Inquiries in cardiac physiology and pathology / by John A. McWilliam.

INQUIRIES IN CARDIAC PHYSIOLOGY AND PATHOLOGY.

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I.

CARDIAC FAILURE AND SUDDEN DEATH.

No doubt there is often a considerable amount of looseness in the use of such phrases as "death from sudden failure of the heart's action," "death from syncope, occurring in a weak heart," etc. Such conclusions are often made to cover cases in which the fatal issue has, in all probability, resulted from other (unascertained) causes. But when all such instances are excluded, there remain a large number of cases, accurately observed and carefully recorded, in which there is every reason to believe the occurrence of death to be directly determined by a sudden and complete cessation of the cardiac function—a catastrophe attributed sometimes to the influence of more or less slight causes (for example, increased strain on the organ), at other times remaining apparently mysterious.

The organic lesions most commonly associated with sudden cardiac failure are well known, namely—degenerative changes of a fatty or fibroid nature in the muscular walls, aortic regurgitant disease with its more or less effective compensatory changes in the organ, and diseased conditions (atheromatous, calcareous, or sclerotic) of the coronary arteries. But sudden stoppage of the heart's action has often been observed apart from the occurrence of gross structural lesions, associated with no very obvious or extensive alteration in the cardiac tissues. All that has been noticed in some cases has been a "flabby condition of the muscular tissue," and other appearances of more or less uncertain significance; not infrequently the cardiac substance has exhibited no pronounced morbid change. How then is an abrupt cessation of the heart beat brought about?

Sudden cardiac failure is usually assumed to take the form of quiescent standstill in a state of diastole, as a result of the action of one or more of a variety of causes—over-distension or strain of the organ due to sudden exertion or excitement, pressure on the heart or rupture of its walls, inhibitory influences transmitted by the vagus nerve, or some cause involving an abrupt loss of contractile power from failure of the intrinsic mechanism (for example, an impairment or arrest of the coronary blood-supply, or exhausting influences of a more obscure character.)

A long series of experiments on the mammalian heart has convinced me that in ordinary circumstances sudden cardiac failure does *not* usually take the form of a simple ventricular standstill in diastole; indeed, such a mode of failure is, in my experience,

very exceptional, if one exclude those cases in which there has been some very obvious cause acting from without upon the heart, for example, excess of chloroform, profuse hæmorrhage, asphyxia, etc. When sudden failure occurs apart from the influence of such gross causes—when the cardiac collapse has been due to more obscure and impalpable changes—the state of the organ is, as a rule, entirely different from simple standstill in a state of diastolic quiescence. It assumes, on the contrary, the form of a violent, though irregular and inco-ordinated, manifestation of ventricular energy. Instead of quiescence, there is tumultuous activity, irregular in its character and wholly ineffective as regards its results. And a similar condition obtains even in many cases due to some of the obvious and tangible causes of cardiac failure above alluded to, for example, excess of chloroform.

Such an irregular phase of cardiac activity was first described by Ludwig and Hoffa[1] many years ago, as the result of the application of strong galvanic currents or faradic currents to the ventricles of the dog's heart. Kronecker[2] found more recently that he could induce a similar result by puncturing with a needle a certain part of the ventricular substance. The condition in question has been studied by various investigators, and its general characters are now clearly ascertained. The normal beat is at once abolished, and the ventricles are thrown into a tumultuous state of quick, irregular, twitching action; at the same time there is a great fall of blood-pressure. The ventricles become distended with blood, as the rapid quivering movement of their walls is wholly insufficient to expel their contents. The muscular action partakes of the nature of an arhythmic, inco-ordinated, and rapidly-repeated contraction of the various muscular bundles. Some bundles are in a state of contraction while other bundles are relaxed, and so, instead of a co-ordinated contraction leading to a definite narrowing of the ventricular cavity, there occurs an irregular and complicated arhythmic oscillation of the ventricular walls which remain in a position of diastole. This condition is very persistent, and it is easy to kill a dog by applying a faradic current to the ventricles. Various names have been applied to denote the peculiar form of action under consideration—fibrillar contraction, delirium cordis, intervermiform movement, etc.

