SOME APPLICATIONS OF PHYSIOLOGY TO MEDICINE.

II.—VENTRICULAR FIBRILLATION AND SUDDEN DEATH.*

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It may be permissible to recall that in the pages of this JOURNAL¹ thirty-four years ago I brought forward a new view as to the causation of sudden death by a previously unrecognized form of failure of the heart's action in man—a view fundamentally different from those entertained up to that time. In the course of a long series of experiments on the mammalian heart, sudden deaths, occurring under varied experimental conditions, were found to be invariably associated with a very definite mechanism of failure entirely different in character from what had hitherto been believed to be present in cases of sudden dissolution in man depending on cardiac failure. Little attention was given to the new view for many years. At that time the current conception of the relations of the experimental physiology of the heart to practical medicine was widely removed from what it now is, thanks very largely to the work of Sir James Mackenzie and his associates and followers; it was not then recognized that most of the disturbances that have been experimentally induced in the mammalian heart (for example, fibrillation, flutter, heart-block, extra-systoles of various types, rhythms of abnormal origin, alternation of the heart beat, etc.) have their clinical counterparts in the manifold derangements of function in diseased conditions in man.

In more recent times the view that ventricular fibrillation is a cause of sudden death in man has been² accepted by numerous observers. Sir James Mackenzie² writes of sudden death in auricular fibrillation: "It has appeared to me probable that in these cases the ventricle has passed into fibrillation as MacWilliam suggested." Again, referring to sudden death from heart failure: "The cause of sudden death is almost certainly due to the onset of an abnormal rhythm, probably ventricular fibrillation." Sir Thomas Lewis³ states that "We have now the strongest *a priori* reasons for believing that sudden and unexpected death comes to many patients in a manner suggested by MacWilliam in 1889." The causation of death in this way has also been recognized by Hering⁴ and many other observers.

Direct electrocardiographic curves indicative of ventricular fibrillation at the moment of death have been recorded, notably by an American observer, Halsey.⁵ Opportunities for the gaining of such direct evidence are naturally scanty, but the indirect evidence that has accumulated is sufficient to show that this is, in all probability, not only a common cause but the usual cause of sudden and unexpected death of cardiac origin.

Relation of Death from Fibrillation to Ordinary Myocardial Failure.

The mode of death described in this article is a failure of the heart's action essentially different from cardiac (or myocardial) failure in the sense of exhaustion of the contractile power of the cardiac muscle; a verdict in the latter sense would usually be, in cases of sudden death, fallacious. While such exhaustion of contractility is, of course, of common occurrence in disease, it is a gradual process, leading to more and more marked impairment of the pumping power necessary to maintain a good circulation and to respond to the increased demands of muscular exercise, etc. There is no ground for the assumption that a sudden loss of power can occur—that is, that muscular fibres endowed with contractility adequate for a tolerably good blood pressure and blood flow should abruptly become enfeebled or paralysed, apart, of course, from the sudden

action of violent poisons or such gross causes as asphyxia, obstruction of coronary supply, haemorrhage, etc. What really happens in the supervention of ventricular fibrillation is a misapplication of contractile energy thrown away in a turmoil of fruitless activity, a disastrous change occurring in muscle that may be already more or less limited in power, or that may, on the other hand, be possessed of vigour more than sufficient for the ordinary needs of the circulation. This often occurs in a heart showing no failure of rhythmicity, excitability, or contractility.

The inadequacy of former explanations of sudden death by failure of the heart to contract and expel its contents, asystole, etc., need hardly be emphasized; such explanations assumed a failure of rhythm or a sudden loss of muscular power for which there is no warrant. Before the experimental investigation of the mammalian heart had shown the actual mode of abrupt and complete failure of the ventricular pump by the occurrence of fibrillation, the observed phenomena of sudden death by syncope—the sudden abolition of pulsation and all other manifestations of the beating of the heart—naturally led to the belief that the end had come through a simple failure of contractility. It was not realized how extraordinarily resistant and enduring the contractile power of the heart is, even under experimental conditions of exposure, manipulation, and severe strain of various kinds.

Sudden Death and the Pathological Changes found post Mortem.

