A STUDY OF THE HEART MUSCLE IN HEALTH AND DISEASE.

by

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IV. THE HEART MUSCLE IN DISEASE.

V. DEGENERATIONS OF THE MYOCARDIUM.

VI. IRREGULAR ACTION OF THE HEART.

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I. INTRODUCTION.

One of the most remarkable features of Physiology is the continued contraction of the heart muscle, commencing as it does, before birth and continuing until death. At first sight it would appear impossible that the heart should continue beating for such a remarkably long time and yet when examined scientifically it is found that although the heart appears never to rest in reality it rests as much as it works, and it is only in virtue of this rest-period that it is enabled to carry out the remarkable amount of work which it accomplishes, keeping up a continuous circulation of blood in all parts of the body and varying this supply at all times according to the requirements of the individual. By means of the central nervous system the activities of the mechanism can be varied in response to the ever changing requirements either of the body as a whole or of its different parts.

Should disease invade the body how vital becomes the action of the heart muscle! Often enough the degree of toxaemia and treatment of the myocardium becomes of greater importance than the actual disease itself.

To Harvey we owe the discovery of the circulation and it is interesting to note the wonder and amazement with which he and others regarded the heart's action. Harvey says, "When first I gave my mind to vivisections, as a means of discovering the movements and uses of the heart, and sought to discover these from actual inspection, and not from the writings of others, I found the task so truly arduous, so full of difficulties, that I was almost tempted to think (with Frascatorius) that the movement of the heart was only to be comprehended by God. For I could neither rightly perceive at first when the systole and when the diastole took place, nor when and where dilatation and contraction occurred, by reason of the rapidity of the movement, which in many animals is accomplished in the twinkling of an eye, coming and going like a flask of lightning; so that the systole presented itself to me now from this point, now from that - the diastole the same; and then everything was reversed, the movements occurring as it seemed, variously and confusedly together".
(Harvey - An Anatomical Dissertation upon the Movements of the Heart. Ch.I. Canterbury. 1894)

But though Harvey found the heart-beat difficult to interpret he was not to be beaten by the difficulties/

difficulties as will be seen by the following:-

"In the pause, as in death, the heart is soft, flaccid, exhausted - lying as it were at rest. In the movement and interval in which this is accomplished, three principal circumstances are to be noted.-

(1) That the heart is erected, and rises upwards to a point, so that at this time it strikes against the breast, and the pulse is felt externally.

(2) That it is everywhere contracted, but more especially towards the sides, so that it looks narrower, relatively longer, and more drawn together.

(3) The heart being grasped in the hand is felt to become harder during its action. Now, this hardness proceeds from tension; precisely as when the forearm is grasped its tendons are perceived to become tense and risilent when the fingers are moved.

(4) It may further be observed in fishes and colder blooded animals, such as frogs, serpents, etc., that the heart when it moves, becomes of a paler colour; when quiescent, of a deeper red colour." (Ibid. Ch.II.)

He observed also "a certain obscure undulation and lateral inclination in the direction of the axis of the right ventricle, as if twisting itself slightly/

slightly in performing its work". (Ibid Ch.V.)

Harvey analysed the movements of the heart chambers as follows:- "First of all the auricle contracts, and in the course of the contraction forces the blood (which it contains in ample quantity as the head of the veins, the store-house and cistern of the blood) into the ventricle, which, being filled, the heart raises itself straightway, makes all its fibres tense, contracts the ventricles, and performs a beat, by which beat it immediately sends the blood supplied to it by the auricle into the arteries."

Thus we have an excellent description of the heart action, marvellous for its accuracy.

But the myocardium was a source of interest long before this time.

Ambrose Paré (1510-90) was interested in the heart and describes it as "the beginning of Life, the fountain of the vital spirits ..... the first living and last dying, which because it must have a natural motion of itself was made of a dense, solid and more compact substance than any other part of the body."

It is interesting to note that so long ago as the time of Ambrose Paré it should have been recorded that "the heart has a natural motion of its own".

The discovery of the circulation had almost been made by half a dozen of Harvey's predecessors, who appeared to have stood on the very brink. As Cuvier says, we are often on the edge of discovery without suspecting it. There can be little doubt that the pulmonary circulation had been recognised by the unhappy Servetus, who, with his works, was burnt as a heretic at Geneva in 1553 by Calvin.

In 1559 a pupil of Vesalius at Padua, Realdus Columbus, may be said to have suggested the existence of the circulation by inductive reasoning, but the minds of men were hardened to ingenuous speculation.

Harvey's discovery was finally due to his application of the experimental method of Archimedes and Galen to a problem of which many of the factors were already known. The reception of his discovery was generous at home, tardy and reluctant or openly hostile abroad; but it was everywhere eagerly and hotly discussed. Riolan, for example, a distinguished anatomist, and professor at the College de France, denied and derided it.

In 1829 Louis published his "Recherches Anatomiques, etc. sur la Gastro-enterite" tom I.
He was particularly interested in the effect of acute diseases on the myocardium. He likens the heart-muscle to a "wet rag" ("linge monille".)

Stokes/

Stokes (Diseases of the Heart etc. Dublin 1854) ascribes the disappearance of the heart sounds in acute diseases to softening of the cardiac muscle.

Graves (Clinical Lectures 1864, reprinted from 1848 ed.) held that the lack of heart sounds in acute diseases was due to debility of the heart and not to softening of the muscle (p.192). He appears to have been the first to advance the view that the condition of the myocardium is of greater significance than the seat and nature of a valve lesion (p.232).

As a rule greater attention has been paid to the condition of the endocardium and particularly the diagnosis and prognosis of the various bruits heard in the heart than to the actual condition of the myocardium, and the condition of the myocardium in such valvular lesions is often overlooked, whereas it is of far greater prognostic significance than the actual valve lesion. In many diseases the condition of the myocardium is of greater import than the actual disease, owing to the susceptibility to toxaemia of the heart muscle, e.g. in pneumonia. The study of the myocardium thus becomes one of the greatest interest.

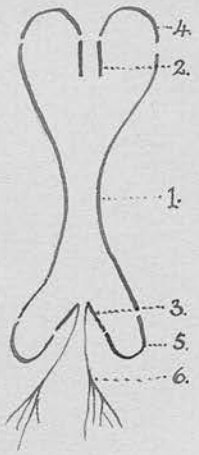
II. NORMAL HEART MUSCLE.

1. DEVELOPMENT.

In the embryo the first evidence of circulation is a rythmical contraction of the vessel walls, but eventually two primitive vessels are fused into a single propulsive organ.

In mammals the first evidence of blood vessels is found outside the body of the embryo in the wall of the yolk sac. The splanchnic mesoderm proliferates to form masses of protoplasm called "Blood-islands". These unite to form canals. Later these are also found inside the embryo either as an ingrowth from the above or as a separate development.

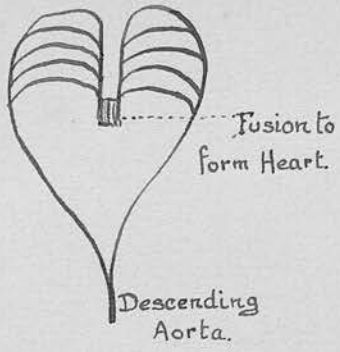
The Primitive Blood Canals are two short tubular vessels. They at once extend forwards into the extra-embryonic region and back towards the body. Each longitudinal canal is separable into six main portions:-

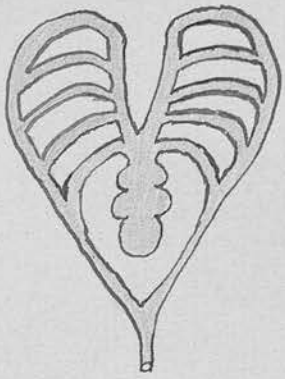


1. A dorsal section (the primitive dorsal aorta.
2. An anterior ventral section.
3. A posterior " "
4. An anterior communicating section (The first cephalic aortic arch).
5. A posterior " " " (The primitive caudal arch).
6. An allantoic artery along the allantois.

Subsequently four additional arches are formed between the anterior ventral and the dorsal sections. In this way the five cephalic aortic arches are produced and from these are developed the blood-vessels at the base of the heart, etc.

The two anterior ventral sections unite to form the heart, so that originally it is a bilateral organ. After the union however the two cavities become one which is subsequently divided.



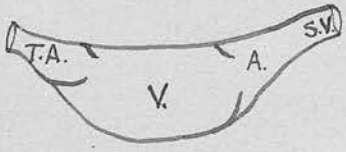


After the union of the two anterior ventral sections, one wall of the combined tube enlarges much more rapidly than the other.

Thus from a single tube is formed a definite cavity constituting the A  B 

ventricle.