In regard to the readiness with which the heart assumes this peculiar and disastrous mode of action, I have been able to form some important conclusions in the course of a long series of experiments on the mammalian heart, conducted with the organ exposed, the thorax being laid open, and artificial respiration kept up through a cannula in the trachea. I have again and again been impressed with the fact that in certain conditions of the cardiac tissue, the fibrillar mode of contraction (delirium cordis) may be induced with the greatest ease; it may occur as a result of apparently trivial causes. Gentle handling of the organ, contact with the cut end of a rib, slight friction of the ventricular surface at any part, or indeed a mere touch with the finger, may be followed by the immediate manifestation of this remarkable form of inco-ordinated action. And not only is this the case—that extremely slight causes are often sufficient to induce the fibrillar condition—but a similar phenomenon not very infrequently occurs in the absence of any distinct and tangible exciting cause; the ventricles suddenly go off into a state of de-

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[1] *Zeitschrift f. rat. Medicin*, 1850, vol. ix.

[2] *Sitzungsberichte d. Berliner Academie*, 1884.

lirium quite apart from the operation of any recognisable irritant or immediate disturbing agency. *It is quite palpable that in certain circumstances the readiness of the ventricles to assume the fibrillar form of contraction is strikingly augmented; their susceptibility becomes so heightened that the sudden change in their mode of action occurs as a result of very slight causes* (which would be entirely insufficient to bring about such a result in ordinary circumstances), *or even in the absence of any direct recognisable cause.*

Such a heightened ventricular susceptibility is associated with circumstances and conditions which are very difficult to define with precision. Broadly stated, the conditions obtaining are always abnormal ones, involving a more or less marked disturbance of the normal nutrition of the cardiac tissues. They are frequently present in the course of prolonged experiments, when the thoracic cavity has been laid open for some time and the natural circulation has been in some degree modified by this and other causes (for example, stimulation of various nerves, imperfect aëration at times, etc.), though the heart continues to beat regularly and forcibly, and the blood-pressure is tolerably high. The nutrition of the ventricular substance has been altered and impaired, its irritability has been markedly exaggerated, and the tissue has passed into a state of unstable equilibrium. Such an association is, as is well known, a very common one both in physiological and in clinical experience; an imperfectly-nourished tissue usually shows, at some phase or other, a pronounced alteration and temporary exaltation of its excitability. The liability of the mammalian heart to sudden failure from the supervention of the fibrillar mode of contraction (delirium) has often been unpleasantly impressed upon me by the not infrequent interruption of experiments bearing on other points of inquiry in consequence of the ventricles unexpectedly going into delirium.

It seems to me in the highest degree probable tuat a similar phenomenon occurs in the human heart, and that it is the mode of cardiac failure and the direct and immediate cause of death in many cases of sudden dissolution. It is strange indeed if the phenomenon of fibrillar contraction is never manifested in the human heart, iu any of the various conditions of altered and disordered nutrition to which it is liable. For this phenomenon has been observed in all warm-blooded animals examined; it is, as far as I am aware, a universal feature in the behaviour of the mammalian heart; and at the same time it is much more readily induced and much more persistent in the higher mammals than in the lower forms. In the hedgehog, guinea-pig, and rat, for example, ventricular delirium is often of tolerably brief duration; the normal mode of action is after a time recovered. In the cat delirium is easily induced and is very persistent, generally, if not uniformly, fatal in the absence of remedial measures; and in the dog all observers concur in regarding it as invariably destructive of life.

It is hardly to be expected that such a widespread and probably universal feature of mammalian cardiac action should be unrepresented in the case of man. It probably does occur in man, and as a rule, if not invariably, with fatal result. In this way can be reasonably explained many instances of sudden and unexpected heart failure that without such an explanation must be regarded as inexplicable and mysterious, For anyone, I think, who has looked closely into the mechanism of abrupt cardiac failure as the determining factor in many cases of sudden death, must admit that there are many things very hard to explain, or rather unin-

