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MUSCULAR CRAMP, IN RELATION WITH THE PHENOMENA OF ANGINA PECTORIS AND "INTERMITTENT CLAUDICATION OF THE EXTREMITIES."

By F. Parkes Weber, M.A., M.D., M.R.C.P. (Lond.)

Edward Jenner,[1] founding his views on observations of his own and others, first suggested the "coronary" theory of angina pectoris—that is, the theory which supposes angina pectoris to be due to disease of the coronary arteries—which was afterward supported and developed by Parry,[2] Kreysig,[3] Reeder,[4] etc. H. Huchard, of Paris, has recently in an admirably clear manner formulated his views on the subject (*Maladies du Cœur*, 2d ed., Paris, 1893). Huchard fully supports the coronary theory as far as true angina pectoris[5] is concerned; he thinks that it is a cramp of the heart-muscle, at first suggested in 1768 by Heberden,[6] and considers it to be a condition of the heart analogous to that in the limbs, which has been described by Charcot as "claudication intermittente des extremités." Huchard thus unites the "coronary," "cramp or spasm," and "claudication intermittente" theories of angina pectoris.

According to this author, the case stands as follows: H. Bouley, Jr.[7] (1831), described an affection of the extremities in horses, to which he gave the name of "claudication intermittente." In this rare affection the horse's hinder extremities are usually affected. The animal goes naturally for some distance, but after some time commences to limp, and then, if whipped on, appears to suffer greatly, and at length something like the following takes place: The animal falls down, is evidently in great pain, and there appear to be anæsthesia and a condition of rigidity in the affected extremities. These phenomena gradually pass off in

[1] See his letter in "An Inquiry into the Symptoms and Causes of Syncope Anginosa," by Caleb Hillier Parry, M.D., Bath, 1799, p. 3.

[2] Parry: Op. cit.

[3] See F. L. Kreysig: "Die Krankheiten des Herzens." Berlin, 1816, Pt. ii. pp. 543–546.

[4] H. Reeder: "A Practical Treatise on the Diseases of the Heart." London, 1821.

[5] He separates from true angina pectoris the nervous affections which he designates as "pseudo-anginas," a term previously employed by Dr. W. H. Walshe in "Diseases of the Lungs and Heart," 1st ed., 1851, p. 435.

A. Lartique already in 1846 distinguished what he called "pseudo-angina," or "pneumogastralgia" from true angina pectoris. See "De l'Angine de Poitrine." Paris, 1846, p. 112.

[6] See William Heberden, the elder: "Some Account of a Disorder of the Breast," in Med. Transactions of the Coll. of Phys. in London, 1772, vol. ii. p. 64. Heberden's account of angina pectoris was read in 1768, the same year in which it was independently, though less thoroughly, described in France by Rougnon.

[7] Arch. Gén. de Médecine, vol. xxvii., Paris, 1831, p. 425.

half an hour or so; the animal regains its natural appearance, but, if whipped on again, the attack will recommence, and so on. At necropsies upon such horses, obliteration of the main artery of the affected limb (the abdominal aorta in cases where both hinder extremities were affected) has been found, and this obliteration of the arterial channel may be from thrombosis, pressure by a tumor from without, etc., but the precise nature of the cause does not affect the present subject under discussion.

Charcot (1858) described similar symptoms in a man. In all cases[1] the cause is arterial ischæmia of the affected extremity, due to the obliteration of the main artery supplying it, and the collateral circulation, though sufficient when the limb is at rest, is quite insufficient during exertion when an increased blood-supply is demanded. The condition, then, is a premonitory sign indicating the danger of gangrene, and Charcot narrates that one of his patients suffering from this affection subsequently underwent amputation of his leg for gangrene.

According to Huchard,[2] Dr. Potain was the first (1870) to point out the analogy between "claudication intermittente des extremités" and angina pectoris, and to strengthen this analogy it may be stated that one of Charcot's patients, suffering from intermittent claudication of an extremity, subsequently died in an attack of angina pectoris.

If this analogy be strictly accepted, the coronary ischæmia[3] resulting from stenosis of the coronary arteries causes the cardiac muscle to fall into a state of spasm, just as the limb in Charcot's cases of "claudication intermittente des extremités." But if angina pectoris is really due to spasm of the cardiac muscle, how is it that the pulse may appear, at least in some cases, unaffected or comparatively little affected during the attack?

