SOME APPLICATIONS OF PHYSIOLOGY TO MEDICINE.

II.—VENTRICULAR FIBRILLATION AND SUDDEN DEATH.

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(From the Physiological Laboratory.)

(Concluded from page 219.)

Sudden and Unexpected Death during Rest.

THE difficulty of explanation of such deaths has long been felt, in the absence of recognized conditions (effort and excitement) tending to make an extra call on the heart and of such powerful afferent excitation as might be assumed to be provocative of reflex inhibition of intensity and duration sufficient to be fatal. Recourse has constantly been had to the verdict of "failure of the heart's action," though the sudden collapse in cardiac efficiency remains unaccounted for, *post-mortem* examination often affording no explanation of the abrupt ending of life. Sir Clifford Allbutt,²⁷ while suggesting vagus inhibition as a mode of death during anginal attacks, writes with regard to the class of deaths now under consideration—during conditions of rest, apart from anginal attacks, and where no exciting cause is apparent:

"But the riddle, which I have done so little to read, is the frequent suddenness of death in one who, having scarcely known illness, expires under no extraordinary effort; or in the peace of his own bed or chair passes silently away. The reading of this riddle is not yet."

In this relationship the facts that have been stated with regard to the more or less remote effects of experimental interference with the coronary blood supply by ligation of a branch are obviously of profound significance, showing as they do that ordinary life can go on for prolonged periods (months), either with or without signs of cardiac disturbance, until a sudden ending comes by fibrillation, sometimes during muscular effort, but often apart from this, in the absence of recognizable exciting cause or in presence of causes too trivial to have any effect under ordinary conditions. And, apart from sudden obstruction, there is no reason to doubt that a gradual interference with the blood supply as a result of coronary disease can, by damaging the nutrition and altering the properties of the muscle, lead to an abnormal susceptibility to fibrillation.

The applicability to man of these results is naturally easy in view of the widespread tendency to serious impoverishment of the blood supply (local or general) of the cardiac muscle in the later half of life, the period when deaths of the class under consideration usually occur. In the light of these facts we have a rational basis for many unexplained disasters—fatal events that otherwise remain shrouded in mystery.

While the effects of limitation of the blood supply are proved by abundant and convincing evidence, other agencies, such as perverted nutrition, toxic influences, generative changes, etc., can be effective causes. Interference with normal functioning of the Purkinje fibres on the inside of the ventricles comes into question as well as alterations in the ordinary myocardium. Fibrillation may be determined at a certain point of time by a sudden aggravation or cumulation of the toxic condition, etc., aided, it may be, by the incidence of some disturbance of the vascular system too slight to produce any serious effects except in the specially predisposed condition.

It is to be borne in mind that the conditions disposing to and leading up to fibrillation need not pervade the whole of the ventricular musculature, but may be limited to a certain amount of that tissue, as after obstruction of a coronary branch and in other conditions; changes in restricted areas can set up fibrillation, which involves the rest of the muscle, healthy as the great bulk of it may be. It is readily intelligible that such limited changes may naturally be associated with little or no recognizable altera-

tion in the force of the ventricular beat or its general efficiency. Thus there may be little or no warning of the impending catastrophe, even at a time when the mine has been laid and only a spark is needed to precipitate the explosion.

Abnormal Cardio-vascular Variations of Obscure Origin.

Under certain conditions of cardio-vascular instability—usually occurring in association with morbid states of the arterial system—irregular tides of circulatory change, often obscure as regards their exciting causes, are sometimes recognizable; one manifestation of these is found in the extensive variations of arterial pressure as measured under similar conditions from day to day or at shorter intervals. These variations are sometimes, but not necessarily, associated with discrepancies in the sphygmomanometer readings from different limbs, depending on local causes—the presence or absence of strong contraction in the large arteries of the respective limbs. Actual rises of general arterial pressure involve cardiac changes in addition to vascular constriction, etc. It is evident that disturbances of this kind may be influential with regard to the onset of anginal attacks or of sudden death in subjects where a special predisposition exists.

The Question of Coronary Spasm.