telligible, when viewed in the light of the usual hypothesis of diastolic standstill from such causes as the following: inability to contract against the arterial pressure, from over-distension of the cavities, from reflex inhibition, from an interference with the coronary blood-supply, from direct pressure on the organ, from rupture, or from some obscure cause such as an abrupt (and unaccountable) loss of the "intrinsic irritability." For in the case of a heart which has been doing its work sufficiently well (as has often happened) to enable its possessor to discharge all the duties of a fairly active though not laborious life, it seems incomprehensible how an abrupt, utter, and irretrievable collapse should occur even in the absence of any sudden and material increase in the amount of work to be done by the organ; how a heart that has been beating in such a way as to keep up an arterial pressure compatible with moderate exercise of mind and body should all of a sudden become incapable of maintaining in favourable circumstances even the lowest arterial pressure compatible with the very existence of life. Examples of fatal heart-stoppage, even under favourable conditions (for example, during periods of inaction or even during sleep) will readily present themselves in the minds of my readers, and also the occurrence of cardiac failure in circumstances of so slightly unfavourable a character as to make it hard to conceive how these could on purely physical grounds have determined the disastrous result. I may quote a very few brief statements bearing on this point, and applicable to heart failure in different classes of cases. Dr. Gairdner[3] (writing on angina pectoris and sudden death) observes: "In some of the very worst cases indeed it has been clearly ascertained that very shortly before a fatal paroxysm the patient has been in a state of entire comfort and tranquillity, with a regular and normally-acting heart, and all the functions apparently so well adjusted as to involve no appearance of any disease tending to shorten life.....It is plainly out of the question to suppose that a chronic, and in its very nature gradually advancing lesion like fatty degeneration or disease of the coronary vessels, is the direct and immediate cause of a death which occurs in a moment..... The cardiac fibre which carried Dr. Chalmers safely over the last three weeks of his life, with its harassing duties and active exertions in various places, cannot be reasonably supposed to have become suddenly so much more diseased (physically speaking) that it must needs be disabled to the extent of ceasing to act altogether in the absolute quiet of an undisturbed night, after a day peacefully and happily spent in his own home."

Hilton Fagge,[4] referring to fibroid disease of the heart, remarks: "In a great many cases, however, the heart has gone on discharging its functions quite normally, as far as can be known, until the patient has suddenly fallen down dead. For example, Dr. Whipham[5] has recorded the case of a gentlemen, aged 29, who fell dead from his horse while riding in Hyde Park, having started in good spirits and apparently perfectly well, and having never before exhibited any symptoms of cardiac disease. The abrupt stoppage of the organ in such circumstances is at present altogether unintelligible."

Walshe,[6] speaking of valvular diseases, states: "There is one

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[3] Reynolds's *System of Medicine*, p. 582 and pp. 559-560.

[4] *Principles and Practice of Medicine*, edited and completed by Dr. Pye-Smith. Second edition. Vol. i, p. 939.

[5] *Pathological Transactions*, xxi.

[6] *Diseases of the Heart*. Fourth edition, pp. 394-395.

among the number of which the tendency to kill instantaneously is so strong that the fact must always be borne in mind in estimating its prognosis—and that is aortic regurgitation.....The manner of death is clearly syncopal; but the immediate mechanism, whether mechanical or dynamic, is difficult enough of comprehension.....That aortic reflux may at any moment kill instantaneously, and it kills by syncope, from which Nature makes no appreciable effort to rouse the victim, stand then as incontestable truths.....I am unable to supply any theory, based on actual observation, capable of explaining the clinical fact."

It will thus be seen that in certain forms of cardiac failure none of the usual hypotheses are at all sufficient to meet the case, and that there is much room for the assumption of such a cause as I put forward in this paper.

There is no doubt that syncope of a *non-fatal* character is associated with an inefficient action of the heart, which may depend on one or more of a number of different causes, such as—inhibitory influences exerted through the vagus nerves, and dependent on reflex excitation, on blood conditions in the medulla, etc.; inability of the ventricles to act effectively on account of abnormal conditions of over-distension or emptiness of their cavities; pressure on the organ; defective coronary supply; etc. The temporary cardiac failure is accompanied by a rapid fall of arterial pressure, and increased facility for the ejection of the ventricular contents, especially in those cases where the heart has been over-distended or struggling against a high arterial resistance. Many temporary attacks of syncope met with in the course of organic cardiac disease and in other conditions are probably of this character. Indeed, it would seem probable that an essential difference between the state of the heart in many cases of non-fatal syncope and that present in other cases that prove fatal, is that in the former instance the cardiac insufficiency is due to a change in the rhythm and force of the *ordinary* movements of the organ, while in the latter instance there is present an *extraordinary* change in the character of the ventricular activity, involving a practically irremediable abolition of its function as a muscular pump. In other words, it is probable that *fatal* syncope often differs from *non-fatal* syncope in the supervention in the former case of fibrillar contraction (or delirium) in the ventricular muscle; this seals the fate of the depressed heart by arresting the circulation and by causing a rapid exhaustion of the ventricular energy in consequence of the violent and continued excitement of the contractile mechanism.