It is well known that in many cases where death is believed to have resulted from cardiac failure the heart has been found *post mortem* to present structural characters apparently little if at all removed from the normal. An elaborate study of sudden death and the pathological conditions associated with it was made by Brouardel and Benham.⁶ In this book, extending to more than 300 pages, a great deal is to be found as to numerous and varied morbid conditions and structural changes in various organs, etc., in cases where death has occurred suddenly, while it is stated that in some cases no lesion is found. Elaborate details of dead-house anatomy are presented, but no explanation is given, or indeed attempted, as to how vital function has suddenly broken down, when up to that point in many cases, in spite of pathological conditions that have often been present, the individual has been able to go about his affairs with fair or good activity of body and mind. It is obvious that such structural alterations (coronary lesions, myocardial degenerative changes, etc.) as were found after death were, up to the sudden catastrophe, quite compatible with tolerable efficiency of the functions necessary for the maintenance of a more or less active life; to determine death an abrupt change must have occurred—a process fundamentally different from such slow impairment or limitation of function as may have been present up to the final disaster.

Some Characters of Ventricular Fibrillation.

The inception of ventricular fibrillation is a sudden event, though very often preceded by more or less complex disturbances in the normal action. There is an abrupt replacement of the effective systole by a continuous turmoil of inco-ordinated activity in the muscular bundles, excessively rapid small contractions, each of short duration, coursing over the intercommunicating muscular fasciculi, so that, while some portions are contracted, others are relaxed; the result is mere oscillation or inco-ordinated quivering of the ventricular walls, with complete loss of the expulsive power normally brought to bear on the contained blood by the strong and simultaneous state of mechanical tension present in all the muscular fibres during the normal systole. The effect on the circulation resembles that of absolute stoppage of the ventricular beat with complete cessation of its muscular activity, such as may be caused by the introduction of certain poisonous agents, etc. While fibrillation is of universal occurrence, under certain conditions, in all warm-blooded animals—both mammals and birds—its tendency to persist when once established varies greatly, being much greater in the higher mammalian types. In some animals among the lower mammals (rat, rabbit, etc.), as well as in

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some birds, spontaneous recovery from fibrillation frequently occurs, but in the higher mammals, and probably in man, the condition of true fibrillation seems to be invariably fatal—in the absence of the remedial measure of cardiac “massage,” which may, in animals at least, be supplemented by the administration of certain drugs, while artificial respiration is maintained. Some instances of assumed spontaneous recovery in the higher mammals and in man are probably not cases where true fibrillation had been fully established, but a related though essentially different condition, which may easily be mistaken for true fibrillation.

The essential feature of fibrillation is the establishment of a mechanism of circulating excitation in the musculature, depending on a derangement of the normal relations of (1) the time taken for conduction of the excitation wave over the ventricular muscle, and (2) the refractory period of the individual fibres. If the conduction time is unduly prolonged, or the refractory period is relatively too short, re-excitation is apt to occur when the excitation wave reaches fibres that have already recovered sufficiently after the previous excitation to respond again; the excitation wave can then circulate through the complexly arranged intercommunicating fasciculi; after a time it becomes feebler and slower as exhaustion develops, until in a few minutes all visible movement becomes extinguished. If rhythmical compression (massage) of the ventricles is employed (while artificial respiration is kept up) the fibrillation may be maintained for prolonged periods (an hour or longer), with ultimate recovery under favourable conditions; and such a heart may show regular and vigorous action for the remainder of a long experiment extending over hours.

So long ago as 1887⁷ I described this mechanism as a peristaltic contraction wave along the complexly arranged and intercommunicating muscular bundles, in contradistinction to the normal beat.

“The peristaltic contraction travelling along such a structure as the ventricular wall must reach adjacent muscle bundles at different points of time, and since these bundles are connected with one another by anastomosing branches, the contraction would naturally be propagated from one contracting fibre to another over which the contraction wave had already passed. Hence, if the fibres are sufficiently excitable and ready to respond to contraction waves reaching them, there would evidently be a more or less rapid series of contractions in each muscular bundle in consequence of the successive contraction waves reaching that bundle from different directions along its fibres of anastomosis with other bundles. Hence the movement would tend to go on until the excitability of the muscular tissue had been lowered so that it failed to respond with a rapid series of contractions. Then there might be some isolated peristaltic contractions, such as I have often seen after the cessation of the fibrillar movement.”