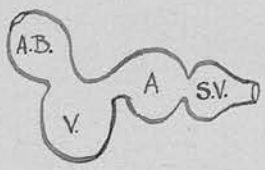
Four dilatations with intervening constrictions can be distinguished.-



T.A. Truncus Arteriosus.
V. Ventricle.
A. Auricle.
S.V. Sinus Venosus.

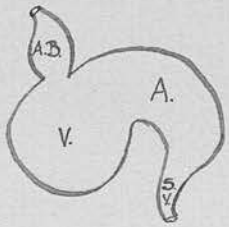
The/

The tubular heart then grows irregularly and of necessity it becomes bent upon itself.



A.B. Aortic Bulb.
V. Ventricle.
A. Auricle.
S.V. Sinus Venosus.

The bending of the heart causes the formation of a U shaped loop and the auricle eventually ascends behind the ventricle, and the ventricular opening of the auricular canal, the short communicating passage between the auricle and ventricle, is seen as a transverse slit at the upper part of the left or posterior end of the ventricle.



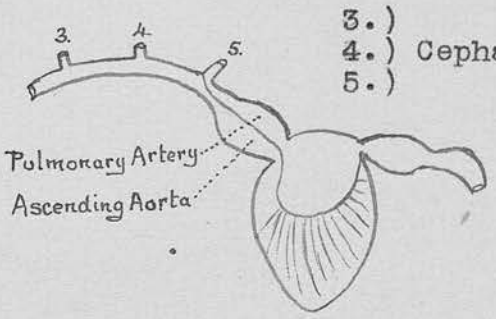
Division of the Heart into chambers.

The Heart is divided into four chambers and the Aortic Bulb into Pulmonary and Aortic portions. This is accomplished by the formation of septa and by fusion of thickened portions of the auricular canal respectively.

A Ventricular Septum grows from the wall of the ventricle and divides the Ventricle into two chambers and unites with the septum of the aortic bulb.

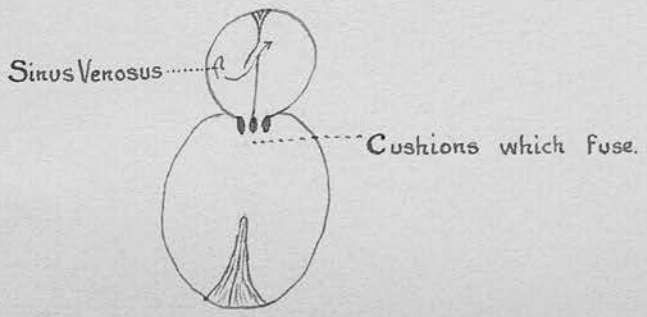
The/

The Aortic Bulb is divided by the development of two auricular cushions which develop into a septum by union and fuses with the Ventricular Septum below. The auricular septum is placed transversely, but it twists as it descends in a spiral manner.-



3.)
4.) Cephalic Aortic Arches.
5.)

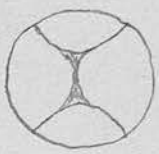
Two separate auricular septa constitute this seperation: one grows downwards and approaches the endocardial cushion in the auricular canal and for a time a small opening is left called the Ostium Primum. This is closed by the fusion of the septum with the endocardial cushions, but before this is complete an aperture appears in the upper part of the septum called the Foramen Ovale. A second auricular septum grows downwards to the right of the first septum: its lower margin passes the foramen ovale but never quite reaches the posterior wall of the auricle. The two septa only fuse after birth

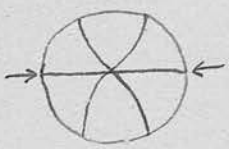


The Sinus Venosus is also divided into two parts. The venous blood is eventually all discharged into the right end of the sinus, and ultimately into the auricle as the right part of the Sinus is absorbed into it. The left part of the Sinus becomes a mere appendage of the right and is transformed into the Coronary Sinus which receives the blood from the walls of the adult heart.

Indications of the primitive separation of the auricle from the sinus venosus are still recognisable in the adult as the sulcus terminalis on the exterior, the corresponding crista terminalis in the interior and the Eustachian and Thebesian valves.

The Valves which guard the auriculo-ventricular orifices are downgrowths from the lower end of the auricular canal. At first there are four valves The septum of the bulb then descends and divides this in half, producing six. Thus the semilunar valves of the pulmonary and aortic apertures are developed.





The course of the foetal circulation differs from the adult in so far as the foetal blood is oxygenated at the placenta, not in the lungs. The main function of the right ventricle is thus quite different in the foetus where the blood is transferred from the pulmonary artery to the aorta via the ductus arteriosus, which disappears after the pulmonary circulation is established: a small amount of the blood traverses the lungs and returns to the left auricle by the pulmonary veins.

The blood which enters the right auricle from the inferior vena cava is mixed: it consists partly of purified blood from the placenta and partly of impure blood returning from the abdomen and lower extremities. This mixed blood passes through the right auricle, traverses the foramen ovale in the interauricular septum, and enters the left auricle from where it is transferred to the left ventricle through the left auriculo-ventricular opening, and the left ventricle ejects it into the aorta.

The development is not always correctly carried out, some infants die and others survive defects in development - case of May Forrest, No.40.

2. INNERVATION OF THE HEART. (Anatomical)

Below the arch of the aorta lie the superficial and the deep cardiac plexuses from which the heart receives its nerves. Through these plexuses it is connected with the Vagus Nerve, the Spinal Accessory Nerve via the Vagus nerve and the Sympathetic Nerves.

The Tenth Nerve in the neck gives off Superior and Inferior Cardiac branches. On the right side both cardiac branches pass downwards along the trachea to join the deep cardiac plexus. On the left side the superior branch passes alongside the trachea and also joins the deep cardiac plexus, but the inferior nerve accompanies the Vagus nerve over the aortic arch along with the superior cervical cardiac branch of the sympathetic and ends in the superficial cardiac plexus.

The Eleventh Nerve is composed of a spinal portion and an accessory portion. This latter sends a small branch to the ganglion of the root of the Tenth Nerve by means of which the Vagus Nerve receives cardio-inhibitory fibres.

The Sympathetic supplies the Superior Cervical Cardiac branch to the heart. This slender nerve

****** on the right side, passes down behind the large vessels to join the deep cardiac plexus. On the left side the course of the nerve is different: it passes between the left carotid and subclavian arteries, and over the aortic arch, to join the inferior cervical cardiac branch of the Vagus in the formation of the superficial cardiac plexus. In their course both nerves form connections with other cervical cardiac nerves of the sympathetic, and with cardiac and other branches of the Vagus.

From the cardiac plexuses many of the nerves enter the auricles and anastomose in the sub-epicardial tissue, forming a plexus connecting up a quantity of ganglion cells, these are especially found at the terminations of the inferior vena cava and the pulmonary veins. From this sub-epicardial auricular plexus nerve filaments with nerve ganglion cells, pass into the auricular myocardium.

From the cardiac plexuses nerve fibres pass along with the coronary arteries to the ventricles. On these, ganglion cells are present below the auriculo-ventricular sulcus.

The nerves leading from the ganglionated cardiac plexuses are non-medullated fibres. They form around the fibres of heart muscle fine plexuses and terminate either on the surface of the muscle-fibres in fine fibrils or in nodulated ends which are in contact with the muscle cells.

3. VASCULAR SUPPLY OF THE HEART.

The Coronary Arteries supply the myocardium with blood. The endocardium and valves have no blood vessels. The capillaries form a close anastomosis around the muscle fibres.

The Right Coronary Artery starts from the anterior sinus of Valsalva and divides into the small Transverse and the larger Interventricular branches. The former anastomoses with the transverse branch of the left coronary artery, and the latter supplies both ventricles and anastomoses at the apex of the heart with the interventricular branch of the left coronary.

The Left Coronary Artery starts from the left posterior sinus of Valsalva and also divides into Transverse and Interventricular branches. The former supplies the left auricle and left margin of the heart and the posterior part of the lower surface of the left ventricle and anastomoses as described. The Interventricular branch passes along the anterior interventricular sulcus to the apex of the heart. It supplies both ventricles and anastomoses as described.

The/

The Coronary Sinus receives the majority of the veins of the heart and lies between the auricle and ventricle in the auriculo-ventricular sulcus. The coronary sinus opens into the right auricle.

Lymphatic Vessels are distributed freely through the whole of the heart muscle, being most numerous in the sub-endocardial and sub-pericardial tissues. The lymphatic trunks travel backwards beside the pulmonary artery, perforate the pericardium and end in the glands which lie around the bifurcation of the trachea.

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4. STRUCTURE OF THE HEART.

The walls of the heart or myocardium, are enclosed between the visceral layer of the pericardium or epicardium outside and the endocardium inside. The musculature is of the greatest interest as its histological structure and its arrangement explains the method of the heart-beat.