In the great majority of cases where sudden death is caused by cardiac failure, there is, no doubt, an altered and impaired state of nutrition in the cardiac tissues, sometimes rendered palpable by degenerative changes recognisable with the microscope or pointed to by the presence of disease in the coronary arteries or conditions indicating a changed coronary supply (for example, aortic regurgitant disease). In other instances there is, no doubt, a disordered nutrition, which gives no outward and visible sign to histological examination, just as conditions of defective nutrition (attended by a striking increase in the ventricular susceptibility) occur, without recognisable structural change, in the heart of a healthy mammal when the organ has been placed under abnormal conditions—as in the course of experiments conducted with the thoracic cavity laid open.

With regard to angina pectoris, there is no ground for supposing that fibrillar contraction is present during the paroxysms. Indeed, such an idea is negatived by the fact that the pulse-beat

can commonly be felt during the attack, whereas the fibrillar mode of ventricular action involves a complete abolition of both cardiac and arterial pulsation. Moreover, we know that angina is closely associated with a high blood-pressure; on the other hand, fibrillar contraction is accompanied by a rapid fall of blood-pressure towards zero. At the same time, there appears to be a great probability that the occurrence of sudden death in angina is often determined by the ventricles passing into delirium. There is good reason to believe that it is in this way mainly that some fatal attacks of angina reaching a suddenly fatal issue differ from previous attacks.

In some cases of fatal syncope there appear to be good grounds for assuming that the heart has been brought to a diastolic stand-still by powerful inhibitory impulses transmitted along the vagus nerves. But even in such instances it is very possible that fibrillar contraction ultimately comes into play—that the inhibited heart may be put beyond the chance of recovery by the lapse of the ventricles into delirium. We know that in mammals it is possible to keep the heart arrested only for a comparatively short time by vagus stimulation; the rhythmic action then becomes resumed. Of course it is possible, on the other hand, that the inhibition is a very powerful one in man, and that the period may be long enough to cause death. The assumption of inhibitory arrest cannot, in any case, be made to cover the whole of the cases of sudden cardiac failure, for such failure, involving immediate death, is often unattended by any grounds for inferring the occurrence of a sudden excitation of the cardio-inhibitory mechanism.

Rupture of the heart has been found only in a small minority of the recorded cases of sudden death, and plugging or obstruction of the coronary vessels must be very rare as an immediate cause.

Over-distension of the cardiac cavities has already been mentioned as a cause put forward to explain a sudden arrest of the heart in diastole, the state of matters being comparable to what obtains in an over-distended bladder. Such a sudden arrest I have never witnessed in the course of my experiments on the mammalian heart, apart from inhibitory causes or excess of anæsthetics; I have never seen any sudden and fatal ventricular failure taking place in this way, even in hearts that had been exposed for hours, and had become much enfeebled, though still able to keep up a sluggish circulation of the blood. In man, again, it is important to note the significant fact that *sudden and unexpected cardiac failure occurs by no means most commonly in cases of dilated heart with thin and flabby walls, upon which over-distension or strain might most readily tell in the way of arresting their action in a purely mechanical fashion.*

The danger of gastric flatulence in heart disease has recently been emphasised by Dr. George Harley in this JOURNAL (1888), the danger of cardiac failure from the direct pressure of a wind-distended stomach affecting the heart through the diaphragm. Sudden death brought about in such a way would probably be determined by fibrillar contraction in the ventricles. I have often seen such a result from pressure on excitable ventricles.

The mechanism of fibrillar contraction I cannot enlarge upon here. I have already discussed it in the *Journal of Physiology*, Vol. viii. I regard the condition as an outburst of disordered excitement in the ventricular muscle; a rapid succession of incoordinated contractions travelling peristaltically along the complexly interlaced anastomosing fibres of which the ventricular walls are built. Recovery is quite possible in many animals,

most readily in the lower mammals. In the cat I have seen a restoration of the normal cardiac beat after the ventricles had been in delirium for more than an hour. Rhythmic compression of the ventricles with the hand was kept up, and pilocarpine was injected into the circulation. Pilocarpine has a decided tendency to check the fibrillar movement, depressing, as it does, the excitability of the cardiac muscle.

In conclusion, admitting the possibility of sudden syncope from plugging or obstruction of some portion of the coronary system, and the probability of sudden syncope from inhibitory influences, or in consequence of mechanical overdistension, or from pressure on the organ, *it is very probable that in many of these cases the fatal issue is directly determined by the occurrence of fibrillar contraction in the ventricles. Moreover, there is reason to assume that, in a certain number of instances, where none of the above-mentioned causes are present in any marked or dangerous degree, a sudden, unexpected, and irretrievable cardiac failure may, even in the absence of any prominent exciting cause, present itself in the form of an abrupt onset of fibrillar contraction* (*ventricular delirium*). The cardiac pump is thrown out of gear, and the last of its vital energy is dissipated in a violent and prolonged turmoil of fruitless activity in the ventricular walls.[7]

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[7] I have used the term "syncope" in the general sense in which it is often employed, to indicate cardiac failure irrespective of the consideration as to whether the heart's contractions are entirely abolished, or are merely so much enfeebled as to be ineffective.