Such is the difficulty when the *heart* is concerned, but even in *intermittent claudication*[4] *of the extremities*, in spite of Charcot's cases, perhaps it may be asked if cramp forms a necessary feature. Charcot thought it was allied in nature to rigor mortis, but an earlier observer who apparently alluded to cases of the same nature (though he did not

[1] Soc. de Biologie, Comptes-rendus, 1858, vol. v. p. 225 et seq. The part affected was the right lower limb of a man, aged fifty-four, who suffered from aneurism of the right common iliac artery. See also later papers by Charcot and others.

[2] H. Huchard: "Maladies du Cœur." 2d ed., Paris, 1893, p. 608.

[3] On the subject of coronary ischæmia some of Cohnheim's experiments are remarkably interesting. See Cohnheim's "Lectures on General Pathology," New Sydenham Society's Transactions, 1889, vol. i. p. 35. After recalling the experiments of Von Bezold, Samuelson, and his own, he states that the ischæmia in such experiments probably produces its baneful effect through the action of the cardiac ganglia. He goes on to point out the difficulties in the way of accepting the "coronary" theory of angina pectoris. It must be remembered, however, in such experiments that the lesions are acute and affect a previously healthy cardiac muscle. The experiments do not, therefore, necessarily afford an analogy with what takes place when the coronary arteries become gradually stenosed by disease.

[4] This term admits of being used in English, and is, perhaps, as satisfactory as "meiopragie," the other term used by Huchard, and which he states that he has borrowed from Dr. Potain (Huchard, op. cit., p. 209).

use the same term as Charcot), made no mention of either rigor-mortis-like rigidity or ordinary cramps in his description. I refer to Sir Benjamin Brodie, to whose writings on this subject my attention was directed by Mr. Maidlow.

Charcot, who described his first case in 1858, considered intermittent claudication of the extremities as a warning sign of impending gangrene; and Sir Benjamin Brodie, in 1846 (*Lectures on Pathology and Surgery*, London, 1846, p. 360), when describing the premonitory signs of senile gangrene, though he did not use the term "intermittent claudication," evidently alluded to the same class of phenomena as Charcot. Brodie must therefore, I think, be considered as having not only described the condition in man before the celebrated French doctor, but, as may be seen from the following quotation, he at the same time clearly drew up the analogy between it and angina pectoris.

Brodie's exact words are:

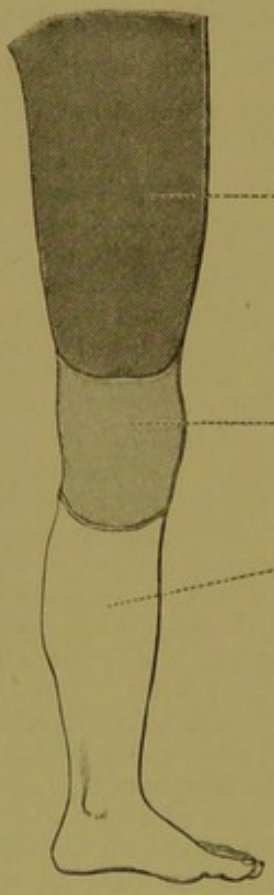
"If you cross-examine a patient who has mortification of the toes, he will generally tell you, that for three or four years preceding, he has had occasional pains in the lower limbs; a sense of numbness in them; that his feet were liable to be cold; that when they again became warm, after having been cold, they have been very painful; and that he has had a sense of weakness of the muscles. Such patients walk a short distance very well, *but when they attempt more than this the muscles seem to be unequal to the task, and they can walk no further. The muscles are not absolutely paralyzed, but in a state approaching to it.* The cause of all this is sufficiently obvious. The lower limbs require sometimes a larger and sometimes a smaller supply of blood. During exercise a larger supply is wanted on account of the increased action of the muscles; but the arteries being ossified or obliterated, and thus incapable of dilatation, the increased supply cannot be obtained. This state of things is not peculiar to the lower limbs. Wherever muscular structures exist the same cause will produce the same effect. Dr. Jenner first, and Dr. Parry, of Bath, afterward, published observations which were supposed to prove that the disease which is usually called "angina pectoris" depends on ossification of the coronary arteries. . . . When the coronary arteries are in this condition they may be capable of admitting a moderate supply of blood to the muscular structure of the heart; and as long as the patient makes no unusual exertion, the circulation goes on well enough; when, however, the heart is excited to increased action, whether it be during a fit of passion, or in running, or walking up stairs, or lifting weights, then, the ossified arteries being incapable of expanding so as to let in the additional quantity of blood which, under these circumstances, is required, its action stops and syncope ensues; and I say that this exactly corresponds to the sense of weakness and want of muscular power which exists in persons who have the arteries of the legs obstructed or ossified."