The muscle fibres are not the same as voluntary muscle fibres, nor yet the same as involuntary fibres, but form a sort of intermediate structure peculiar to the heart. They are shorter than voluntary fibres, many being oblong cells with forked extremities which are closely joined to like processes of adjacent cells: they form a reticulum or syncytium of branching cells. The cells communicate both laterally and longitudinally, so that if any one part is stimulated the wave of contraction passes through the whole muscle. The ends of the various cells are not distinct from one another, but fine fibrils are continued into the cells both above and below. The muscle being only indifferently striated acts slowly in consequence. Minimal stimuli produce maximal contractions, so that whether cardiac muscle is stimulated maximally or/

or minimally the effect is the same. This is called the "all or none contraction".

In the child up to the end of the first year, and occasionally in the adult as well, curious fibres called the Fibres of Purkinje are found immediately below the sub-endocardial tissue. These are large cells which unite with each other at their extremities. The body of the cell consists of granular protoplasm, and in this are found one, or two, nuclei: the peripheral portion of the cell is transversely striated. These cells are an example of a permanent condition which is only transitory in other striped muscle cells.

The reticulating muscle cells are grouped in sheets and strands having a more or less characteristic and definite arrangement in different parts of the heart. It is possible to recognise different layers and bundles, but it is likely that sometimes these are artificially produced.

The outer layers of the heart take an oblique direction from right to left and the inner layers lie in an oblique direction from left to right: the intermediate fibres run transversely. Ludwig has demonstrated that whatever part of the heart is taken the inner fibres take a direction across those of the/

the outer fibres, the intermediate fibres following a corresponding change of direction.

Krehl (Abhandl. d. math-phys. Gesselsch. d. Wissensch., 1891. Bd XVII S. 346) calls the middle muscular layers of the heart the driving mechanism. These fibres run in circles and almost transversely round the ventricles. When these fibres are seperated from the rest of the musculature they form a cylinder open above and below.

The various fibrous structures of the base are all associated together forming one common fibrous membrane. This surrounds the aortic, the mitral and the tricuspid orifices, and resting upon the base of the ventricles given an excellent insertion for the muscular fibres of both ventricles and a general origin for the muscular fibres of both auricles.

Ventricular Musculature.

(A) The external layer of muscle arises from the auriculo-ventricular groove and after running obliquely down the ventricle it turns in at the apical whorl. From there the fibres ascend in the internal layer and are again inserted in the auriculo-ventricular/

ventricular fibrous septum. These hook-shaped fibres shorten the cavity of the ventricle, while the middle circular layer compresses it.

Besides those fibres which turn in at the apical whorl, others come down from the front and back side and enter the base of the posterior papillary muscle, whilst others from the back and right side end in the anterior papillary muscle. The muscle bundles which enter the papillary muscles are continued by means of chordae tendineae, to the flaps of the mitral valve and so to the fibrous ring round the mitral orifice. Thus many of the superficial fasiculi of the ventricles form simple oblique loops which commence externally at the fibrous rings round the right and left auriculo-ventricular openings, and end internally by being attached to the ring round the mitral valve.

(B) The Internal layers of the ventricles may be divided into (1) muscle bands common to both ventricles and (2) muscle bands special to each ventricle.

(1) The fasiculi common to both ventricles include (a) Fibres which commence above from the posterior part of the fibrous ring at the base of the right ventricle. They pass obliquely downwards as described and ascend to the front of the fibrous ring at the base of the left ventricle.

(b) Fibres from the anterior portion of the fibrous ring at the base of the right ventricle - the transverse fibres.
(c) Annular fibres which encircle both ventricles.

(2) The deep fibres special to the left ventricle are (a) V-shaped loops which commence at the fibrous ring at the base, and having descended to the apex they turn upwards in the septum and terminate by being inserted into the central fibro-cartilage.
(b) Fibres which descend from the base, enter the lower and front part of the septum, and, having passed through it assume an annular course in the posterior wall.

During the contraction of the ventricles, the papillary muscles and the columnae carneae shorten and are pressed together by the circular layer contracting on them.

The contraction of the muscular wall is materially lessened by the contraction of the papillary muscles and columnae carneae which fill up the interior of the ventricle and the force of the beat is thus economised.

The contraction of the ventricle takes place in a twisting manner. The blood is, as it were, wrung out/

out of the cavity, and with a current that naturally takes the spiral direction of the arch of the aorta.

In the right ventricle muscular bands cross the cavity and act as stays or braces and help to prevent overdistension of the cavity. The musculi papillares and chordae tendineae also help in this. The community of the musculature determines the synchronism of the two ventricles.

Auricular Musculature.

Here again there are the two groups of muscle bundles (1) Those common to both auricles and (2) those special to each auricle.

(1) The former are superficial to the latter and for the most part run transversely across the auricles. A few of them run into the interauricular septum.

(2) The latter are composed of.-

(a) looped fibres which cover the auricles from before backwards or from side to side: they are attached to the fibrous rings which surround the auriculo-ventricular orifices.

(b) Annular fibres which surround the extremities of the large vessels opening into the auricle, the auricular appendages, and the fossa ovalis.

The/

The heart is nearly always on the left side of the body: I have come across only one case, Miss T. of Currie, who suffers from Bronchiectasis, where it is on the right hand side. Dextro-cardia is usually associated with complete transposition of all the viscera but in rare cases the heart only may be abnormally placed.

5. SIZE OF THE HEART.

The heart is roughly five inches long (125 mm.) and three and a half inches broad (87 mm.): the greatest depth is two and a half inches (62 mm.). The size is variable, the volume increases rapidly at first from 22 cc. at birth to 155 cc. at the age of fifteen and to 250 cc. at the age of twenty. The increase up to the fiftieth year is very gradual, by which time it has attained the volume of 280 cc. After that age a slight decrease sets in. The volume is the same in both sexes up to the age of puberty, after that period it preponderates in the male.

6. WEIGHT OF THE HEART.

At birth the heart weight 13½ drachms (24 grms.) and its relation to body weight is as 1 to 130: in the adult the proportion is as 1 to 205. It increases rapidly in weight up to the seventh year, then slowly up to puberty, after that there is another acceleration. The weight increases very gradually up to the seventieth year.

The average weight in an adult male is eleven ounces (310 grms.) and in the female nine ounces (255 grms.).

7. CAPACITY OF THE HEART.

During life the capacity of the two ventricles is about the same and each holds about four ounces of blood. The auricles are a little less capacious. The capacity of the left ventricle appears greater than that of the right after death.

III. THE PHYSIOLOGY OF THE HEART.

1. THE PHENOMENA OF THE NORMAL HEART-BEAT.

The Neurogenic Theory. In Haller's time the beat of the heart was thought to be due to the blood passing through the heart. This theory was put an end to when it was observed that the frog's heart could be removed from the body of the frog and still continue to beat although no blood was passing through it. Some other theory had to replace the old one and it appeared evident that the cause must be within the heart itself since it could continue to beat after removal from the frog's body.

Remac (Arch. f. Anat. Physiol. U. Wisserisch. Med. 1848, S 139) examined the frog's heart and discovered groups of ganglion cells in the sinus venosus.

Ludwig then discovered ganglion cells in the interauricular septum. (Ibid. 1848 S 139)

Bidder subsequently found ganglion cells in two large masses at the junction of the auricles and ventricle (Bidder and Rosenberger, ibid. 1852. S 172)

At this time, groups of nerve cells in the respiratory/

respiratory centre were held responsible for the automatic rythm of respiration, so that it was inevitable that the ganglion cells discovered in the heart should be considered to cause the cardiac rythm.

The experiments of Stannius (Ibid. 1852 S. 85) appeared to confirm the nerve cell theory according to Rosenthal (Bemerkungen ueber die Thaetigkeit der automat. Nervencentra insbesoudere ueber die Athembewegungen", Arlanger, 1875). This experiment consisted first in tying a ligature between the sinus venosus and the auricles: when the ligature is tightened both auricles and ventricle stop, but the sinus beats as before: if then a second ligature is tied between auricles and ventricle, the ventricle beats again.

The explanation according to this was that the first Stannius ligature removed the automatic motor centre, or Remac's ganglia. While the second ligature supplied the additional stimulus necessary to excite the less active motor cells in Bidder's ganglia.

Such was the neurogenic theory of the heart beat. It was taken for granted that a motor centre was present in the sinus and that this sent out discrete impulses to the auricular and ventricular muscle.

This/

This centre was supposed to be supplemented by a less excitable motor centre between auricle and ventricle which was usually inactive, but available upon emergency for action, e.g. stimulation by the second Stannius ligature. This latter corresponded to the extraordinary centres of respiration, just as the former was supposed to correspond to the ordinary centre of respiration.

The Myogenic Theory of the Heart Beat.