II.

ELECTRICAL STIMULATION OF THE HEART IN MAN.

It is, of course, only in a very limited number of the cases of cardiac failure that the question of artificial excitation of the heart beat becomes one of practical importance. In the majority of instances where a more or less sudden heart stoppage occurs there are underlying conditions which obviously render direct stimulation of the organ inapplicable or hopeless. But, on the other hand, in certain forms of cardiac arrest there appears to be a possibility of restoring by artificial means the rhythmic beat, and tiding over a sudden and temporary danger. Such is especially the case in those instances where cardiac failure assumes the form of an inhibition of the heart beat by impulses reaching the organ along the vagus nerves. There is much reason to believe that among cases of sudden and fatal syncope such a mode of heart failure is not very rare. It probably obtains in many of those instances where sudden death has been caused by blows upon the præcordia or stomach (apart from the occurrence of any important visceral lesion); also in many cases of death from fright; and, as Lauder Brunton has urged, from irritation of sensory nerves (for example, the fifth) in certain phases of the administration of chloroform. It is in regard to sudden cardiac failure from the last-mentioned cause that the question of direct excitation of the heart becomes most important; for in most of the other examples the circumstances are usually such that death occurs before any remedial measures—however effective they might be known to be—could be employed. There is much probability in the view that sudden stoppage of the heart under chloroform may often be due to a sudden excitation of the cardio-inhibitory centre in the medulla oblongata and a consequent arrest of the cardiac beat by inhibitory impulses carried down from the medulla by the vagus nerves; such appears to be especially probable when the heart suddenly stops, early in an operation, when a comparatively small amount of chloroform has been given and the anæsthesia is more or less incomplete.

Now we know that when the mammalian heart has been inhibited through the vagus nerve it is quite possible to excite an immediate renewal of the rhythmic action by direct stimulation of the organ; during the inhibitory standstill the spontaneous

beat is arrested, but the mechanism for the execution of a beat can still be brought into action by the application of a direct excitant. Hence it would seem very probable that in sudden cardiac failure depending on inhibitory causes in man, the heart's action might readily be excited by artificial means and the temporary danger to life averted. No doubt it is very possible, as I have already suggested in a former paper, that the fate of the heart may be sealed in cases of fatal inhibitory arrest by the supervention of fibrillar contraction or heart-delirium in the ventricles. But, apart from the occurrence of such a disastrous complication, or before its inception, artificial excitation might be useful in rousing into action a heart that has been arrested by a temporary cause; for example, by inhibitory impulses profoundly depressing the rate and force of its action, or causing it to stand still in diastole.

As regards the means to be employed for the purpose of directly stimulating the cardiac action in cases of sudden failure, various expedients have been recommended—among others the application of galvanic and faradic currents to the region of the heart, electro-puncture, mechanical irritation by a fine needle passed into the organ, and the application of heat to the præcordia.

Now, with reference to the influence of such excitants upon the heart, there are some experimental results fraught with the greatest significance. It is within the experience of everyone who has worked with the mammalian heart that the application of strong galvanic and faradic currents to the ventricles is attended with disastrous results; an immediate abolition of the normal beat, and the occurrence of a wildly inco-ordinated, arhythmic contraction of the ventricular muscle (fibrillar contraction or heart-delirium), attended by a great and rapid fall of blood-pressure, and, in the higher mammals (the dog at least) by speedy death. The ventricles are utterly unable to pump out the contained blood, and the circulation quickly comes to an end.

If such a phenomenon had been observed simply in one or two sorts of animals, it might be urged that the human heart is possibly exempt from such a danger. But the phenomenon in question is one that occurs in more or less extreme form in every mammal examined, and, indeed, in birds as well. I have myself observed it in the hearts of the dog, cat, rabbit, hedgehog, rat, mouse, guinea-pig, hen, pigeon, and blackbird. It is, in all probability, of universal occurrence in the hearts of warm-blooded animals; and the fact that it is more easily excited and more extreme in its character in the higher mammals than in the lower forms is one of much significance.