With regard to sudden interference with coronary blood supply, apart from the rare accident of embolism and the less rare occurrence of thrombosis, there arises the question of spasmodic contraction—an old hypothesis as applied to the explanation of anginal attacks. The obscure cardio-vascular disturbances already referred to might be invoked to account for the onset of some anginal attacks during rest; the attack might be determined by antecedent unrecognized changes in blood pressure and heart action. But there are on record cases in which such attacks during a period of rest are found to be unattended by elevation of the blood pressure or recognizable changes in the heart's action. Again, there are instances among anginal subjects who have varying periods of relatively low and high pressures, where no greater tendency to angina has been found in the phases of high pressure; a notable example of this has been recorded by Sir James Mackenzie. In such cases the idea of coronary spasm has commended itself to many observers, supported by such analogies as the extreme arterial constriction of Raynaud's disease, the thickening and narrowing of the temporal artery on the same side as the pain in migraine and the occasional association of Raynaud's disease and migraine, the occurrence (sometimes in relatively youthful subjects) of transitory aphasias, hemiplegias, etc., attributed to an acute temporary anæmia or ischaemia from extreme constriction of a cerebral artery, and sometimes associated with migraine. Again, there is the so-called "abdominal angina," which has been correlated with spasmodic contraction of sclerosed mesenteric arteries. Sir William Osler,²⁸ referring to arteries in general, states that in a certain stage of sclerosis arteries are very prone to spasm—a view repeatedly urged by Professor W. Russell²⁹ and supported by Pal³⁰ and many others.

It is evident that a temporarily excessive contraction of some part of the coronary system (especially in cases where the blood supply is already reduced or minimal) would induce an ischaemic condition which might be responsible for the onset of fibrillation or of an anginal attack; in this way an apparently unprovoked paroxysm of pain during rest might be accounted for and also its equally unexplained passing off after variable periods. It might also be surmised that amyl nitrite may relax a constricted coronary as part of its general vascular effect, with relief of pain which may last after the general blood pressure has again risen to its former level. The spasm hypothesis has been subjected to searching criticism by Sir Clifford Allbutt, who among many other considerations states that amyl nitrite gives no relief in transitory hemiplegias and aphasias. There is also the occurrence of dyspnoea in coronary obstruction from sudden thrombosis or embolism (as established *post mortem*) and its absence as a necessary feature of typical angina; but a possible explanation of this difference can be suggested.

Evidence of the occasional presence of strong contraction of large arteries (brachial, etc.) in diseased conditions in man was obtained by the present writer, in conjunction with Professor G. Spencer Melvin³¹ and Dr. J. E. Kesson,³² in an investigation of blood pressure a number of years ago, and surviving sclerosed arteries from the legs of old horses were found to show extraordinarily intense contraction, causing complete obliteration of their lumen and an enormous resistance to attempts to force blood through them.

In relation to the question of arterial spasm, doubt as to the existence, at least in effective degree, of vasomotor innervation of the coronary arteries is not a consideration of decisive moment. For there is no proof that the forms of excessive contraction now under discussion—for example, in the brachial artery or the horse's leg, etc.—are vasomotor phenomena; it is more probable that they are directly dependent on morbid conditions present in the arterial muscle at the time.

It may be argued that the foregoing considerations point to the feasibility of the hypothesis of coronary spasm, in view of there being no sufficient reason to assume that the coronary vessels—specially prone as they are to sclerotic changes—should in diseased states be immune from such functional disturbances as seem to occur in other arteries. But the question is far from being closed.

Fatal and Non-Fatal Angina.

It is necessary to discriminate clearly between the separate questions of (1) the mechanism of pain production in anginal attacks, and (2) the mechanism of death occurring during or between attacks, sometimes without warning.

The striking tendency of the graver forms of angina to terminate in sudden death need not be emphasized. There is every reason to believe that, in many cases at least, the end comes by ventricular fibrillation, and that the different issue in fatal and non-fatal cases hangs on the supervention of fibrillation in the former and its absence in the latter. The development of fibrillation as a frequent and characteristic result of defective coronary blood supply, as demonstrated experimentally, has already been described; and we know, from abundant pathological evidence in man, the association of coronary and myocardial impairment with fatal angina. Further, the clinical features of many recorded anginal cases, where sudden death took place either in an attack of pain or apart from such, are very noteworthy in their similarity to those attendant on death by ventricular fibrillation. The significance of these facts taken together need not be enlarged upon.