These conclusions were confirmed and extended in a paper in 1918,⁸ while in the interval similar views had been advanced and supported by experimental evidence—by Mines⁹ (1913) in the case of the frog heart and by Garrey¹⁰ (1914) in the mammalian heart. Suggestive experiments had been made by Mayer¹¹ (1908) on medusa, etc.

Relations of Refractory Period and Conduction Time.

If the fundamental importance of the relation between the duration of the refractory period and the conduction time of the muscle is kept in view it is easy to understand how the mechanism of circulating excitation may come into operation under very diverse conditions affecting the ventricles. Any influence cutting down the refractory period or lengthening the conduction time disproportionately must naturally tend to favour the process of re-excitation; a combination of such changes is, of course, still more effective. Hence the development of fibrillation is witnessed at one time as an apparently “spontaneous” event in a vigorous heart manifesting signs of extreme irritability (for example, from chloroform, digitalis, etc.) or in a normal heart subjected to stimulation—excessive rapidity of excitation playing an essential part by shortening the refractory period and slowing the conduction time. At another time fibrillation appears in a heart that presents features of grave depression, diminished contraction force, loss of tone, lessened excitability, and—what is the determining factor in this case—pronounced slowing of the propagation of the excitatory wave, relatively to the duration of the refractory period; such may be seen in poisoning by potassium salts, extreme cooling, etc.; thus it is often a terminal, or approximately terminal, phase in

the dying heart. In some depressed hearts there is a decided liability to fibrillation from mechanical disturbance, external pressure on the ventricles, incising the pericardium so as to remove its support from a relaxed ventricular wall, etc. In either case, whether fibrillation is a manifestation of perverted irritability or of abnormal depression, the same explanation of disturbed relationship between the processes named holds good.

In accordance with this conception it is readily intelligible that the *absolute* values of refractory period and conduction time may undergo extensive variation without fibrillation being set up; if both of these factors vary proportionately the conditions of circulating excitation do not arise. Thus artificial cooling of the heart, stopping short of a certain extreme point (about 23° C. in the perfused cat's heart), does not induce fibrillation, there being a concurrent lengthening of refractory period and conduction time as the cooling goes on—within the limit stated.

Conversely there may be, as results of a rise of temperature, etc., a marked shortening of the refractory period without fibrillation occurring, the rate of conduction of the excitatory impulse being also accelerated. The essential dependence of the mechanism of fibrillation on the factors named makes it clear why there should be no constant or necessary relation between the incidence of fibrillation, and even great alterations in contractile force, tone, etc. Dilation of the ventricles is credited by Levy with a protective influence; this view is negated by various observed facts—among others the proneness to fibrillation seen in ventricles weakened, relaxed, and distended as a result of potassium poisoning; and conversely, the marked resistance or insusceptibility to fibrillation in ventricles that are of small volume, acting strongly and rapidly and well emptied at each beat, in sequence to a large dose of adrenaline, etc. So long as the essential relationship, described above, is not upset a heart may be profoundly influenced in many ways without fibrillating: it may beat very rapidly or very slowly, regularly or very irregularly, powerfully or feebly; it may be well emptied or imperfectly emptied at each beat—with consequent distension in the latter case; it may be very sensitive or very dull to direct stimulation, and its muscle may be lax or firm, etc.

Pseudo-Fibrillation.

Apart from the disastrous event of true fibrillation there is also to be observed under certain experimental conditions (for example, rapid artificial excitation by a series of electrical shocks, mechanical stimulation, etc.) a temporary condition in the ventricles presenting many points of resemblance to true fibrillation—a degree of inco-ordination or asynchronous contraction of the musculature, recognizable on inspection and on palpation of the ventricular substance, attended by great reduction in the range of contraction movement and very little expulsion of blood at each beat, a great fall in arterial pressure, and a failure of recognizable pulsation in the peripheral arteries, etc. It is impossible to distinguish this condition from true fibrillation by examination of the arterial pulse.