The neurogenic theory began gradually to be doubted, but it was not till Gaskell's time that the theory was overthrown.

Gaskell found that sufficient pressure inside the isolated apex of the frog's ventricle is an essential element in the beat, and showed, by clamping the aorta, and in this way increasing the blood pressure within the ventricle, that the apex of the frog's heart would beat rythmically provided there were enough tension of its walls. (Gaskell - "On the Tonicity of the Heart and Blood Vessels", Journ. Physiol., Cambridge and London, 1880-82, vol. III p.51.) The apex of the frog's heart is free from nerve cells and will not beat spontaneously.

Gaskell/

Gaskell also showed that a strip of the ventricle of the tortoise could beat rythmically for hours without any external stimulus, provided always that it were kept moist and under tension. ("On the Innervation of the Heart, with special reference to the Heart of the Tortoise", Journ. Physiol., Cambridge and London, 1883, Vol.IV, p.51-56). Histological examination of the tortoise muscle showed that there were no nerve cells present and Gaskell was able to perform the experiment over and over again.

It is possible in the frog's heart to remove almost all the sino-auricular and auriculo-ventricular ganglia without upsetting the cardiac rythm in any way. Even after removal of ganglia, the heart can be cut so as to leave only a bridge of musculature: the heart will continue to beat. Also the heart beats in the embryo where there are no ganglion cells. The presence of the auriculo-ventricular bundle (hereafter elaborated) supports the myogenic theory, being an obvious channel for the propagation of the contraction.

If a normally beating frog's heart be taken and successive single stimuli applied to the ventricle more frequently than the rate at which the heart is beating,/

beating, the rythm of the heart can be reversed, so that the beat commences in the ventricle, travels to the auricles and eventually reaches the sinus. This reversal of the cardiac rythm is not compatible with the neurogenic theory. In fishes hearts also it is an easy matter to reverse the rythm.

Gaskell (Journ. Physiol., Cambridge and London, 1883, Vol. IV, p.61-81) explains the difference in rythmical power of the different parts of the heart by the morphological differences in the muscular tissue and not upon the presence or absence of ganglion cells. He holds that with the special growth of the parts of the tube from which the heart cavities are formed, is corelated a more rapid contraction of the specialised parts of the muscular walls in order to ensure a quicker emptying of the separate cavities, and thus make the heart more efficient as a pump. The development of this nearer approach to striated muscle is made at the expense of the original rythmical power, so that eventually the heart muscle becomes differentiated into muscle of varying rythmical power, according to the amount of deviation from the original embryonic rythmical muscle. The parts which remain least altered, both in the circular arrangement of their/

their fibres and in their physiological status are the large veins, the sinus and the junction of sinus and auricles, the circularly arranged fibres of the auriculo-ventricular groove and the conus arteriosus. Thus, the auricular and ventricular muscle being specially modified to act as a heart rather than a rythmically contracting tube, the unmodified parts would continue to carry the main intracardiac nerves with their accompanying ganglion cells.

If then the rythmical power of the heart depends on the amount of embryonic muscle, those parts which contain such muscle ought to be especially rythmical, quite apart from the presence or absence of ganglion cells. Thus it is found that the bulbus arteriosus contracts rythmically with great ease, either spontaneously or with just that amount of stimulus which is supplied by distension of its walls. The muscular auriculo-ventricular junction responds to stimulation with a series of contractions, not with one single contraction as does the auricular or ventricular muscle: in this way Stannius second ligature supports the myogenic theory rather than the neurogenic theory, as was supposed at first. Lastly in the large veins spontaneous rythmical contractions take place upon isolation, even in those parts/

parts which are outside the region of ganglion cells.

Englemann has shown how easily the bulbus arteriosus of the frog when isolated, will beat rythmically. (Arch. f. f. ges. Physiol., Bonn. 1882. Bd. XXIX S. 425) and this can be even better observed in the skate where the merest touch will start a series of rythmical contractions.

Englemann has shown that parts of the large veins which continue to beat normally and spontaneously after seperation, which in fact are part of the early rythmical tissue from which the heart-beat starts, are outside the region of ganglion cells, and that consequently the heart-beat starts from a non-ganglionic region of muscle tissue. (Arch. f. d. ges. Physiol., Bonn. 1896. Bd. LXV. S. 109.

Conclusion: That the beat of the heart depends on the rythmical power of the muscle tissue of the large veins and sinus being greater than the rythmical power of the other parts of the heart, and that in all cases the greater or less rythmicity of any part of the heart depends on the nature of the muscular fibre of which that part is composed, and not upon the presence or absence of ganglion cells. The sinus, as it were, sets the pace of the heart, and the ventricle responds to the stimuli reaching it, doing its work under the control of the sinus.

2. SEQUENCE OF THE HEART BEAT.

The heart may be cut up so that a bridge of muscular tissue is isolated: this bridge may then be conveniently studied. This experiment was first performed by Romanes (Phil. Trans., London. 1875, Vol. CLXVI. P. 269).

It is possible so to divide the heart that only a bridge of tissue unites the sinus with the ventricle. It is then found that the ventricle contracts after the auricle only when a wave of contraction is able to pass along this bridge.

Gaskell showed that in the tortoise if this bridge were reduced sufficiently a block ensued. If the block is slight every wave of contraction is unable to pass the blocking point and only after every two contractions of the auricle does the ventricle contract. If the block is made more severe, only every third or fourth contraction will be able to pass, so that the ventricle contracts to every third or fourth contraction of the auricle. This cannot be kept going for any length of time, but it is quite easy to keep the block small enough to allow every second wave of contraction to affect the ventricle. If the block is absolute, no contraction passes/

passes so that the ventricles remain quiet until their own independent rythm asserts itself. (Gaskell. Journ. Physiol., Cambridge and London 1883. Vol.IV, pp.61-81.)

The same experiment can be performed in the frog by clamping the auriculo-ventricular groove with a screw-clamp. If the clamp is tightened a little, the ventricle contracts only after every two beats of the auricles. If further tightened the ventricle only responds to every third or fourth beat and if tightened sufficiently it remains quiescent. (Gaskell. Phil. Trans. London, 1882, pp.999-1031.)

The muscular tissue of different parts of the heart can be blocked in the same way. If the ventricle is clamped, its apex can be made to respond to only every second contraction of its base.

So long as the continuity of the musculature is maintained the wave of contraction will pass through the block irrespective of nerves or ganglion cells. Thus no nervous mechanism is required to explain the sequence of ventricular following on auricular contraction.

The contraction wave passes most quickly over those parts of the heart which are modified for rapid contraction, e.g. auricle and ventricle, and more slowly/

slowly over the more primitive parts such as the auriculo-ventricular muscular ring or the conus arteriosus which, as has been already elaborated are more rythmical, the auricle and ventricle being correspondingly less so.

The apparent anomaly of the irregularity of the beats of the various heart's cavities during the process of dying are fully explained by the blocking principle: first of the auriculo-ventricular junction, then at the sino-auricular.

The theory of the sequence of contractions thus comes to be a rythmical series of contractions starting in the sinus and traversing the heart in peristaltic waves finishing at the bulbus arteriosus. If this theory is correctly due to the continuity of the musculature, it must be possible to pass the wave of contracture in the opposite direction and it has already been shown that this can be quite easily done.

The Mammalian heart has a sinus only while embryonic, not after birth. It is represented by some specialised tissue termed the sino-auricular node. This lies near the entrance of the superior vena cava and extends along the sulcus terminalis of the right auricle.

The/

The two ventricles are connected by a band of tissue called the Auriculo-ventricular Bundle or the Bundle of His and starts near the opening of the coronary sinus into the right auricle: the point where this bundle starts is called the auriculo-ventricular node. After traversing the top of the interventricular septum, it divides into two branches, one branch running down the one side of the septum and the other down the other side of the septum, right and left. The bundle breaks up into innumerable branches some of which go to the papillary muscles, others to the walls of the heart. The main bundles are composed of small, somewhat fusiform fibres; these are faintly striated. The cross-striation of the fibres of the terminal branches is very incomplete, part of each fibre being protoplasmic in character and are known as Purkinje's fibres.

An isolated strip of mammalian ventricle has the same inherent rythmical power as Gaskell's strip of tortoise heart. Porter ("On the Cause of the Heart-beat". Journ. Exper. Med., New York, 1897, Vol.II, p.391)

A reversal of the sequence of the beat takes place when the ventricle is made to beat at a quicker rate/

rate than the auricles and large veins. (MacWilliam. Journ. Physiol., Cambridge and London, 1883, Vol.IV, p.70.

The auriculo-ventricular junction can be blocked so that the ventricle responds only to every second or third beat of the auricle.

The mammalian heart is in all ways similar to the cold-blooded heart, except that the ventricle when isolated has a greater rythmical power of its own: thus, as would be expected, the standstill after Stannius ligature does not exist.