It has been argued that the strength of current employed clinically would not be sufficient to bring about the grave result in question. In regard to this consideration there is a point of much importance, namely, that the susceptibility of the ventricular tissue to the occurrence of fibrillar contraction may become enormously augmented in certain conditions of impaired and disordered nutrition. Of this fact I have been most fully convinced in the course of my investigations on the mammalian heart, for on numerous occasions I have seen the ventricles going off into fibrillar contraction under the influence of extremely slight irritant causes, slight pressure or friction, faradic currents too weak to be felt on the tongue, etc.—causes which would have been entirely insufficient to bring about such a result in a normal, or approximately normal, heart. Now it is precisely in conditions of heart failure, where the nutrition of the cardiac tissues must

necessarily be impaired and altered, that it is proposed to apply galvanic and faradic currents clinically. In the light of physiological experience such a procedure would seem to be liable to the gravest issues, and to be wholly unjustifiable. Of needle puncture the same must be said.

On the other hand, it is to be borne in mind that von Ziemssen, Dixon Mann, Hartmann and others have described the results they obtained from the application of electrical currents (galvanic and faradic) to the human heart, and no untoward event occurred. But these experiments were conducted on healthy persons; and,

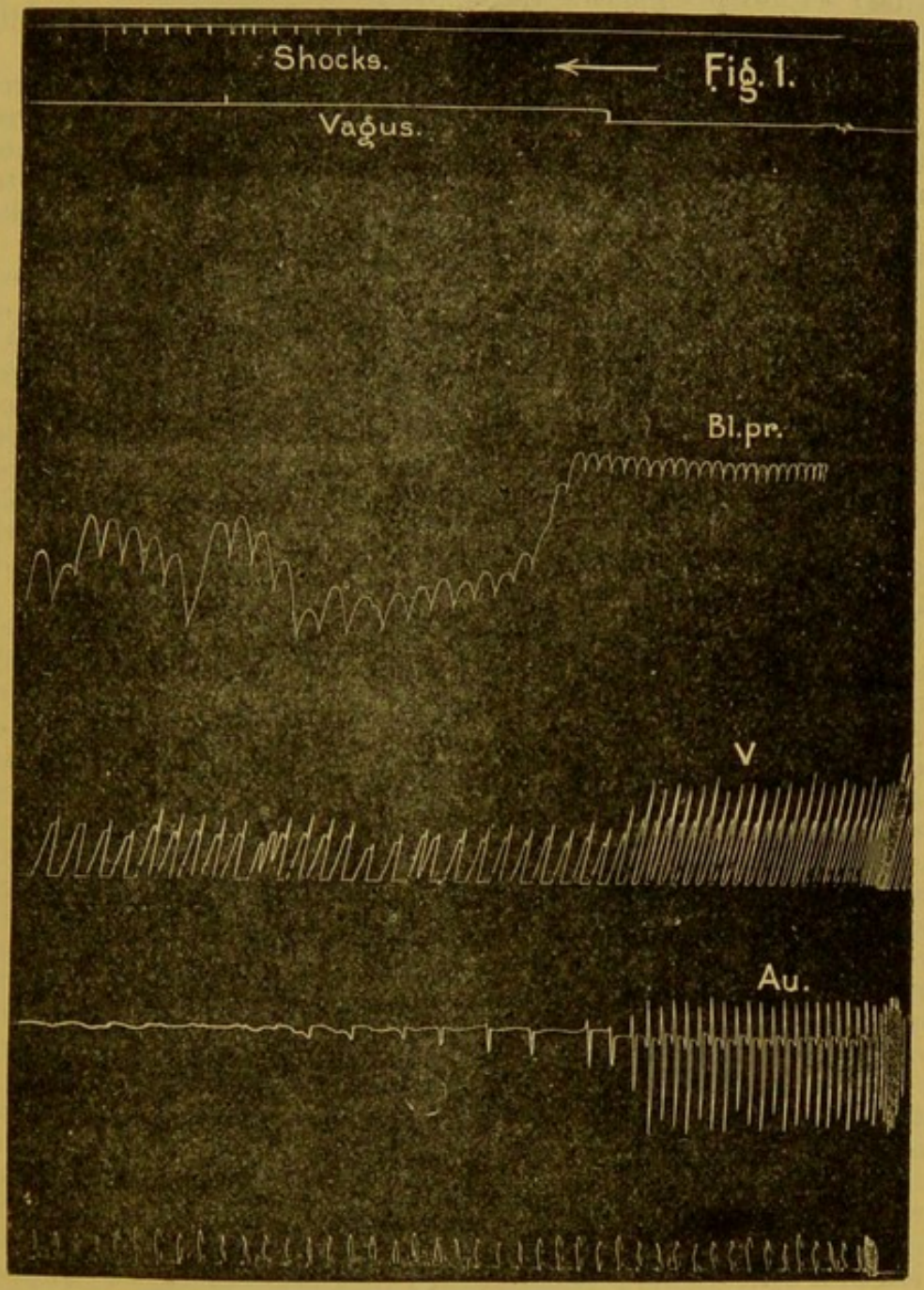

Fig. 1 (to be read from right to left).—Cat's heart. Shows the action of the auricles (Au) and ventricles (V), and also the course of the blood-pressure (Bl. pr.). In the ventricular trace an upward movement of the lever = contraction. In the auricles a downward movement = contraction. The heart was depressed by vagus stimulation at the point indicated in the second tracing from the top of the figure. A marked fall of blood-pressure ensued. Then (at the points indicated by the marks in the uppermost tracing) single induction shocks were sent into the heart. These shocks cause a more rapid and more effective series of ventricular beats, accompanied by a decided improvement in the blood-pressure. The lowest tracing marks time in half-seconds.

as I have already stated, the susceptibility of the normal heart is not to be taken as in any measure a test of the strength of stimulating current which can be borne without disastrous result by hearts that have suffered from a derangement of their nutritive processes, and have passed into a state of unstable equilibrium. It seems to me that the experiments of the observers above mentioned do not in any way serve as proof of the safety of galvanic and faradic currents applied to the heart; they do not deal with the organ in such conditions as those in which the clinical use of such currents is often recommended—conditions that physiological experience shows to be frequently associated with an immensely increased susceptibility to the occurrence of fibrillar contraction and fatal collapse of the cardiac function.