If we accept the view, which has commended itself to many observers, that an important factor in the production of pain in angina is to be found in the heart muscle working with a defective blood supply, it becomes plain how increased demands on the organ by muscular effort or emotional stress may excite an attack by leading to a relative anaemia, the blood supply, which was sufficient during rest to ensure the absence of pain, now becoming inadequate for the muscle. Such a conception might be brought into relation with the results obtained in an ischaemic limb where, on working a muscle, acute pain is caused long before the fatigue point (as indicated by inability to raise the weight in ergograph experiments) is reached—a mechanism of pain production apparently different from that present in a muscle working to fatigue while its normal circulation is going on (MacWilliam and Webster³³).

It may be conceived that in the close and striking association of angina and sudden death we see the working of distinct but related mechanisms, based in part at least on a common underlying process, essentially similar in character but differing in intensity and in the fatal or non-fatal issues, these issues being no doubt also influenced by other conditions which affect the results of the fundamental process. A conception of this kind would include two categories of dangerous anginal conditions—one with more or less transient attacks of pain of varying grades of severity, the other with the process, common to the two categories, going further in some directions, attaining greater intensity, and culminating in ventricular fibrilla-

tion. But the pros and cons of the vexed question of pain production in angina are beyond the scope of this paper.

In connexion with the well known fact that pronounced coronary sclerosis very frequently exists without angina, it has to be borne in mind that an essential point is, not the structural change in the arterial wall, but the amount of actual defect of blood supply through the sclerosed vessels from greater or less narrowing of their channels, the presence or absence of contraction in their muscular coats, the state of the capillary field, etc. Much depends no doubt on the more or less gradual development of obstructive change, the establishment of more or less efficient collateral circulation, etc. It is known that life may go on after the gradual development of complete occlusion of one coronary while the other may be found to be very greatly reduced in calibre; also after complete blocking of a large branch. Collateral circulation obviously plays an important part; it is now well known that the coronary branches are not end arteries, as was at one time believed, but have numerous anastomotic connexions. Further, as described by Gross,³⁴ the blood vessels (*arteriae telae adiposae*) of the subepicardial fat, which increases in amount as life advances, can in some measure exercise a compensating influence, supplying a considerable amount of blood to the subjacent muscle. Belonging to this system are delicate parallel vessels accompanying (at some distance) the main coronary branches, as well as a feltwork of vessels in the fat of the auriculo-ventricular groove. Gross emphasizes the importance of a relative anaemia of the muscular walls of the right heart in old age, as bearing on failure in pneumonia, etc.; he suggests a variation of the adage that a man is "as old as his arteries" to "as old as his right coronary artery."

It is noteworthy that when the main trunks and large branches of the coronaries are the seat of pronounced sclerotic changes the intramuscular twigs and finer ramifications may remain practically unaffected. The extent and efficiency of the capillary system are obviously of prime importance.

Syncope from Ventricular Standstill due to Heart-block.

In cases of heart-block, Adams-Stokes syndrome, etc., where death occurs suddenly, it is uncertain whether simple stoppage of the ventricular beat in the state of diastolic relaxation always lasts long enough to kill by paralysis of the nerve centres, following the phases of unconsciousness and convulsive phenomena. The time needed in man for irretrievable damage of these centres by acute anaemia is not known; in the ordinary experimental animals it is relatively long—a number of minutes. Of course, there may sometimes be morbid conditions present in man which would shorten the time that circulatory arrest can be survived. But in view of the associated structural damage present in the Adams-Stokes syndrome, the possibility of the ventricular standstill terminating in fibrillation in some instances must not be overlooked, though there seems to be at present no actual evidence of this happening in man. On the other hand, it is true that a fall of blood pressure, such as accompanies ventricular standstill, exercises a restraining influence on the development of fibrillation under certain conditions; but this does not always hold good under other conditions—for example, fibrillation sometimes develops in the gravely depressed or dying heart, notwithstanding the fact of excessively low blood pressure. In any case it must be concluded that only a fraction of cases of sudden death can possibly be attributed to ventricular standstill depending on the relatively rare condition of heart-block.