Usually this condition is soon recovered from when the artificial stimulation is discontinued, though it may persist for variable periods. It apparently differs essentially from true fibrillation in its dependence on a rapid series of excitatory impulses emanating from a stimulated area or “spontaneously” from one or more irritable foci, these impulses forcing a succession of ventricular beats at a rate incompatible with their normal characters; cessation of these excitatory impulses is speedily followed by a reversion to the more moderate rates that are compatible with the normal type of beat. The non-persistence of the abnormal condition is due to the fact that the mechanism of circulating excitation, characteristic of true fibrillation, has not been fully established. To this condition I have applied the term “pseudo-fibrillation.” It is probable that most, if not all, the alleged instances of transient fibrillation in man where recovery occurred—apart from the application of cardiac massage—were examples of pseudo-fibrillation as recognized experimentally; this would account for the features observed in man during the brief attack, and for the subsequent recovery.

Such is probably the explanation of such a case as that described by Robinson and Bredeck¹² in which there were repeated syncopal attacks simulating those of the Adams-Stokes syndrome; in one of these attacks an electrocardiographic record was obtained and showed characters apparently resembling in some measure those of ventricular fibrillation—followed by recovery. In another case F. M. Smith¹³ obtained an electrical record suggestive of fibrillation, the attack being recovered from within a minute. Hoffmann¹⁴ has also published an electrical curve of a temporary condition evidently approximating to, but not reaching, the fully developed condition of fibrillation. There is also a case reported by Kerr and Bender¹⁵ of temporary fibrillation or a closely allied condition (under quinidine therapy) where records were obtained.

The Question of Recovery from True Fibrillation.

As has been already stated, spontaneous recovery from ventricular fibrillation is very frequently seen in the hearts of the lower mammals—for example, rat—but recovery is increasingly difficult and rare in the higher forms. In the cat and rabbit, after true fibrillation has lasted for periods of considerable duration (minutes), the ventricles may, without treatment, sometimes show cessation of the fibrillation movement and “spontaneous” recovery of the power to execute weak co-ordinated beats, with more or less definite pumping out of blood from their chambers into the arteries. This recovery occurs too late as a rule, if not invariably, to provide for continuance of the life of the animal, for the collapse of the circulation has lasted too long for restoration of the vital functions. Most instances of true fibrillation do not show this sort of ventricular “recovery”; the fibrillation movement usually becomes slower and feebler and gradually flickers out in the dying muscular fibres.

In the human subject it is probable that true fibrillation is (apart from treatment by cardiac massage, etc.) usually if not invariably fatal, though it seems possible that such recovery of separate though feeble ventricular beats too late for the survival of the individual may occur—if one may put this interpretation on the phenomena of some cases that have been recorded by Sir William Osler,¹⁶ cases presenting in striking fashion the external features associated with the occurrence of fibrillation. Referring to sudden death in some anginal subjects, he writes:

“Possibly in some act combining intense emotion with muscular effort there is a rapid change, a sudden unconsciousness, a stony stare, a slight change in the facial expression, and then with two or three gasps all is over; no pulse is felt at the wrist; the respiration stops; but even when the patient is apparently dead a feeble heart impulse may be felt or faint heart sounds heard.”

In one of these cases where slowly recurring respiratory gasps occurred over some minutes, cardio-puncture with a long thin aspirator needle showed movements indicating heart beats at gradually diminishing rates—52, 44, 32 per minute—ceasing fifty minutes after collapse and forty-five minutes after the last inspiratory gasp. (It may be remarked that the phenomena here described by Osler were not such as to be suggestive of reflex vagus inhibition.)

Osler's description of such deaths may be compared with what Sir Thomas Lewis¹⁷ says of deaths from ventricular fibrillation, occurring in subjects of auricular fibrillation:

“From time to time a sudden and unexpected catastrophe happens; regarded as convalescent, the patient is sitting in bed, chatting or feeding maybe. A nurse in charge, or perhaps a neighbouring patient, hears a cry or choking sound; the patient falls back on the pillows intensely pale, there are a few gasping respirations, a little convulsive movement, and the pulseless patient, rapidly becoming livid, is still.”