From this quotation it may be seen that Brodie, whilst clearly describing an intermittent claudication of the extremities, certainly did not consider cramp or other rigidity of the muscles to be a necessary accompaniment. Conversely, the following case shows that cramps in an extremity, though induced by arterial obliteration, may be unaccompanied by the other features of intermittent claudication of the extremities. The case is that of a man who, in 1889, had his left common (?) iliac artery obliterated from an accident. He is now able to get about fairly well, and has not noticed anything analogous to intermittent claudi-

cation of the limb as described by Charcot, though, when sleeping with both legs drawn up, he is awakened by cramp in the left leg (only). Here are some short notes of the case, which I am enabled to give through the kindness of Sir William Savory, whose house surgeon I was when the man was admitted at St. Bartholomew's in 1889.

Michael M., aged forty-one years. Admitted December 20, 1889; discharged May 30, 1890, for the Convalescent Home. The diagnosis in the case-book is "Injury to spine and abdomen—occlusion of left common iliac artery; partial paralysis of left lower extremity."

Anæsthesia on May 16, 1890.



Following is a short abstract of the most important features:

The patient was crushed between buffers of engine and stage; very much collapsed; numbness of left leg; pain and tenderness in lower part of back and abdomen; loss of power over left leg; complete loss of sensation below middle of thigh except over an area somewhat corresponding to that of the small sciatic. Pulsation in left posterior tibial artery could just be felt. Marked tenderness over lower part of abdomen. A distinct tumor (perhaps of extravasated blood?) could soon be felt in left inguinal region of abdomen.

Dec. 26, 1889. Bowels opened (without medicine) for the first time since admission.

27*th*. Can move thigh fairly, not leg or foot.

31*st*. Certainly no pulsation in femoral. Discoloration of heel.

Jan. 1, 1890. Very slight pulsation made out in femoral.

8*th*. Gangrenous patch formed on outside of leg.

10*th*. Sensation returning in leg slightly; gangrene of heel.

The sloughs separated and the sores became healthy. Anæsthesia

appeared to be gradually and partially clearing up and power of motion to be returning. On May 30, 1890, could raise the leg from the bed. Opposite is a diagram of the anæsthesia in the limb on May 16, 1890.

Patient seen again in November, 1893.

Had had in the meantime gangrene of portions of the toes of left foot and had undergone tenotomy of the left tendo Achillis (with wrenching of the ankle-joint) on account of the left foot having become fixed in position of equinus.

Very faint pulsation can be felt in the left femoral artery. There is still considerable impairment of sensation in left lower extremity. Over the greater portion of the front of the leg below the knee, and over part of the sole of the foot, superficial sensation is entirely absent (as tested by gentle touching with a pin, etc.), but there is hyperæsthesia for deep sensation, especially when pressure is applied over the shin. Perhaps some of this tenderness is periosteal. The left leg feels colder to the touch than the right one. The left lower extremity is decidedly somewhat wasted, especially at the calf. He does not complain of any symptoms on walking analogous to "claudication intermittente des extremités" as described by Charcot in man. He walks about fairly well considering that in spite of the operation there is still partial ankylosis in the left ankle-joint. A curious fact is that when lying in bed and asleep he is sometimes awakened by cramp in the left leg. This is only when he goes to sleep with his legs drawn up, and although both legs be drawn up, the cramp is only in the *left* leg.

An explanation of ordinary[1] muscular cramps (such as those sometimes affecting the legs of healthy persons after violent exertion and at night time) is that they are due to the irritation caused by the accumulation of waste products in the affected muscles. By the waste products the endings of the centripetal nerves in the muscles are irritated and the cramp or tetanic contraction is a reflex result. This theory explains the frequency of cramps on going to sleep, for the commencement of sleep favors reflex movements and cramps are perhaps merely an exaggeration of the jerking movements in limbs which in some people not infrequently wake them up just as they are dosing off. This theory of cramps accounts, moreover, for the tendency to their occurrence in healthy people after violent physical exertion, such as rowing in a race, etc. The violent exercise causes an excessive catabolic process in the muscles and consequently such an accumulation of waste products in these parts that even a healthy circulation takes considerable time to remove them. In the meantime there is a tendency to cramps in the muscles which have been most used, and a rowing man who assures me of the frequency of cramps after violent rowing says that, when they occur, they do not necessarily follow immediately after the exercise, but may come on at any moment for some time afterward.