Syncope during Tachycardia.

There is strong reason to believe that the fibrillation mechanism is operative in many cases of sudden death associated with ventricular tachycardia. The myocardial conditions underlying tachycardia are closely related to those on which fibrillation is dependent, and there is abundant evidence that the former may develop into the latter. The excessive rate of beat—whatever be the origin of the tachycardia—is in itself favourable to this development, since it involves shortening of the refractory period and lengthening of the conduction time. The rapidity of succession of contractions—whether arising in the ventricles

themselves or transmitted with abnormal frequency from the auricles as in auricular flutter—that the ventricles can stand without fibrillating varies much in different conditions; when the conductivity is already depressed and the conduction time long, a much lower grade of acceleration naturally suffices to establish fibrillation, as can be demonstrated experimentally.

From the work of many observers we know that in certain hearts (for instance, after coronary ligation, etc.) there is often a characteristic sequence of events illustrative of the close relations of tachycardia and fibrillation—extra-systoles, first singly, then in irregular runs, more or less continuous tachycardia, and finally fibrillation. Apart from the super-vention of fibrillation it is known that the fall of blood pressure attendant on tachycardia is compatible with life for very considerable periods; there have been recoveries after periods of excessively low blood pressure attended by unconsciousness, etc., for hours. It remains to be seen whether the fall of blood pressure is often or ever sufficient *per se* to kill, or whether the fatal issue is always determined by the occurrence of fibrillation. There are no grounds for accepting vagus inhibition as a mode of sudden death during tachycardia. The vagus is known to lose effectiveness in this condition. Auricular flutter, etc., may induce unconsciousness lasting for hours without causing death; a very small blood supply can suffice to keep the nerve centres alive, as Leonard Hill showed many years ago. The absence of fibrillation is an essential feature in the recovery from ordinary cases of fainting due to temporary vascular relaxation due to vasomotor failure or to vagal inhibition, etc.

Status Lymphaticus, Electrical Shock, Digitalis.

A possible development of the mechanism of fibrillation is worthy of consideration in connexion with the sudden and unexplained deaths of the status lymphaticus, occurring, as they often do, in the absence of any recognized causation. The features of some recorded examples would fit in with the known phenomena of fibrillation—for example, such cases as have shown an abrupt abolition of the signs of heart action while the respiratory movements persisted for some little time, in marked contrast to the order of events in death by asphyxia.

Fibrillation is one of the modes of death in electrical shock, and according to Jex-Blake³⁵ it is operative in death from lightning. There is convincing evidence—experimental and clinical—that the same mechanism is responsible for sudden death during overdosing with bodies of the digitalis series.

Sudden Death in Aortic Regurgitation.

The frequency of absolutely sudden death in this condition has long been recognized. In view of the usual coronary and myocardial involvements, the causation of the fatal issue—sometimes occurring without antecedent signs of cardiac failure—may naturally be ascribed to ventricular fibrillation. There seem to be no good grounds for the assumption of protracted vagus inhibition as an effective cause.

Reflex Cardiac Inhibition.

Reflex vagus inhibition has in the past been freely invoked to account for sudden death in many diseased conditions and even in healthy persons—for example, from a violent blow on the epigastrium, etc. On the experimental side extended investigations on a great number of healthy animals and a considerable number of diseased ones have failed to lend support to the hypothesis; it has usually been found impossible to stop the heart long enough to kill by reflex inhibition or even by strong direct stimulation of the vagus, escape of the heart or the ventricles usually occurring much too soon for death to be caused by circulatory arrest. The conclusion has been reached by different observers that the possibilities of a fatal issue in this way have, to say the least, been greatly exaggerated, and that there is no sufficient ground for assuming reflex inhibition *per se* to be a frequent or important mode of death.* In some instances

where the vagal hypothesis had met with a large measure of acceptance—for example, in cases of sudden death during an early phase of ordinary chloroform anaesthesia—the view has not proved to be tenable, since such deaths have been shown to be essentially due to an altogether different mechanism—ventricular fibrillation.

