With regard to the cholecystectomy, I found it a much simpler operation than I had expected, and as the patient seems to do quite well without her gall-bladder, I should not hesitate to perform it again in any case in which the bladder was much damaged, or in which it was impossible to properly suture it into the external wound. Looking to the tedious convalescence in the cases in which the gall-bladder is sewn into the parietal wound, and to the possibility of re-formation of stones, I think the question of cholecystectomy *versus* cholecystotomy as a rule of practice will have to be seriously considered.

Watching these various cases, and the nature of the fluids contained in the distended bladder and discharged during healing, I cannot help having doubts as to whether physiologists are right in regarding the gall-bladder as a storeroom for bile; it seems to me that it is very likely intended to secrete a mucous fluid to mix with the bile, and render its passage through the duct more easy; the fact that bile finds its way into it and through it when it is cut open does not militate against this suggestion, because any slight obstruction or closure of the outlet of the common duct would naturally cause the bile to flow back along the cystic duct and in the direction of least resistance when the bladder is open, and when it is closed it must act somewhat like one of the little rubber bottles with which we are all familiar, and squirt out fluid whenever its muscular coat contracts, and suck it in when it relaxes. Whatever its function, it is to many a source of infinite pain, and trouble, and danger, and if it is found that they get on as well without it, so much the better for their future peace. I do not wish it to be thought that I am advocating the performance of cholecystectomy, and leaving the stones, as a rule of practice; I merely did in this particular case what seemed to me at the time to be best, and the sequel proved that I did right.

The only remark I have to make about the fifth case, the simple cholecystotomy, is that the breaking down of the wound from the passage of the bile over it, and the slow closure and great discomfort suffered by the patient from the large quantity of discharge soaking through everything, made me often think whether it would not have been better for her if I had removed her gall-bladder. However, she has got her gall-bladder, and the wound has healed, and she is as well as her companion in misfortune who is without her gall-bladder. Since this was written she has been to see me, complaining of a good deal of pain about the site of the suture of the gall-bladder to the parietes, but I can detect nothing wrong, and think it must arise from the drag on the gall-bladder.

CARDIAC FAILURE AND SUDDEN DEATH.

By JOHN A. McWILLIAM, M.D.,
Professor of the Institutes of Medicine in the University of Aberdeen.

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.

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 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 delirium quite apart from the operation of any recognisable irritant or immediate disturbing agency. It is thus 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

[1] *Zeitschrift f. Nat. Medicin*, 1850, vol. ix.
[2] *Sitzungsberichte d. Berliner Academie*, 1884.

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 that 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, in 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 unintelligible, 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 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 tissues.

In the great majority of cases where sudden death is caused by

[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.

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 standstill by powerful inhibitory impulses transmitted along the vagus nerves. But even in such instances it is very possible that fibrillar contraction 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.

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, 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 anastomising 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 determined or ensured 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]

AN ANOMALOUS FORM OF ECZEMA.[1]

BY E. D. MAPOTHER, M.D.,
Ex-President R.C.S.I.; Consulting Surgeon to St. Vincent's and the Children's Hospitals, Dublin.

LAST January I was consulted about a raw surface involving the right tragus, and the hairless skin in front of it, and at once its likeness to Paget's disease of the mammary areola struck me. It was oval, about 1½ inch vertically and an inch transversely, florid and moist as is the glans penis during balanitis. The patient was a married woman of 40, long troubled with uterine maladies. Referring to my notes, I found records of two very similar cases, unilateral and in females, but of diverse ages—45 and 12. In the latter there was also a small patch a little above the eyebrow. The uniform, florid, oozing surface without granulations, hard and slightly raised, but without the rolled over edge of rodent ulcer, without pain or much itching, stubbornness to treatment, without occasional disappearance, characterised all the cases. Dr. Crocker has observed a like condition on the scrotum.

Some physiological analogies group these regions: in all the sebaceous glands are very large; those round the nipple were, by Bidloo in 1685, described as supplementary mammary glands The soakage of the skin by the overflow of milk or by the saliva. of the infant may be prevented by their oily secretion, and in the scrotum and parotid region a similar waterproofing protection may be afforded against the urine and the sweat falling from the temple respectively. All agree as regions in which the arrival of puberty is manifested. In the female the scanty hair in front of the ear remains of the lanugo kind, and the glands are correspondingly large. The raw surfaces occasionally noticed round the lips, the nostrils, the eyelids, and round the anus are somewhat like, but they are rarely so chronic as Paget's disease, or the condition I note in the parotid region. All the cases healed, a slightly depressed unpigmented cicatrix remaining. Some forms of eczema undoubtedly leave scars, reaching well into the papillary layer. In 1879 Professor McCall Anderson saw with me a girl aged 4, who had somewhat similar raw eczematous patches on one cheek, and on the flexures of both knees and one elbow and ankle. The oozing was excessive, and did not cease for six years after, when rickets of the spine appeared, due probably to the saline flux. Pale, somewhat concave scars endure.

It may be mentioned that Paget's disease of the mammary areola appears to be rare out of England; so say Scotch and American observers; and except a case which I observed in St. Vincent's Hospital, I can find no record of an instance in Dublin. My patient was an Englishwoman. It may be that there is a peculiar microbe which has not been freely imported, nor has found a fit soil. The curative effect of that greatest of parasiticides, mercury, in the cases here noted, supports that probability, as also does the apparent usefulness of exclusion of air. All forms of eczema on the face are obstinate, because of the unavoidable

[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.

[1] Read in the Section of Pathology at the Annual Meeting of the British Medical Association held at Glasgow, August, 1888.