[1] Of course cramps may arise from different causes. They need not be reflex, but may arise from direct stimulation (experimental or due to disease) of any part in the nervous motor tract. The cramps of cholera, however, and of choleraic diarrhœa may be due in part to the accumulation of waste products, which the sluggish blood-stream and diminished quantity of blood are insufficient to clear the muscles of.—Cf. Dr. P. Z. Murphy, *Lancet*, 1853, vol. i. p. 511.

The same theory of cramps accounts also for the peculiarity of their occurrence in the patient Michael M., whose case I have just given. In his case they occur only when sleeping "with his legs drawn up" and are limited to the left lower extremity. This is the extremity in which the blood-supply is defective, owing to the obliteration of the common(?) iliac artery on that side, which took place some years ago. In his case probably the blood-supply furnished by the collateral circulation is insufficient and has difficulty in washing away the waste products, and this difficulty is increased when the position of the patient in bed ("with his legs drawn up") renders the circulation in the extremity still more defective. Hence the cramps in that extremity at night-time, the time which favors reflex movements.

If the "waste-products" theory of muscular cramps be correct, then it is not surprising if, quite apart from Charcot's rigor-mortis-like rigidity, ordinary cramps should occur frequently in such patients as those whose cases Charcot has described under "claudication intermittente des extremités," since this is a condition primarily due to arterial ischæmia, and in which the waste products are imperfectly washed away. These cramps, however, need not necessarily be associated with intermittent claudication of the extremities, and the previous quotation shows that Brodie did not attach great importance to them. It is, I think, the powerlessness described by him, rather than cramps or rigor-mortis-like rigidity, which is to be considered as the essential part of "intermittent claudication of the extremities."

I will endeavor to make my meaning clearer by the following consideration: "Claudication intermittente" is obviously a question of degree; a muscle imperfectly supplied with blood cannot do as much work as one properly supplied, but a limit to the possible work exists in both cases, easily reached in the former, but only on the most violent exercise in the latter. Thus an athlete in a race may, so to speak, "use himself up" completely, so that at the end of the race he is incapable of any further exertion. This is really nothing but a "claudication" in a healthy man, exactly analogous to "claudication intermittente" in the extremities of patients with diseased arteries. It is noteworthy that when a man has undergone continued violent muscular exercise cramps may follow, but in this case no cramp necessarily takes place at the moment of giving up; on the contrary, as I have already mentioned, a tendency to cramps may exist for some time after. These considerations, I think, illustrate the subject under discussion and really help toward explaining the relation of the cramps to the sudden feebleness in "claudication intermittente" of the extremities, namely, that both are favored by the same condition, which is stenosis of the main artery of the affected part.

From the extremities I shall now return to the heart, and think the same considerations have some bearing on the subject of angina pectoris.

If, as seems probable, angina pectoris is really a "claudication intermittente" of the cardiac muscle, due to coronary ischæmia, then, as in the extremities, cramps will not be a necessary feature; and, indeed, the pulse almost precludes the idea that ordinary angina pectoris is a cramp of the heart-muscle. It will then be asked: When the main arteries are stenosed, why should not cramps be likely to occur in the heart as in the extremities? It probably does occur[1] at times, and is expressed by syncope, which[2] may be the fatal termination in angina pectoris.[3] According to this view one may discard the "cramp" or "spasm" theory of angina pectoris, whilst still retaining the "intermittent claudication" theory, regarding the latter term as synonymous with the "coronary" theory, and as affording the explanation of the phenomena of true angina pectoris; when it is accompanied by syncope, then, and then only, can cramp of the heart be considered as playing any part in the *ensemble* of an attack of angina pectoris.

I will only add a few words, and these are regarding the "coronary" theory of true angina pectoris, which I have almost taken for granted in the foregoing words, but which seems to be as yet not universally accepted. A chief objection to the "coronary" theory is that stenosis of a coronary artery may be found post-mortem in individuals who have never been known to suffer from angina pectoris. This has been accounted for by anastomoses in the coronary circulation (see those pointed out by Dr. Samuel West, *Lancet*, 1883, vol. i. p. 945, and others). The alleged cases of true angina pectoris without organic disease[4] of the coronary arteries form a second objection to the "coro-

[1] A cessation of the heart's action in systole (or spasm) is probably the most frequent form of cardiac syncope.

[2] This is the real justification for the name "syncope anginosa," suggested by Parry. Parry wrote: "I think it evidently appears that the angina pectoris is a mere case of syncope, or fainting, differing from the common syncope only in being preceded by an unusual degree of anxiety or pain in the region of the heart," etc. See Parry, op. cit., p. 67.