On the other hand, the possibility of increased susceptibility to vagus inhibition under certain abnormal conditions must be borne in mind. In Embley's³⁶ work on chloroform it was found that under special conditions, in dogs after a large dose of morphine, the inhalation of strong chloroform vapour may cause great slowing of the heart, fall of blood pressure, and stoppage of respiration—consequences evidently depending on excessive vagus action, and obviated or removed by exclusion of such action by section of the vagi or by atropine. But the conditions present in these experiments differ widely from those of simple chloroform anaesthesia as ordinarily conducted in man. It is well known that in dogs morphine tends to exaggerate the controlling influence exercised by the vagus centre over the heart.

In Laslett's³⁷ well known case it was clear that vagus inhibition induced repeated syncopic attacks, causing cardiac standstill of the whole heart, sometimes lasting for periods of six to eight seconds, but not long enough to cause death; atropine was found to be effective as a counteracting agent.

In this connexion certain observations by Sir Hugh Anderson, cited by Sir Clifford Allbutt,³⁸ are very noteworthy. These were on cats in which the cardiac augmentor nerves were cut, by the stellate ganglia being excised some time previously. It was found that swinging the animal in the air caused pronounced slowing of the heart—for example, from 120 down to 40 in the case of *old* cats.* (It may be remarked that such degrees of slowing were not at all dangerous, and probably did not even cause much lowering of the systolic blood pressure.) But a remarkable tendency to sudden death was observed in these animals. No evidence is stated to show whether such deaths were actually due to extraordinarily prolonged cardiac standstill, or to the super-vention of some other change—for example, ventricular fibrillation, to which cats are known to be specially prone under various conditions. So far as the available evidence goes, there is nothing to indicate that the observed slowing was more threatening to life than similar slowing as seen often in common cases of non-fatal syncope in man.

With reference to possible applications of indications afforded by such experiments to the human subject it has to be remarked that we know of no clinical condition in man where there is reason to believe that conditions at all resembling those stated above are ever present—consequences of an interruption of the various paths that traverse the stellate ganglia, loss of the nerve cells contained in them, etc.† Experimental investigation shows that the cardiac augmentor nerves are very persistent in their action, extremely resistant against drugs and various abnormal conditions, and demonstrably capable of strikingly effective action in many gravely depressed states of the cardiac muscle—in contrast to the vagus functions, which are well known to be readily diminished or cut out altogether by various chemical agencies, etc.

In many cases of common syncope vagus slowing of the heart down to 50, 40, etc., a minute is a feature, but such slowing is wholly insufficient to account for the fall of blood pressure and the loss of consciousness; there are other factors concerned. Such cases do not have a fatal issue. Some instances of this condition were described by Lewis³⁹ a few years ago in subjects of "irritable heart." Pretty extensive cardiac slowing is quite compatible with a fairly

* Sudden death during operative procedures in the thoracic cavity (thoracocentesis, etc.) seems, in the light of the work of Capps and D. D. Lewis, to depend on fall of blood pressure due to vasomotor changes rather than to reflex cardiac inhibition (*Arch. of Int. Medicine*, 1907, cxxiv, 868).

* Sir Clifford Allbutt predicated an increased potency of the vagus in old and damaged hearts. Gilbert (*Arch. of Int. Med.*, 1923, xxxi, 423) has recently found in old people a more ready response of the vagus to digital compression—an age effect, apart from pathological cause. The mechanism of digital pressure is undecided—whether it acts directly by stimulation of efferent (inhibitory) fibres or reflexly by excitation of afferent fibres. There seems to be no proof of actual danger to life in this way.

† Jonnesco's operation for angina pectoris is an example, resection of the lower cervical and the first thoracic sympathetic ganglion being done with the object of interrupting afferent paths from the heart. Jonnesco recommends the bilateral operation, regarding it as harmless; he does not seem to have recognized any such dangers as were noted in Anderson's experiments. (Jonnesco, *Bull. de l'Acad. de Méd.*, Paris, 1920, lxxx, 93; *Presse Méd.*, 1921, xxix, 193; *Ibid.*, 1922, xxx, 353.)

good blood pressure—vastly higher than what is necessary for the continuance of life. Pronounced slowing (without danger to life as a rule) is, of course, familiar in some forms of violent pain—for example, in biliary and renal colic, etc.