Further, even if galvanic and faradic currents were free from the grave dangers to which I have alluded, there would be very little to be said in favour of their use as direct cardiac stimulants in a sudden and urgent crisis. Galvanic and faradic currents too weak to induce fibrillar contraction in a heart of depressed excitability have a comparatively trivial influence in exciting or accelerating its beat. We want a much more effective and speedy mode of exciting rhythmic contraction, and one that will have a direct and powerful influence in calling forth a series of beats in the depressed or inhibited heart, while at the same time free from the danger of throwing the ventricles into delirium. Such a mode of excitation seems to be available in the form of a periodic series of single induction shocks sent through the heart at approximately the normal rate of cardiac action. A single induction shock readily causes a beat in an inhibited heart, and a regular series of induction shocks (for example, sixty or seventy per minute) gives a regular series of heartbeats at the same rate. Never on any occasion have I seen fibrillar contraction excited by such a mode of stimulation. In order to elucidate more fully the influence of a series of induction shocks upon the inhibited heart, I have frequently (in the dog, cat, and rabbit) performed such experiments as the following. The animal being chloroformed, and means being taken to preserve, as far as possible, the normal temperature, the thorax and pericardial sac were laid open; artificial respiration was kept up through a cannula introduced into the trachea. The heart was inhibited by stimulation of the vagus nerve in the neck, and then a periodic series of induction shocks (regulated by a metronome) was applied to the apex of the ventricles. Contraction of the auricles and ventricles was recorded by an adaptation of the graphic method; a blood-pressure tracing was simultaneously made in the usual manner. In this way I was able to obtain an accurate record of the various changes, while at the same time some further information was obtained by direct inspection of the heart. A series of single induction shocks excites a corresponding series of cardiac beats; the ventricular contraction precedes the auricular contraction when the exciting shocks are applied to the ventricles. Each systole causes the ejection of a considerable amount of blood into the aorta and pulmonary artery, and a marked rise of the blood-pressure at each beat. The mean pressure is raised from the low point to which it had fallen in consequence of the cardiac standstill; it does not, however, attain the normal height, even though a long series of beats is elicited by the stimulating shocks. This fact is due to the feebleness of the auricular contraction under inhibitory influence. For the auricles beat so feebly (in response to the stimulation) that they are unable to pump their contents into the ventricles in the normal vigorous fashion. The ventricles fill very