Relation of the A-V Junctional Tissues to Ventricular Fibrillation.

Unlike auricular fibrillation, which, as is well known, may go on over a long term of years, ventricular fibrillation is promptly fatal, involving as it does an abrupt and complete abolition of the pumping action of the ventricles and a speedy cessation of the circulation. The effect of auricular fibrillation is to impose a more or less extensive limitation to the efficiency of the heart in two ways—by the absence of the normal action of auricular systole in completing the filling of the ventricular force-pump, and still more by the

rapid and disorderly lead given to the ventricles by the rapid and irregular excitations transmitted from the fibrillating auricles to the ventricles; the latter involves a wasteful and exhausting expenditure of the energy of the ventricular muscle, attended by relatively poor results in the way of maintaining the circulation.

Fortunately the structure and properties of the junctional tissues between auricles and ventricles are such as to afford a most important protection against transmission of the fibrillation from the auricles to the ventricles. In default of such protection the onset of auricular fibrillation would necessarily be fatal by extension to the ventricles. It is known from experiment that in the auricular and ventricular muscle, with their complexly arranged bundles of fibres, fibrillation can be propagated across an artificial isthmus, made by incision, connecting portions of the musculature, only as long as the isthmus is of sufficient breadth; in the junctional mechanism, comprising the A-V node with its special properties, and the narrow A-V bundle with its longitudinally arranged fibres terminating in the Purkinje network on the inner surface of the ventricles, the fibrillation process fails to be transmitted (as such) either from auricles to ventricles or from ventricles to auricles. The junctional mechanism, while able to conduct impulses sufficiently fast to give ventricular beats at rates much above those of the normal heart under resting conditions or in moderate exercise, is not able to transmit the vastly more rapid and characteristic series of excitations of fibrillation. It is only under certain very special conditions, where the susceptibility of the ventricles to fibrillation (at relatively slow rates of excitation) is extraordinarily great, that the writer has in rare instances seen any experimental evidence of the possibility of ventricular fibrillation being excited from the auricles by impulses passing through the A-V bundle. The possibility of such occurring under pathological conditions in man is worthy of consideration.

The Purkinje network formed in the interior of the ventricular walls by the arborizations of the fibres of the A-V bundle form a sort of distributing board over which the normal impulses descending the bundle spread swiftly so as to be delivered almost simultaneously to the myocardium at the various parts of the ventricular cavities, bringing about the normal co-ordinated contraction of the ventricular fibres at each beat. Such mode of excitation is obviously unfavourable to the development of the fibrillation mechanism, but the latter might under certain conditions be promoted by an abnormal delivery of irregular and aberrant exciting impulses, dependent on defects in the branches of the bundle or its terminal arborizations, such partially interrupted or distorted impulses impinging upon the myocardium at different points in abnormal fashion—alterations in timing or direction of the impulses as might readily be determined by morbid conditions in some parts of the conducting network. An exaggeration of these functional aberrations may easily be favoured by circumstances making an extra call on the heart, such as emotion or muscular effort, or by the succeeding phase of reaction with its increase of vagus control and other changes.

There is some clinical and pathological evidence that points to the operation of such causes, resulting in ventricular fibrillation and sudden death in the human subject, though the particular mode of causation indicated above does not seem to have been suggested. Thus Nuzum¹⁸ described a case of sudden death in a man, aged 38, who had shown no definite signs of ill health and where there were no adequate microscopic *post-mortem* findings to account for the fatal issue, apart from marked alterations present in the branches of the A-V bundle in the shape of fatty infiltration or replacement, the Purkinje fibres being beset with fatty droplets, etc.