The conditions under which sudden death most commonly occurs—namely, muscular exertion—are not favourable to prolonged vagus inhibition, but on the contrary are associated with reduction of the normal vagus control over the heart and concomitant activity of the augmentor nerves, leading to acceleration with increased force of the beats, etc.—conditions favouring the development of fibrillation in predisposed subjects. The same holds good generally in emotional excitement, apart from the very brief standstill (or prolongation of diastole) which may be caused by sudden fright.

Vagus Inhibition succeeded by Fibrillation.

There is another possibility with reference to the effects of vagal inhibition. Experiment has shown that attacks of auricular fibrillation of varying duration sometimes develop after a period of inhibition, sometimes after one or more recommencing auricular beats have occurred. The development of fibrillation in these instances is definitely related to the occurrence of the preceding phase of inhibition induced by vagus stimulation.

Fibrillation of the ventricles has also been seen following vagus standstill, but this is a rare phenomenon as compared with the development of auricular fibrillation in similar circumstances. In the course of researches over a great many years I have obtained records of only a very few examples. Still these are enough to indicate that, in presence of undue ventricular susceptibility, fibrillation may sometimes be determined in this way in some of the manifold varieties of abnormal conditions that can occur in man. Thus vagus inhibition, much too brief to be dangerous through the standstill induced, may possibly involve a mortal issue through a succeeding fibrillation.

Fibrillation or Reflex Inhibition in Sudden Death in Anginal Subjects.

The following considerations may be cited in favour of the fibrillation view of death in angina.

1. The presence of coronary and myocardial conditions which are known in certain circumstances to predispose to fibrillation. The invariable or almost invariable occurrence of coronary lesions in cases of *fatal* angina is a matter of general agreement. Sir Clifford Allbutt has adduced a wealth of facts and considerations marshalled with his usual skill, in favour of his view of the aortic origin—as contrasted with coronary and myocardial origin—of anginal pain. But this veteran clinician at the same time recognizes that the question of a *fatal* issue to an anginal attack is essentially associated with the condition of the myocardium.

2. The recognition by numerous observers, in some anginal attacks, of acceleration of the heart's action with irregularities, extra-systoles, etc.; such are known in many conditions to herald the onset of fibrillation. Among others, Windle recorded a fatal attack of angina in which the heart rate rose from 75 to 150 and became very irregular.

3. Though some slight slowing of the heart may occur in an anginal attack, there seems to be no direct evidence of the occurrence of pronounced inhibition, such as might, if somewhat intensified, threaten a suddenly fatal issue; the degrees of inhibitory slowing observed have been far removed from determining circulatory failure or even causing any considerable fall of blood pressure. There seems to be no relation between the severity and duration of the pain and the tendency to die in the paroxysm.

4. Death often occurring at the beginning of an anginal attack or in one that is relatively slight as regards pain, etc., is probably of the same mechanism as absolutely sudden death occurring between attacks or in persons who are not subjects of angina; the considerations bearing on such deaths are probably applicable to deaths during anginal attacks.

5. In the case of death between attacks or during relatively slight pain there is no evidence of such powerful afferent excitation as might be supposed to produce cardiac inhibition of such intensity and duration as to be fatal.

Of course it would be rash to dogmatize at the present time on an exclusive application of one mechanism as being the only one operative in all instances of anginal death; it may be that one or other form is present under different conditions. But there is a strong case for fibrillation as a common mode of death in anginal subjects, whatever the precise mechanism of pain production in angina may be.

Conclusions as to Sudden and Unexpected Death of Cardiac Origin.

Rupture of the heart is a very rare accident. Simple standstill of the ventricles in complete heart-block can only be a rare cause—assuming that such standstill may sometimes kill without fibrillation as the terminal event. As has been stated, it is open to grave doubt whether reflex vagal inhibition *per se*—that is, without the supervention of fibrillation—is responsible for many deaths. Blocking of the mitral orifice by a thrombus and embolism of the pulmonary artery are known to be of very rare occurrence. (Thrombosis and embolism of the coronary arteries kill, as has been already stated, by fibrillation.)

The old idea of a heart that is working with fair efficiency abruptly “failing to contract” against excessive resistance may be set aside as untenable, in the absence of the sudden action of violent poisons, etc.