The sequence is the very same as in cold-blooded hearts: the impulse starts in the sino-auricular node, travels over the walls of the auricles, reaches the auriculo-ventricular node and passes along the auriculo-ventricular bundle to the ventricles.

That the auriculo-ventricular bundle is essential for the propagation of the beat is demonstrated by the blocking of the bundle in man by a gumma or otherwise, the ventricle then beats 2:1 or 3:1 or may beat entirely according to its own rythm if the bundle is completely blocked.

It is interesting to note that although the whole ventricle usually contracts as a whole, yet in-coordination of the different parts of the ventricle does/

does take place, and is known as Auricular Fibrillation, which will be discussed later under the Heart in Disease. This may be brought about by electrical stimulation, ligature of the coronary arteries, causing anaemia of the musculature, and in other ways; and it appears more than likely that fibrillar contractions are brought about by a blocking of the connections between the branching muscle cells of the ventricle itself so that a difference in conductivity takes place in consequence of which contractions travel at different rates and are blocked at different distances through the whole of the ventricular muscle.

The Time occupied in the Cardiac Cycle.

The whole cycle takes on an average 0.8 second. The cycle may be taken as commencing with the auricular systole: this lasts 0.1 second and is followed by the ventricular systole which lasts 0.3 second. It follows that for 0.4 second the heart is relaxed and at rest. Thus during normal rate the heart rests as much as it works although the ratio is altered as the rate of the pulse alters. The faster the pulse the shorter the diastole of the heart, and the slower the pulse the longer is the period of diastole.

3. THE INFLUENCE OF THE NERVOUS SYSTEM ON CARDIAC CONTRACTION.

The anatomical arrangement of the nerves having already been discussed, the physiological effect of the nerves must be shortly mentioned.

The presence of the ganglion cells must first be explained. Just as ganglia are present in the viscera in connection with the efferent nerves, so are ganglia present in the heart. The efferent nerve fibres to the cardiac ganglia come from the vagus and the augmentor nerves. The ganglia are part and parcel of the mechanism of inhibition and of the augmentor mechanism respectively and there is no reason to apply special powers to the ganglia in the heart any more than elsewhere.

The Vagus Nerve.

If the vagus nerve is divided and the peripheral end stimulated, the heart beats more slowly, and the blood-pressure gradually falls. If the stimulus is increased the heart comes to a standstill until the rythmical power of the ventricles asserts itself and they commence to beat at first feebly but soon/

soon with greater energy than before the stimulus was applied, and the blood pressure rises again to normal or even higher.

If the stimulus is prolonged the ventricles will start beating according to their own rythm, and this is known as the "vagus escape". This is a matter of the utmost import in man.

In mammals the vagus is distributed only to the auricles in which case stimulation of the peripheral end of the divided vagus nerve affects only the auricles directly, the ventricles stop owing to lack of the muscular wave from the auricles. The vagus usually affects the sino-auricular node inhibiting the impulses normally produced there, in this way bringing the whole heart to a standstill. Sometimes however it seems to act more on the auriculo-ventricular bundle so that the inhibition affects the ventricles and not the auricles.

The Sympathetic Nerves.

Stimulation of the Sympathetic in the frog or mammal accelerates the cardiac rythm and as a rule the force of the heart is increased. The effect is not instantaneous, but only commences after

after a latent period and lasts for a while after the stimulus has been removed. The latent period may be some seconds. The blood pressure may be unaffected or may be raised a little.

Cardiac Reflexes.

Impulses are constantly passing from the Medulla Oblongata down the vagus nerve to the heart, restraining the force and rate of the cardiac contractions. This tonic inhibitory action of the vagus nerve can be demonstrated by section of the nerve when the tonic action is abolished and the heart beats more rapidly. Impulses may affect the vagus nerve and so affect the tone of the heart reflexly increasing or diminishing it. The most important paths by which this is accomplished are three in number, the depressor nerve, afferent nerve fibres running in the vagus from the heart itself, and many sensory nerves.

The Depressor Nerve. This is an afferent nerve to the medulla oblongata. It starts in the walls of the aortic arch. If it is divided and the central portion stimulated, the heart is slowed and the blood-pressure falls. This is due to a reflex increasing the tone of the vagus nerve, so that if the vagi are first/

first divided stimulation of the central end of the depressor nerve produces no effect at all.

The blood pressure affects the vagus centre, viz., a rise of blood pressure increases the tonic action, probably because the stretching of the walls of the aorta stimulates the ends of the depressor nerve whose origin is in the aortic walls. In this way the blood pressure affects the pulse, or according to Marey's law, "the pulse-rate varies inversely with the blood-pressure" which does not hold good during muscular exercise or during painful stimuli.

If the volume is increased in the heart either by increased blood or saline solution into a jugular vein, the venous pressure rises and the pulse quickens. This acceleration is due mainly to the tone of the vagus being lost and also to increase of the tone of the accelerator. This is a reflex, the afferent impulses probably originate in the heart and pass to the medulla via the vagus nerve.

Pain causes reflex quickening of the heart, a phenomenon well known to most of us. This can be artificially produced by stimulating the central end of a sensory nerve. The rise of pulse rate is due mainly to diminution of tone of the vagus centre and partly to reflex stimulation of the accelerator nerves.

A/

A blow on the abdomen causes slowing of the pulse rate, and this is artificially brought about by stimulating the central end of a divided splanchnic nerve.

The brain also has some control over the vagus centre, e.g. in emotion.

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4. VOLUNTARY CONTROL OVER CARDIAC CONTRACTION.

A point of great interest here falls to be mentioned, viz. the case of Colonel Townsend, reported by Cheyne (Hill and Barnard, Journ. Physiol., Cambridge and London, 1897, Vol. XXI, p.333).

Colonel Townsend was in the last stage of some form of renal disease, and life was at its lowest ebb. Cheyne reports the experiment he witnessed along with two other medical men. "The man could die or expire when he pleased, and yet, by an effort, or somehow, he could come to life again. He composed himself on his back, and lay in a still posture some time. While I held his right hand, Dr Baynard laid his hand on his heart, and Mr Skrine held a clear looking-glass to his mouth. I found his pulse sink gradually, till at last I could not feel any, by the most exact and nice touch. Dr Baynard could not feel the least motion of his heart, nor Mr Skrine the least soil of breath on the bright mirror he held to his mouth; then each of us by turns examined his arm, heart and breath, but could not by the nicest scrutiny discover the least symptom of life in him. We began to conclude that he had indeed carried the experiment too far, and at last were satisfied that he was actually/

actually dead, and were just ready to leave him. As we were going away, we observed some motion about the body, and upon examination found his pulse and the motion of his heart gradually returning; he began to breathe gently and to speak softly."

He died the same evening.

The explanation of the case lies probably in the fact that the circulation was failing, the blood pressure very low and the vascular tone almost absent. The heart-beat was so feeble that the vis-a-tergo was not enough, even in the recumbent posture, to return the blood to the auricle without the aid of respiration.

So soon then as Colonel Townsend held his breath the circulation stopped entirely. But the heart though empty still continued feebly to beat, though this was imperceptible to the observers. Then the respiratory centre discharged a group of respirations which re-established the circulation. The man had, in fact, sunk into the condition of a hibernating animal.

In this way what was originally supposed to be a case of voluntary control can be more scientifically explained. Anyone can of course increase or diminish the work of the heart by exercise and by lying perfectly still respectively, but we cannot simulate Colonel Townsend's experience. Similarly singing, coughing, forced breaching etc. increase the frequency of the heart-beat.

5. THE EFFECT OF RESPIRATION ON THE HEART.

The pulse is normally more frequent during inspiration and less frequent during expiration. This is due to a slight diminution of the tone of the vagus during inspiration, so that the heart is allowed to beat a little more quickly. During expiration there is a negative pressure inside the chest, so that the pressure on the heart and great veins is negative: but the pressure on the jugular vein, for example, is slightly higher than atmospheric pressure. The natural result of this variation of pressure inside and outside the chest, is that blood is sucked from the outside veins to the veins inside the thorax towards the heart. The auricles tend to be dilated by the negative pressure and this assists the filling of the auricles: the thicker walled ventricles and arteries are scarcely affected at all. Thus the output of the heart is greater during inspiration, for more blood is coming to the heart, and the arterial blood-pressure is increased, also vice versa. The descent of the diaphragm tends to diminish the volume within the abdomen and blood is consequently squeezed out of it along the inferior vena cava towards the heart.

6. THE WORK OF THE HEART.

Since the ventricles have to force blood into the aorta and pulmonary veins against the pressure of their contents, the work of the heart consists in Q, the amount of blood expelled by the ventricles, and R the resistance against which the heart has to work which is approximately the mean arterial pressure. Thus:- Work done = Q x R.