Sapegno,¹⁹ from a study of seventy-two cases, inferred that rapid unexpected death may be due to acute or chronic lesion of the bundle fibres, the acute changes being predominant in these fibres when both they and the ventricular myocardium were affected. He cites an instance of sudden death in a girl twelve days after recovery from typhoid fever, not associated with the acute changes sometimes seen in the myocardium after typhoid, but with lipomatosis in the bundle fibres—the cell substance being largely replaced

by fat—from the point of division of the main stem to the point where the two main divisions decrease rapidly in size. Monckeberg²⁰ described two cases of sudden death associated with lesions of the Purkinje fibres. One was a diphtheria case, where no gross changes were found in the myocardium, but fatty changes were shown by staining with Scharlach R in the main stem, branches, and subdivisions; the myocardium was fat-free. In the other case (trephining of skull, etc.) there were more pronounced fatty changes in the bundle fibres than in the myocardium, though some of the Purkinje fibres seemed to have remained normal.

It need hardly be recalled that several observers have described certain abnormal features in electrical records from the heart as being indicative of defective conduction in the main branches, bundle block (Lewis and others), or in the intraventricular Purkinje network (arborization block)—bizarre ventricular complexes with notching or splintering of the deflections, prolongation of the duration of the Q R S group oscillations, inversion of the T wave, etc., in addition to a remarkably low altitude of the curves in arborization block. (Oppenheimer and Rothschild,²¹ Carter,²² Willis,²³ and others.)

Exciting Causes of Ventricular Fibrillation.

In the many observed cases where all the facts point to ventricular fibrillation as the immediate cause of sudden death the common association of muscular exertion or emotional excitement is notable. The ever-recurring reports of sudden deaths during or shortly after exertion, in persons who up to the fatal issue had been able to pursue their usual avocations, emphasize the importance of the conditions attendant on muscular effort—those involving an increased demand on the powers of the heart and more or less stress on the organ. This is brought about in various ways: by the augmentation of rate and force and irritability through the agency of the cardiac nerves (diminution or suspension of vagus control and excitation of the cardiac sympathetic augmentor fibres), increased arterial pressure presenting greater resistance to the pumping out of the ventricular contents, increased diastolic filling due to more rapid inflow from the venae cavae, etc. Similar changes attend emotional excitement, with the exception of the greatly increased venous return to the heart depending on the pumping action of the working muscles during exertion driving on the blood in the veins.

While the normal heart is not injured by such changes, and in virtue of its great reserve power easily responds to increased demands on it, a heart that is temporarily or permanently in an abnormal condition of excessive susceptibility is apt to be thrown into fibrillation.

Susceptibility to Ventricular Fibrillation.

Both in healthy and diseased animals notable differences in the ease with which fibrillation may be induced were seen under experimental conditions that were apparently similar. And a heart may sometimes be seen, under altered conditions, to pass from the susceptible condition to a stable one, which may be exceedingly resistant to the induction of fibrillation by various forms of stimulation that are usually very effective. Or a change in the opposite sense may take place; or there may be variation from one phase to another more than once in the course of a single experiment in the case of a healthy heart subjected to certain abnormal influences. On the other hand, there are many cases where the abnormal susceptibility is a persistent one associated with altered nutritive conditions, toxic agencies, etc., in the muscle.

In healthy animals, cats particularly, a great susceptibility to fibrillation may be established by the administration of chloroform; the relation of this condition to the phase of light chloroform anaesthesia has been specially worked out by Levy, most (though not the whole) of whose results are in agreement with those obtained by the present writer over a long series of years. It is after a deeper phase of chloroform anaesthesia that the lighter phase is apt to be attended by the marked susceptibility referred to.

In the latter condition fibrillation is often readily induced by stimulation of afferent nerves in various ways—resulting in reflex contraction of skeletal muscles, disturbance of

respiration, rise of blood pressure, increased rate and force of the heart with increased return of blood through the great veins, etc.—in short, the same group of changes that occur in muscular effort, and brought about in similar fashion through the instrumentality of the vagus and cardio augmentor nerves, excitation of the respiratory and vasomotor centres, and the mechanical action of the skeletal muscles in propelling blood more rapidly back to the heart by the veins—increasing the rate of its diastolic filling and its output and work per minute very largely.

It is plain that it is through these changes that the sudden fibrillation of the ventricles is determined in a susceptible heart under chloroform, and there is a strong *a priori* case for the presence of a similar mechanism in the occurrence of fibrillation and sudden death (apart from chloroform) during or shortly after muscular effort—granted a condition of abnormal susceptibility in the ventricular muscle.