There remains the conclusion that the great majority of absolutely sudden deaths are to be ascribed to ventricular fibrillation.

Symptoms of Ventricular Fibrillation in Man and Animals.

The similarity between the group of symptoms associated with many cases of sudden death in man and those attendant on ventricular fibrillation experimentally induced in animals is indeed striking. The abrupt abolition of pulse, cardiac impulse, and heart sounds, the sudden fall of blood pressure, unconsciousness, muscular relaxation often preceded by a brief phase of rigidity or convulsive movement, dilatation of the pupils, and the continuance of slow deep respiratory movements, are identical in animals and in the human subject, while in the latter the speedy replacement of the initial intense pallor by lividity or marked cyanosis is a notable feature. The amount of colour in the face, together with the occurrence of several respirations after the collapse, have sometimes made onlookers somewhat incredulous that death has taken place. The occurrence in some cases of premonitory features such as extra-systolic irregularities, bouts of tachycardia, etc., is significant. It is important that, in the collection of evidence as to unexpected and unexplained death, special attention should be devoted to ascertaining the occurrence of the group of associated features just stated; these have been very definitely recognized in many cases of sudden death where accurate observations have been made. Some such cases have come under the direct observation of the writer.

No doubt some of these phenomena are common to certain other forms of sudden circulatory failure—for example, from heart-block, collapse induced by violent afferent impulses (blow on epigastrium, etc.), or in certain cases of auricular flutter, etc. But there are special features in some of these—for instance, as regards the behaviour of the respiratory centre, etc.

Protective and Remedial Agencies.

In animal experimentation—on cats, which are remarkably liable to ventricular fibrillation—the use of certain drugs has been found by the present writer⁴⁰ (working in conjunction with Professor Spencer Melvin and Dr. J. R. Murray) to have decidedly beneficial effects, both in the way of protection against the onset of persistent fibrillation (for example, against faradic currents one hundred times as strong as are usually effective) and—in combination with cardiac massage—as regards recovery from the actual attack. These methods are at present only applicable under experimental conditions. But they give some ground for hope that, with fuller knowledge of the conditions that influence the inception and persistence of fibrillation, much may be possible in the future as regards the warding off of such catastrophic happenings as often bring life to an unexpected

and sudden close from failure of cardiac function, occurring often in persons whose hearts are far from being worn out, but on the contrary are endowed with myocardial power amply sufficient not only for quiet existence but not infrequently for the demands of considerable bodily and mental activity.

There is reason to believe that in man, as in animals, an undue susceptibility to fibrillation is sometimes a temporary phenomenon depending on circumstances that may be more or less markedly transitory, though no doubt it is very often a persistent condition depending on abnormal changes in the ventricular musculature; in the latter case immunity from sudden death must in large measure depend on avoidance of the directly provocative causes of fibrillation in a predisposed heart, such as sudden muscular exertion, especially when accompanied by emotional stress, etc.

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Memoranda :

MEDICAL, SURGICAL, OBSTETRICAL.

AN UNUSUAL VARIETY OF "SORE THROAT."

CASES are frequently met with in which sensations of pain or discomfort in the pharynx are complained of but no cause can be found on physical examination. It has long been recognized that the styloid process may project into the tonsil even to such an extent as to form a projection in the lateral wall of the pharynx. This elongation is probably due to ossification extending down the stylo-hyoid ligament, but has been regarded as an anatomical curiosity rather than as of clinical importance. I have recently met with two cases in which this condition gave rise to symptoms which were entirely relieved by removal of a portion of the bony process.

A woman, aged 30, had for two weeks suffered discomfort and dragging sensation in the left side of the throat, especially on swallowing. There had been no previous throat symptoms. An ill defined projection of the lower pole of the left tonsil could be seen, and the patient had herself discovered this hard "knob" with her finger, and ascribed her sensations to this situation. Under a general anaesthetic an incision was made through the anterior pillar of the fauces, the bony process freed, and three-quarters of an inch removed with bone forceps. The wound was sutured and recovery uneventful.

A woman, aged 37, had had pain in the left side of the throat for three months. On examination a similar condition was found to that existing in the previous case, and here also the patient had herself discovered the hard swelling. A similar operation was performed, during which it was found that the styloid process on the right side was also elongated and palpable, but to a less degree. This was not interfered with.