The heart also expels blood with a certain velocity against the peripheral resistance. Thus Work done = $\frac{Q\ V}{g}$ Q = mass of blood expelled. V = velocity during systole. g = the force of gravity.

It is not intended to do more than mention the above as they are largely experimental.

When the arterial blood pressure rises, the heart does more work, and the output is kept constant. Increased venous blood from any cause necessitates an increased output and thus increases the work of the heart. The greater the output required, the more will the arterial pressure tend to be raised, and this increases the work of the heart still further. In muscular exercise there is both a rise of arterial blood-pressure and a greater diastolic filling of the heart. The output may then be increased by 100 cc. per beat, and since the heart is beating rapidly, the output per minute may be three or four times as great as during rest.

7. THE OUTPUT OF THE HEART.

The more a skeletal muscle is lengthened, the more forcibly it contracts: the heart muscle is stretched in accordance with the amount of contained blood. The more blood in its cavities, the greater will be the stretching of the muscle fibres, consequently the greater will be the force of contraction during systole. The amount of blood sent out from the ventricles thus depends on how full they are at the commencement of systole. Provided the arterial pressure is steady, this depends on the amount of blood entering during diastole. If the individual give deep respiratory movements, as has been explained, more blood enters the heart with each inspiration. The amount of blood may also be increased by muscular exercise which drives the blood along the veins towards the heart or saline solution may be injected into a vein so as to increase the volume of the blood: these by increasing the amount of blood admitted by the heart increase the cardiac output.

An increase in blood pressure causes the ventricles, for a few beats after the rise, to diminish/

diminish output. But since the amount of blood entering during diastole is the same, the bulk of the heart gradually increases. The distension of the muscle fibres of the heart increases the muscular power and the output rises again.

The power of the ventricular muscle to maintain the normal output in spite of the increase of blood pressure is of the greatest import in man.

By this means the heart adapts itself to sudden rises of blood pressure and muscular exercise. When the work of the heart is permanently increased, e.g. by valvular disease, or a permanently increased blood pressure, the muscle of the heart hypertrophies in the same way as skeletal muscle.

The more rapidly the heart beats, the shorter is the diastolic period and consequently less blood is admitted to the heart. Thus the output per beat is diminished. Thus while the heart is beating rapidly the output per minute is not necessarily greater than when the heart is beating slowly.

Should the flow of blood into the heart during diastole be large, the ventricles become almost fully distended early in diastole and very little blood is admitted during the latter part of diastole.
In such a case, an increase in rate will lead to a considerable/

considerable increase in output per minute though not necessarily per beat. Generally speaking, if the venous inflow is large, acceleration of the heart increases output because more blood can be discharged into the arteries in a given time.

Summary: The rate of the heart being constant, the ventricular output varies directly with the venous inflow during diastole, but is, within wide limits, unaffected by changes of arterial pressure.

If the venous inflow is small, the output per minute is independent of the heart rate. If the venous inflow is large, cardiac acceleration leads to increased output.

The variability of the power of the heart and especially its remarkable ability for increased work must surely always remain one of the most astounding of physical phenomena.

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8. INFLUENCE OF DRUGS ON CARDIAC CONTRACTIONS.

Two main groups of chemicals, viz. (A) alkaline sodium compounds and (B) acids such as lactic acid have diametrically opposite effects, as would be expected, on the cardiac contractions. The former tend to produce a tonic contraction of the ventricle or apex of the ventricle, so that less and less complete relaxation takes place between the beats, and finally the heart remains in systolic standstill. The latter produce an atonic condition of the ventricle, the contractions becoming more and more feeble, until the heart eventually stops in diastole. (Gaskell., Journ. Physiol., Cambridge and London, 1880-82, Vol. III, p.53.)

Digitaline, Antiarine and Veratrine produce a systolic standstill of the cardiac muscle; their effect on skeletal muscle being similar. In proper doses they prevent complete relaxation and thus act as muscle tonics. Pilocarpine and Muscarine on the other hand causes a diastolic standstill.

Alternate administrations of digitalin and muscarine solutions in the same preparation show all the intermediate steps between a systolic and a diastolic/

diastolic standstill. It thus appears evident that digitalin produces its effect by direct action upon the muscle substance, bringing about a tonic condition, while muscarine also acts directly on the muscle in the opposite direction, bringing about an atonic condition of the muscle. (Gaskell. Ibid.)

Gaskell showed that a strip of the auricle of the tortoise which was responding well to electrical stimuli, would contract only in the centre between the electrodes after the application of muscarine. At first, the rest of the strip quickly contracted after the central portion, later the rest of the muscle only contracted after every second contraction of the central portion and eventually not at all, the block being complete. If now a drop of atropine be applied over the strip, instantly each contraction passes the block and almost at once the whole strip is contracting as at the outset. Thus in addition to its well known action on nerve endings, Atropine appears to have some direct action on the muscle-substance. (Gaskell. Journ. Physiol. Cambridge and London 1880. Vol. III, p.14)

In the same way, potash salts and the whole group of substances containing trimethyl-ammonium to which muscarine/

muscarine belongs, all act on the heart muscle in the same way, i.e. atonically, weakening the contractions and ultimately bringing the heart to a standstill in diastole, thus opposing such substances as sodium salts, antiarine, digitalin, veratrine, and to a less extent atropine. Muscarine is often regarded as a stimulant of the vagus endings of the heart.

Atropine paralyses nerve endings and in the heart affects both the post-ganglionic and pre-ganglionic fibres of the vagus. After the administration, stimulation of the vagi has no effect. Atropine has no action on the accelerator endings. It may be used to diagnose between bradycardia due to disease of the heart muscle and bradycardia due to inhibition.

Atropine acts as muscle tonic to some extent, it breaks the connection between the muscle and the post-ganglionic fibre, and also breaks the connection between the pre-ganglionic fibre and the nerve cell.

NICOTINE AND CURARI. These drugs have no action on the muscle substance like Digitaline or Muscarine, but their action is confined to the nervous mechanism. Nictoine paralyses the connection of the pre-ganglionic fibre and the nerve cell. It at first stimulates then paralyses the vagus action on the heart, so that if it is painted on, stimulation of the vagus has no effect. Curari acts upon the motor nerve ending of striated muscle. In the snail's heart where the muscle is markedly striped curari paralyses the action of the inhibitory nerve by acting on the nerve endings. In the case of the Frog's heart and vertebrate hearts Curari acts like Nicotine on the junction of the pre-ganglionic fibre with the nerve cell, before it acts on the junction of the post-ganglionic fibre with the muscle.

THE DIGITALIS SERIES. The effect of digitalis can be very conveniently studied on the frog's heart. The heart becomes slower in rythm and contracts to smaller dimensions in systole, at the same time it does not dilate so fully in diastole. The slower rythm/

rythm can be seen to be due to the heart remaining longer contracted than normally, also the dilatation is shorter. Later the ventricular apex does not dilate during diastole or the whole ventricle dilates only once for two auricular contractions. The two halves of the ventricle may contract alternately, throwing the blood from side to side. Eventually the ventricle ceases to contract and remains in systole, with its cavity obliterated. The auricles come to a standstill in diastole since they cannot empty their contents into the ventricle.

Another action may take place in the frog's heart, the slow rythm being accompanied by imperfect systole and the heart may stop in extreme diastolic dilatation This is due to stimulation of the vagus centre in the medulla.

These two actions may be combined, or cardiac inhibition may precede the symptoms of true cardiac change.

In man the action is similar, the nerve inhibition and the muscle stimulation opposing one another.

The action of the Digitalis series may be divided into three stages. The first and third of these stages is always developed provided sufficient quantities are administered. The second stage may not/

not evince itself but is generally present in cases of poisoning.

The first stage: The rythm of the heart becomes slower. The inhibitory apparatus is called into activity and the diastole is prolonged. The ventricles contract to a smaller size, emptying themselves more completely than normally. The papillary muscles also contract more strongly than usually.

In weak or dilated hearts the period of diastole is reduced.

In normal hearts the relaxation period is increased by the inhibitory action on the vagus centre. Should the action of the digitalis and its allies be greater on the myocardium, then the relaxation is lessened, and this is more marked if the vagi are cut prior to the administration. As a rule the action on the muscle is greater than the action on the vagus centre.

The total effect is to increase the amount of blood expelled by each contraction, the amount of slowing is not great enough to counterbalance the increased output per beat.

In irregular hearts the stimulation of the vagus centre slows the rythm and lessens the irritability of/

of the heart, thus reducing the tendency to premature contractions.

In the second stage, which is really one of poisoning and is not always present, there is excessive inhibitory activity: the direct cardiac action being scarcely appreciable. The heart beats slowly and irregularly and the total result is that less blood is expelled by the heart than prior to the administration. The auricle and ventricle may beat in different rythms, the ventricles setting their own pace. This is due to the inhibitory action blocking the passage of impulses from auricle to ventricle.