In addition to chloroform there are various other toxic agencies capable of establishing the hypersensitive state. Referring to drugs, it is well known from experimental evidence (adduced by Cushny and others) that bodies of the digitalis series have a powerful influence in this direction—as also have barium salts, etc.—and when pushed to extremity kill by causing fibrillation. A whole series of chemical substances might be cited as having well defined effects in this direction. And some abnormal metabolic products may well exercise a similar influence in this respect on the cardiac muscle. The development of the hypersensitive condition is not necessarily attended by any recognizable structural alteration. A prominent part in the setting up of abnormal susceptibility to fibrillation must be assigned to defective coronary blood supply.

Some Effects of Experimental Coronary Obstruction.

It is unnecessary to recall in detail the long series of experimental investigations at the hands of many workers which have demonstrated the frequent occurrence of fibrillation as a result of ligation of a coronary artery or one of its larger branches. The evidence available leaves little, if any, room for doubt that death from sudden coronary obstruction in man is due to fibrillation; the clinical features of many recorded cases are very significant, taken in conjunction with the very definite facts established by experiment in animals. The reason of the difference between non-fatal and suddenly fatal coronary obstruction is usually to be found in the non-occurrence or occurrence of ventricular fibrillation in the different cases.

It has been found experimentally that coronary occlusion, if sufficient to prove fatal, may do so in more than one way: (1) it may kill rapidly (minutes) from acute ischaemia causing ventricular fibrillation, or (2) failing this, it leads to damaged nutrition with degenerative changes (anaemic necrosis, fibrosis, etc.); these changes, apart from leading in rare instances to rupture of the heart, naturally diminish the contractile efficiency in degrees varying according to the severity of the anaemia and its distribution. Recovery may occur and life be prolonged indefinitely; or death may be suddenly caused by the supervention of fibrillation. It is easy to understand how altered functional relations in the tissues of the damaged area may lend themselves under certain conditions to the decisive upset of the normal relations of refractory period and conduction time in the ventricular walls. It remains to be seen whether the tendency to fibrillation after coronary ligation is dependent mainly on the conditions induced in the Purkinje system or in the ordinary myocardium or in both of these.

The significance of some of these results of experimental physiology does not seem to have been fully realized in relation to their bearing on the human subject. In this connexion the convincing researches of W. T. Porter²⁴ (1896), Baumgarten²⁵ (1899), Miller and Matthews²⁶ (1909), and F. M. Smith²⁷ (1918) are specially relevant. Numerous observations show that in presence of the defective blood supply following ligation the abnormal conditions that develop in the anaemic areas can not only predispose and lead up—often after months—to fibrillation, either during muscular exertion or during rest, but that the abrupt onset of fatal fibrillation may come without preceding signs of cardiac failure, as tested by exercise, or without immediately premonitory evidences of cardiac disturbance in the shape of

extra-systolic irregularities, tachycardia, etc. Thus fibrillation can occur without apparent exciting cause and quite apart from the sequence commonly observed—extra-systoles, tachycardia, and fibrillation—when death suddenly comes as an early event after coronary ligation. In other cases, at a much later time, disturbances of rhythm and evidences of heart failure present themselves, increasing in intensity and eventuating in fibrillation. Or temporary irregularities may have developed at varying periods after ligation—to give place later to regular rhythm, and then after weeks or months to fibrillation and sudden death.

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(To be continued.)

NINETY-FIRST ANNUAL MEETING

OF THE

British Medical Association.

Held at Portsmouth, July, 1923.

PROCEEDINGS OF SECTIONS.

SECTION OF MEDICAL SOCIOLOGY.

Dr. H. B. BRACKENBURY, President.

PRESIDENT'S OPENING REMARKS.