In addition to these two cases, I have collected eight others from the literature. Of the total of 10 cases, 7 were females, only 3 males. The ages ranged from 23 to 55. In 7 of the cases the left side was involved, and in 2 an elongated process could be felt on both sides, but only gave rise to symptoms on one. The period during which symptoms had been noticed varied from a fortnight to ten years, with an average of three years. In all cases the symptoms were very similar, consisting of discomfort and dragging sensation in one side of the throat, most marked on swallowing and sometimes on talking. In some of the cases there was a history of previous attacks of tonsillitis. Trauma as a possible factor was present in one case, symptoms being first noticed after a violent blow on the head. Nine of the 10 cases were submitted to operation, with relief of symptoms in every case. In some of the cases the tonsil was removed in addition to the portion of the styloid process, but the results seem to have been equally good where this was not done.

Details of the various cases are given in the *Journal of*

Laryngology, 1915, pp. 116, 176, and 303; the *Laryngoscope*, November, 1915, November, 1918, and May, 1920; and in the *Journal of Ophthalmology, Otology, and Laryngology*, May, 1917.

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THE CAUSE OF "STITCH."

"STITCH" is a very common condition among adolescents; it is a very severe pain, felt generally at the costal margin on one side; it is brought on by running or riding, and ceases at once when the pace is reduced to a walk. There is another "stitch," which is felt above the clavicle on one side, in the same circumstances; this is much less common than the subcostal type.

There are two possible causes for "stitch": stretching of pleural or peritoneal adhesions, or interference with the action of the diaphragm by gas in the stomach or colon. The condition seems to me to be too frequently met with to be due to pleural adhesions, since these are not very common in young subjects.

I recently saw a boy, aged 12, who, when he ran, always got "stitch," generally below the left costal margin; he had become so much worse that he had lately been compelled to sit down after walking a few hundred yards. He had had tuberculous glands in the neck, so that the existence of pleural adhesions was quite a reasonable assumption in his case; but I was struck by finding extreme tympany in the splenic region. He was habitually constipated, as many boys of his age are. Thinking that the gas was probably in the splenic flexure I gave him a short course of grey powder, and a prolonged course of dimol. In three days he was able to walk and run without any suspicion of "stitch"—a thing he had never been able to do before. But he gets recurrences if he leaves off the dimol for more than a few days.

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FREWEN MOOR, M.D.

ACTINOMYCOSIS OF THE RIGHT ILIAC FOSSA.

THE case here reported is of interest, not only owing to its rarity, but also because it demonstrates how few symptoms may be present in such cases.

A boy, aged 12, was admitted to the Southend Victoria Hospital at the end of March with a large swelling in the right iliac fossa. He stated that a week previously he had been kicked in this region whilst playing football. Three days later he noticed a swelling, which later on became tender. He felt unwell. There had been no vomiting nor acute pain. He was not constipated. The boy was pale, thin, and looked ill; the temperature was 99.4°, and the pulse 94. There was a large fluctuant, tender swelling in the right iliac region, and the diagnosis of an abscess resulting from infection of a haematoma of the anterior abdominal wall was made. The abscess was incised and packed with flavine gauze; during the next month it healed, leaving a small sinus. The boy put on weight rapidly; the temperature, however, remained persistently high (103° at night, 99° in the morning) and was not affected by vaccine therapy.

Mr. Maingot found that a probe could be passed down the sinus into the right iliac fossa; its passage was followed by escape of offensive pus showing typical sulphur granules, which microscopically proved to be actinomycetes. For the next month intensive treatment with potassium iodide was employed; the sinuses were also excised. The temperature was quite unaffected, however, and after a month it was again considered advisable to explore the right iliac fossa, as the condition of the boy had become very serious. At the time of operation a hard mass was found in the right iliac fossa, but there was no evidence of the pus having tracked towards the kidney or diaphragm. The boy unfortunately was in such a weak condition that he died under the anaesthetic. No *post-mortem* examination could be made. It is of interest that the temperature was not affected by potassium iodide; this was possibly due to a secondary infection being present.

I am indebted to Dr. Cleveland Smith for permission to publish this case.

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