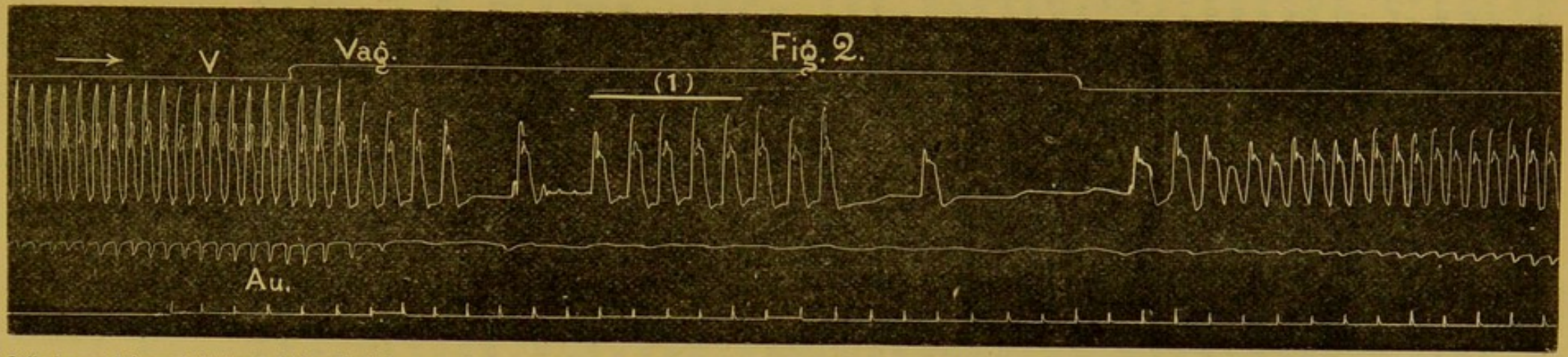

Fig 2 (to be read from left to right).—Cat's heart. Uppermost tracing shows the period of vagus stimulation. Second tracing (marked V) records the action of the ventricles (upward movement = contraction); and the third tracing (marked Au.) the action of the auricles (downward movement = contraction). The lowest line indicates half-seconds. After the heart had been depressed and enfeebled by vagus stimulation a periodic series of (eight) induction shocks was applied to the ventricles. The resulting group of beats is marked (1). The individual beats are much improved in strength as compared with the spontaneous beats occurring before and after. The beneficial effect of direct excitation is very apparent.

slowly, and, at the moment of contraction, contain much less blood than in the normal state; hence the amount of blood thrown into the aorta in a given space of time is much diminished, and the arterial pressure fails to attain its ordinary height. Nevertheless, the artificially excited beats are decidedly advantageous, inasmuch as they arrest the fall of the blood-pressure and even cause a rise —involving an improvement in the circulatory flow in the coronary system, as well as in the other vessels.

Moreover, the advantages of direct excitation of the heart are not by any means confined to the beneficial effect induced in the blood-pressure; nor to this combined with the obviating of any over-distension of the ventricular cavities with blood. For induction shocks applied to the heart exert an important influence, not only in removing the arrest of rhythmic contraction and restoring the periodic beat, but in counteracting in a marked degree the depressing effects of vagus stimulation upon the contraction force of the cardiac muscle. During a period of inhibitory standstill, a cardiac beat excited by a direct stimulus is, as a rule, markedly weakened. But when a regular series of stimuli is employed, the contraction power becomes rapidly improved; the beats increase in force, and often approach the normal strength. Similarly, when the heart has been greatly slowed by inhibitory impulses, the spontaneous beats are frequently reduced in energy; but the application of a periodic series of induction shocks (at approximately the normal rate) leads not only to the manifestation of a regular series of beats at the same rate, but, as a rule, to a pronounced augmentation in the force of the individual beats.

Hence it is evident that, in addition to the improvement in the blood-pressure resulting from direct excitation of the heart by a series of induction shocks, there is also a beneficial effect exercised upon the contractile mechanism of the inhibited heart. The depressing influence exerted through the vagus nerve upon the rhythm and contraction force are in large measure counteracted by direct excitation of the organ. In order that such excitation should be as effective as possible it is probably best to send the stimulating shocks through the whole heart, so that the auricles may come directly under their influence as well as the ventricles. In order to do this in man one electrode should be applied in front over the area of cardiac impulse, and the other over the region of the fourth dorsal vertebra behind, so that the induction shocks may traverse the organ. The electrodes should be of considerable extent (for example, large sponge electrodes), and they and the skin should be well moistened with salt solution. The shocks employed should be strong, sufficient to excite powerful contraction in the voluntary muscles.

Such a method, it seems to me, is the only rational and effective one for stimulating by direct means the action of a heart which has been suddenly enfeebled or arrested in diastole by causes of a temporary and transient character. Of course, at the same time the expedient of artificial respiration must by no means be neglected, but, on the contrary, most sedulously attended to.