THE PRESIDENT, in opening the proceedings, said that the Section had at least one exceptional feature in that, alone among the Sections of the Annual Meeting, it was attended by a very considerable proportion of people who were not members of the medical profession. The medical members welcomed these lay visitors and their contributions to the discussion, because in the case of such a subject as this much that was of value could be contributed by experts other than medical. He read apologies for inability to be present from Sir Leslie Scott, K.C., M.P., a Vice-President of the Section, and from Sir Bryan Donkin, and announced that in the course of the day Miss Evelyn Fox (honorary secretary of the Central Association for Mental Welfare) would exhibit at a neighbouring picture-house a film illustrating the education of mentally defective children; apparatus for mental tests and an exhibit of work done by mental deficients were also to be seen in the Pathological Museum.

DISCUSSION ON
MENTAL DEFICIENCY IN ITS SOCIAL ASPECTS.

INTRODUCTORY PAPERS.

I.—WILLIAM A. POTTS, M.A., M.D.,

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THE importance of the subject of mental defect is not fully realized. The number of mental defectives is large, and their problem intimately interwoven with most social difficulties. In Birmingham, with its population of about a million, the local authority is dealing with 440 defectives by maintaining them in institutions or under guardianship, while 36 more are temporarily or permanently transferred

to asylums: there are 261 on the waiting list for institutions, and 550 more are under observation. The Education Committee is dealing with 1,119 in special schools, and has 435 more waiting for examination, who have been reported as probably mentally defective. The guardians are dealing with many additional cases, some of whom are not classified as defectives at all. Further, there are a number of defective children under 7, not yet causing trouble anywhere, except possibly at home, with whom the Education Committee and the local authority will have to deal later. Nor does this complete the tale. Those responsible for the working of the Act know from the way in which fresh cases of all ages under 40 are continually cropping up that the lists are far from complete.

The number, then, is large. So far from the estimate of the investigators for the Royal Commission on the Feeble-minded in 1906, of one in every two hundred of the population, erring on the side of excess, the mistake may have been in the other direction. The American army tests, introduced when America came into the war, were instrumental in excluding many defectives. Investigation showed that many, who were useless for the army, had been carrying on satisfactorily as hewers of wood and drawers of water. As you know, a mental defective can often manage in a simple environment to which he has gradually become adapted. A change, however, may disturb his equilibrium so much that he is never able to adjust again. But it must be remembered that all mental defectives at large are a potential danger to the community, and that the potential danger increases as modern industry becomes more complicated and the opportunities for going wrong more frequent.

Before the passing of the Mental Deficiency Act it was said that the nation which first had such an Act would benefit enormously, and that nations which ignored the problem would inevitably go under. We were the first to have an Act applicable to the whole country, and not merely to a particular province, as in the State of New York. But are we making proper use of the Act? Are we not inclined to pat ourselves on the back for having passed the Act, and to say that we know it is there if we want to use it, and then forget all about it? This is the attitude of many of those responsible for our municipal government, many of whom have never even taken the trouble to inquire what mental defect is, and still labour under the illusion that mental defect is synonymous with idiocy, and can be recognized at a glance by anyone who has shown such capacity as to secure a seat on the Bench. The truth, however, is that the majority of defectives can only be detected by careful investigation, their diagnosis requiring thorough and sometimes repeated examinations by those who have made a special study.

Nothing is of more importance to this Section than the relationship of mental defect to other problems. Take venereal disease: here mental defectives constitute one of the great difficulties, for they neither protect themselves against disease, nor realize the importance of treatment, and especially of continuing it until the disease is cured. Investigations in infirmary lock wards have shown the presence of a large proportion of feeble-minded women. Not infrequently the local authority for defectives has to delay placing a feeble-minded girl in an institution for defectives till the syphilis from which she is suffering has been cured.

Chronic drunkenness, again, though not pathognomonic of mental defect, often claims as victims men and women whose judgement and self-control are so poorly developed as to constitute a form of feeble-mindedness.

Illegitimacy also is a problem that cannot be considered regardless of mental defect: 30 per cent. of defectives is a proportion often found in rescue homes where the feeble-minded are not carefully excluded, and a specially qualified medical officer appointed to enforce this regulation. Many experienced workers would welcome a thorough medical and psychological examination of the young women coming under their care, not in the narrow spirit of attempting to rake as many as possible into the mentally defective institution net, but on the broad principle that every human