with iron and strychnia it forms a combination specially valuable in all cardiac neuroses. One great difficulty in the administration of arsenic lies in its tendency to irritate the bowels of some patients; this may be overcome by the addition of opium, or by the diminution of the dose, for it not infrequently happens that a constitution sensitive to the injurious action of a drug is also sensitive to its curative action. I have frequently found this to be the case with arsenic, and it is always worth remembering. I distinctly remember one gentleman specially sensitive to the action of all drugs, but particularly to that of arsenic, who could not bear it in larger doses than one milligramme of arsenious acid daily (0·015 gr.); two milligrammes gave him discomfort, one was well borne. He was not aware in what the evil effects of the drug consisted, nor did he know what good I expected from its use, yet in about a fortnight he said, "I feel my breathing easier now; my heart is steadier, and I can go upstairs better than for many years." No better results could have been obtained from larger doses, and, indeed, from his sensitiveness, had these been persisted in, the drug would assuredly have had to be entirely given up. From my belief in the connexion between defective nutrition of the heart and cardiac pain you will readily understand that I put considerable faith in the use of digitalis in these cases, in small tonic doses repeated night and morning. Ten minims of the mixture of digitalis, or one granule of Nativelle's digitaline, is an ample dose, and I have seen nothing but good result from the use of this drug. One granule of digitaline night and morning, with arsenic, strychnia, and iron twice a day after food, is a sort of model treatment for such cases; and this treatment, coupled with nourishing, unstimulating food, abundant rest and fresh air in a mild and equable climate, is often attended by the happiest results in those cases susceptible of improvement, which are, in truth, by no means of infrequent occurrence.