In the third stage the ventricular rythm is accelerated owing to the increased irritability of the heart muscle to such an extent that the inhibitory apparatus is unable to hold it in check. It is not due to paralysis of the vagus, for stimulation of the vagus still slows the heart and causes dilatation.

Auriculo-ventricular arrhythmia becomes apparent and extrasystoles etc. make their appearance: fibrillary contractions commence, the circulation is arrested and the heart dilates.

The output of the heart is augmented during the first part of the third stage but as irregularity increases the output diminishes.

John Scott, aet. 52, whose case will be mentioned more fully later, showed an example of digitalis poisoning. He was in a desperate condition and while resident in Ward 26, R.I.E. I gave him a mixture containing mx of Tr. Digitalis four hourly. His pulse rapidly fell from 116 to 44 and became bigeminal in type. Digitalis was stopped for twenty-four hours by which time the poisoning had passed off.

The most valuable drugs of the digitalis series are Digitalis itself, Strophanthus and Squills. They have a remarkable similarity in their action on the heart. Digitalis is the most useful, and as a rule if it fails to act the other two are of little value. Not only does their action on the vagus prove useful in inhibiting very rapid or irregular contractions, but their stimulant action on the muscle increases the tone of the myocardium by preventing complete relaxation.

The Digitalis Series however includes also Apocyanum the root of Apocyanum Cannabinum official in the U.S.P. Convallaria - Lily of the Valley, also official in the U.S.P. and Euonymus, the bark of the root of the Euonymus Atropurpureus and a host of other drugs, e.g. Helleborein, Adonidin, Antiarin.

ACONITE. Acotine produces strong stimulation of the Vagus and the pulse is slowed.

Dilatation is increased in diastole and this is followed by a powerful systolic contraction. The amount of blood leaving the heart is reduced and the circulation slackened.

Mackenzie (Diseases of the Heart. 1918, p.384) states that he made a series of observations with Aconite and that no slowing of the pulse was observed.

It is a drug that is seldom if ever administered.

VERATRINE. Veratrine like Aconite stimulates the medullary centres and produces slowing of the pulse with a decrease in the output of the heart.

STRYCHNINE. The heart is not directly affected by Strychnine, though it is sometimes slightly slowed by stimulation of the inhibitory centre.

THE NITRITES. The Nitrites do not act on the heart directly, but cause a dilatation of the arteries and veins. The blood-pressure falls and the heart-rate increases.

Amyl Nitrite produces its effect in a few seconds, nitro-glycerine, erythrol tetranitrate and sodium nitrite are slower in action. They are most valuable in cases of angina pectoris. They gradually lose their effect if administered over long periods for high blood pressure.

POTASSIUM IODIDE. Potassium Iodide is frequently of advantage in senile changes in the myocardium. It is often given in cases of high blood pressure without unfortunately much effect, unless indeed there is a syphilitic origin.

OXYGEN. This is often useful in cases of cardiac asthma with cyanosis, Cheyne-Stokes respirations, angina pectoris and heart-block. Its effect is however of only temporary benefit as a rule.

BROMIDES. Where there is a mild degree of heart failure and the patient is worried, sleepless and irritable, although able to be about, the Bromides are of great service, minimising the nervousness and in this way assisting the heart. An ingenious preparation called "Sedobrol" is prepared by the de la Roche Chemical Coy. This consists of tablets of meat extract which is "salted" by means of Sodium Bromide. Dissolved in boiling water a beef tea is produced, the preparation thus combining the sedative action of the bromide with the stimulant action of the meat extract. It appears to be free from the depressing after-effects of bromide administered in the ordinary form.

HYPNOTICS. Marked sleeplessness occurring in cardiac disease should always suggest the possibility of hydrothorax and if this be found, tapping will often give relief.

In other cases Bromides, Acetanelide, Veronal and Sulphonal are of great assistance. Chloral or Opium may have to be used in cases of great restlessness unless there is oedema of the lungs. I have found Dial of great service inducing quiet sleep where a combination of Chloral and Bromide was useless.

In/

In the case of Miss White, death appeared to be somewhat unduly hastened by an injection of rather lessnthan gr.$\frac{1}{4}$ of Morphia. She refused to take anything by the mouth and suffered from delusions which greatly excited her: she required two people to keep her in bed.

Paraldehyde may be used in cases where there is evidence of a wide breech of compensation as it has the advantage of being a cardiac stimulant as well as a hypnotic. The dose used however should not be too small (three drachms) because small doses often produce excitement and restlessness.

ALCOHOL. Alcohol produces a dilatation of the arterioles. It is useful in cases of faintness and prostration where rapid effect is desirable.

CAFFEIN, STRYCHNINE, OIL OF CAMPHOR, KOLA.

These act on the nervous system, and by producing some exhileration are useful in cases of temporary exhaustion.

PITUITARY BODY. Pituitary extract increases the arterial tension by arterial contraction, and slows the heart, partly through inhibitory action and partly from direct action on the muscle. The arterial constriction does not affect the renal vessels which are dilated and the excretion of urine is increased.

ADRENALINE. Adrenaline causes constriction of the arteries of the body except those of the lungs, heart and brain. The heart's action is accelerated by stimulation of the accelerator nerve and the contractions become stronger. The effect of adrenaline is only transitory.

CONIINE AND GELSEMININE.

These act as cardiac depressants, so that the rythm becomes slower and the contractions weaker.

SPARTEIN. In moderate doses spartein acts in the same way as digitalis, but in large poisonous doses it is a cardiac depressant.

9. INFLUENCE OF CERTAIN TOXINS ON THE MYOCARDIUM.

Various toxins have a profound effect on the heart, and the influence of most of them is generally speaking similar. The patient becomes aware of certain symptoms of which tiredness, dyspnoea on exertion and palpitation are generally the first and are usually enough to warrant the assistance of a doctor. Often these are present to such a slight extent that the patient is unaware of their presence until some extra exertion puts a further strain on the heart and the symptoms are increased so that the patient becomes speedily aware of his symptoms and at once ascribes them to his occupation. In this way many cases are described as Heart Strain which are in reality due to some toxin whose action has initiated the train of events.

Some of the common poisons are, alcohol, tobacco, tea, acute specific fevers, influenza, goitre, etc.

The effects of alcohol, and indeed of all toxins, vary greatly in different individuals. I came across a man of 45 in Polmont who stated that he regularly drank a whole bottle of whisky every night and often a great deal more. This was found to be substantially correct/

correct from information obtained from the neighbours, and yet his myocardium gave him no trouble whatever: his only symptoms were gastric in origin.

Chronic alcoholics, and even more so men who indulge in drinking-bouts are prone to dilatation of the heart owing to toxaemia of the heart muscle. There is tachycardia and exhaustion of the heart. The patient becomes prostrated and shuns alcohol and the heart gradually recovers. Or dilatation becomes extreme and death takes place.

Tobacco varies very greatly in its effect on the heart as does also the type of tobacco. The Adjutant, Capt. O'K., of a battalion I was with in France came to me one day complaining that the mere slamming of a door used to make him jump in his chair and that his heart went off at such a rate that he got quite breathless for a moment or two. I told him he need not ask me about the cause of this as I knew he was smoking heavily, and on examination his average consumption proved to be over fifty Abdulla cigarettes per diem, besides an occasional pipe. He was really astonished when I hold him he was poisoning his heart. He managed to reduce his consumption and the effects passed away, much to his relief.

I have found in myself that six pipefuls of tobacco/

tobacco daily without cigarettes, produces extra-systoles though never tachycardia, provided I smoke only about four pipefuls daily I have no extra-systoles. Three pipefuls of John Cotton's "Smyrna" tobacco however will be enough to cause a few extra-systoles at the end of the first day.

Other causes of poisoning are arsenic drunk in beer, such as occurred in 1894 in Lancashire where heart failure was found to be due to heart muscle failure, microbic infections due to toxic agents produced by a microbe, e.g. the colon bacillus, the streptococcus, etc. in which case the condition can often be cured by a vaccine. Gastro-intestinal derangements such as dilatation of the stomach, gastric ulcer, etc. often cause attacks of paroxysmal tachycardia or abnormal rhythm: in such cases the heart symptoms disappear when the original trouble is satisfactorily treated.

Certain definite diseases such as exophthalmic goitre, pernicious anaemia, pulmonary tuberculosis produce marked tachycardia due to poisoning of the heart muscle and the treatment of the tachycardia in the treatment of the disease causing it, or in other words elimination of the specific toxin.

10. THE RESERVE FORCE OF THE HEART.

The Cardiac Reserve is enormous as has been shown by Starling (Law of the Heart: Linacre lecture. Cambridge 1915. Prof. E.H. Starling) and requires great forces to break it down. Even a heart with a diseased valve has an enormous reserve, provided the heart muscle is healthy.