[3] If angina pectoris were a cramp of the heart the pain or anguish felt during the attack might be explained by supposing the nerve-endings to be stimulated by the contraction of the heart-muscle; which explanation would make the sensory phenomena of angina pectoris analogous to the pain felt during cramp in the calf of the leg. If, however, as I maintain, angina pectoris be not necessarily associated with cramp of the heart, another explanation must be adopted, such as that the pain is directly dependent on anæmia of the myocardium (Cf. Huchard, op. cit., pp. 618 and 619). This would make it analogous to the pains long ago recognized (see Percivall Pott, Chirurgical Works, 1775, p. 794) as premonitory signs in senile gangrene; the accumulation of waste products may likewise have something to do with its production, as in the production of the more chronic pains or "stiffness" in the limbs after violent exertion.

[4] On this question some of the earlier recorded necropsies are particularly interesting. In the first necropsy on a case of angina pectoris, recorded by Heberden, nothing to account for death was found. A gentleman had apparently heard of Heberden's description of angina pectoris, and considered that the symptoms corresponded with his own. Accordingly, on April 16, 1772 (see Med. Trans. Coll. Phys., London, 1785, vol. iii. p. 1), Dr. Heberden received a letter, signed "Unknown," from the gentleman describing his symptoms, and saying that he had left directions that, after his death, Dr. Heberden should have an opportunity of dissecting his body to find out the cause of the disease. The gentleman died suddenly within three weeks afterward, and Heberden obtained the services of Mr. John Hunter to make the examination, but no pathological change likely to cause death was discovered. Jenner (see Jenner's letter to Parry, in Parry, op. cit., p. 3), however, thought that in this case the coronary arteries were

nary" theory; but in these cases spasm of the coronaries may explain the attacks, as was maintained in the fatal case attributed by Huchard to the poison of tobacco (see Huchard, *op. cit.*, pp. 626 and 712). These are the chief objections to the "coronary artery theory," and they are not unanswerable; whilst, on the other side, the well-known action of nitrite of amyl and trinitrin affords support to this theory. The beneficial action of nitrite of amyl in some cases of angina pectoris was first discovered by Dr. Lauder Brunton,[1] and has since been abundantly confirmed. He considered that it acted by lowering the peripheral resistance to the arterial stream and thereby diminishing the amount of the heart's work. It is obvious, then, how this therapeutic explanation of Dr. Brunton may be adduced in support to some extent of the theory that in patients with angina pectoris the coronary arteries are stenosed, and that, owing to this, the blood-supply to the cardiac muscle is limited, allowing it to do only a limited amount of work.

More detailed evidence from post-mortem statistics as to the presence of coronary disease in fatal cases of angina pectoris is given, amongst recent authors, by Huchard,[2] and it would be out of place to repeat it here; this paper deals with the part played by cramp in angina pectoris, and the "coronary" theory has been practically assumed.

It now rests with me to recapitulate the chief points in my paper:

True angina pectoris has long been separated from the nervous cases, or "pseudo-anginas;" it has been regarded as due to stenosis of the coronary arteries and as analogous to certain premonitory signs of dry gangrene, due to stenosis of arteries in the extremities. In the present paper I do not pretend to have discussed these questions in their entirety, but have confined myself to the rôle played by muscular cramps in relation to the other phenomena. In this paper it is maintained that muscular cramp does not take any necessary part in angina pectoris or in the phenomena preceding senile gangrene in an extremity, but that cramp is likely to occur in any muscle where an accumulation of waste products takes place, whether this accumulation be caused in healthy muscles by rapid catabolism due to excessive exercise, or by insufficient removal of the waste products in cases of disease due to stenosis of the main arteries and consequent diminution of the blood-stream through the affected part. It is further held that these cramps, when they do occur in angina pectoris, are expressed by the syncope, which may accompany the attack and lead to a fatal termination.

not examined. In a later case, examined by Hunter (narrated by Dr. J. Fothergill, in Medical Observations and Inquiries, 1776, vol. v. p. 225), ossification of the coronary arteries was found, and on Hunter's own death, in 1793, from angina pectoris, Sir Everard Home, forewarned by Jenner, discovered that the coronary arteries were diseased.

[1] See Dr. Lauder Brunton: "Nitrite of Amyl in Angina Pectoris," Clin. Soc Reports, February, 1870, vol. iii.

[2] Huchard, op. cit., pp. 798-847.

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