Should the muscle not be quite healthy, then the full reserve power of the heart may be required at times, but the heart will be capable of regular daily work without exhausting the whole reserve.

If the muscle is diseased there may be little reserve power left, or there may be none, in which case the work of the heart while the body is at rest, together with the additional work entailed by the diseased valve or other deficiency, use up the whole force of the heart and the patient of necessity is confined to bed, there being no reserve left over.

Thus there are the two main elements in the work of the heart - the force required while the body is at rest and the force required when the body is at work. The reserve is held in readiness for emergency, such as is involved in a man's daily work or in special/

special strains such as rowing, cycling, etc.

Man's whole existence is built up on the reserve of the heart, by means of it we are enabled to indulge in violent exercise without causing heart failure.

But in the diseased heart the reserve is not so great and exercise may use up the whole of the reserve and still require more of which there is none available, the result is that the "rest" power of the heart is encroached upon and the danger stage has been entered.

Long before the dangerous stage there will be symptoms, imperiously demanding attention from the individual, generally commencing with breathlessness and ending in dropsy and death. (Case 20 and 29.)

The amount of reserve differs in different individuals. This I found particularly noticable when as Residents we went out to Prof. Alexis Thomson's grounds to fell trees. One found that the continual swinging of a heavy axe soon made one so breathless that one was compelled to desist, whilst another man of more muscular build would be able to continue a little longer before stopping to rest. Also that as the weeks went past I found myself able to continue longer than at first.

This is the principle of the "training" of men for/

for a boat race or other great exertion. The training increases the reserve of the heart, so that it is quite possible to increase or diminish the cardiac reserve by judicious exercise and by lack of exercise respectively.

Fortunately in this country both school and university encourage the training of the body as well as the mind, so that the average man having plenty of reserve is not afraid to exercise this and so keep his reserve efficient. On the other hand is the class who desist from exertion on the first sign of breathlessness and so their reserve remains poor as exemplified by many ladies whose main exercise consists in stepping in and out of a motor car.

The man with a valvular defect and healthy muscle will not be able to fell a tree so soon as another individual without a valve defect, because his reserve is diminished; but by judicious daily training he will surpass the healthy individual who fails to exercise his reserve. "Judicious" training certainly, for to exhaust the reserve will retard the progress and entail the necessity for longer rest periods, while exercise of the reserve increases its capacity. (Case 19.)

Thus/

Thus the response to exertion is of the utmost significance, since by that one can estimate the amount of reserve of the beart, upon which its efficiency depends. This is the functional capacity of the heart.

In disease of the heart, the aim is to find out the effect of the disease on the functional capacity of the heart.

It is with this in view that I look at valvular disease; as an indication of a possible spread of disease to the myocardium in this way involving the functional capacity of the heart, rather than as a disease in itself. (Cases 1 to 6.)

IV. THE HEART MUSCLE IN DISEASE.

1. INTRODUCTION.

I was called in to examine a youth, J.T. to-day complaining of palpitation. I sat down by the bedside to commence a series of questions. The mother, thinking I had not grasped the situation came over and said, "Will you not 'sound' the heart, doctor"? Once the stethoscope had been applied and she was assured that there was no disease of the valves, the mother was quite satisfied and considered the examination should have been at an end.

Thus it comes about that as a general practitioner one is apt easily to fall into the habit of applying the stethoscope to the chest and perhaps making a diagnosis without much further examination. The result is that should there be a murmur, the tendency is to ascribe all the symptoms to that murmur simply because it is so obvious, whereas the symptoms are due to myocarditis, and not to the valve lesion at all. Very likely the valve lesion has led up to the heart failure by the heart muscle being unable to overcome the difficulties created by the damaged valve, but as a rule the heart failure is due to coincident changes

in/

in the heart muscle.

In a case of rheumatic fever a murmur develops and endocarditis is diagnosed. The dilatation of the heart, the irregularity, etc. are regarded as the result of the endocarditis, whereas the real condition is one of dilatation due to myocardial involvement.

It is from recognising the profound importance of the condition of the myocardium in disease, and especially in "heart disease", that I am prompted to write this thesis. I recognise that in any disease there is liable to be certain degenerations of the heart muscle, and that should this take place, the various causes produce much the same result, viz., less power of work, and less reserve for strains although special features may be brought about by the involvement of special parts such as, for example, the involvement of the Bundle of His. What then are these degenerations? What the various causes? How are they to be recognised? and treated?

2. EXAMINATION OF THE HEART.

In the examination of the hearts of patients it is necessary to have a standard with which the patient's present condition may be compared. The standard I endeavour to adopt is the patient himself in normal health and the ideal is for the practitioner to have known the patient for some time while in good health. Failing this one has often to rely on the patient's statements as to his abilities while in good health. If then the patient who has been at work of any sort complains that he now cannot walk so far as formerly without some breathlessness, one can conclude that the ability of the heart to carry out the work required of it, is not so great as formerly.

One may then test the ability of the heart to work by getting the patient to bend down several times rapidly or by other more prolonged exercise tests, and noting the general effect on the patient and the effect on the heart. This is not a very satisfactory method unless one knows the effect of this exertion on the heart of the patient while in his normal health.

There is a clerk who takes the train to his office/

office and whose day's work is mainly mental. On the other hand is a railway porter or a carter whose daily work involves continuous out-door exercise. If the exercise test is applied equally to these two classes of men, both being healthy the exercise tolerance will not be identical. The clerk will experience, provided the exercise is sufficiently prolonged, breathlessness, consciousness of the heart beat, giddiness, faintness or fatigue, etc. before the carter. After the exercise aching of the limbs, tremulousness and exhaustion may be experienced. The more active class of men may pass through the same exercise test without experiencing any of these symptoms. Lewis applies the convenient term "effort syndrome" to these symptoms and signs which follow exercise in health, although the same symptoms may be found in cases of ill health. ("The Soldier's Heart and the Effort Syndrome" Thomas Lewis, page 4. Shaw & Sons, London, 1918) The phrase applies only to patients in whom no evidence of disease can be found, and they are said to suffer from the "effort syndrome" It is the border-line between health and disease: it is an inquiry into the reserves of the body. "Effort-syndrome" cases may later be found really to suffer from/

from pulmonary tuberculosis or other disease which has produced the symptoms, they are only "effort syndrome" cases before such a diagnosis is made.

Each one of us knows that the ability to continue strenuous exercise is limited. The index of ill-health is the relatively small amount of work which will produce discomfort. Physiological sensations might be regarded as abnormal, but the abnormality lies in the circumstances in which they arise, in the demonstrated lack of reserve.

Having got an idea of the patient's response to effort it is then advisable to find out the nature of his occupation, his environment and the history of past illness.

If it is found that the patient complains of one single symptom, e.g. breathlessness and after examination no other symptoms are obtainable, it may be taken for granted that there is no heart failure for there would inevitably be other symptoms.

The same applies to a single sign without abnormal phenomena.

As an example of a physical sign without heart failure may be instanced the case of Mrs Morrison (Case No.1).

She/

She came to me complaining of some muscular pain about the shoulder. It was only after a long series of questions that I found, apparently a history of acute rheumatism in youth: while questioning her about her heart she volunteered the information that she had a cardiac bruit.

When about 10 years old she had a very serious illness which the doctor called Influenza, she thinks. She was confined to bed for three months and did not return to school for six months. She also remembered getting a "very expensive medicine", but did not remember the name of it. She later wrote to her mother who told her the medicine was "Salicin". She remembers wearing a porous plaster over her heart for a year.

In April 1919 she had a Quinzy which was "very bad" and she was confined to bed for a month. "It took fourteen days to burst". Ever since childhood she has had "sore throats", and swollen tonsils "almost choked her". She had them needled, electricity applied, caustic applied, etc., etc. In June 1919 she had the tonsils removed and has been better since then.

She has two children.

On examining the heart one was instantly conscious/

conscious of a loud mitral systolic bruit not much altered by exercise, but becoming quite musical when the patient assumed the recumbent position. She has been told about it by many doctors.

Yet the heart never troubles her at all. She can walk up a hill without discomfort, can climb the Pentland Hills without dyspnoea. I made her run twice up and down a flight of stairs and there was no evidence of dyspnoea or other symptom, and the pulse rate was scarcely accelerated.

B.P. 120. Pulse 85. Urine: clear yellow. No deposit. Acid S.S. 1023, no abnormal constituents.

Her myocardium is normal and the valve lesion makes no difference to her.

Also case of Mrs C. (No.2), A.J. Black (No.3), Mrs Anderson (No.4), Henry Fowler (No.5), and H.M. Moir (No.6).

3. THE EFFECT OF A SENSITIVE NERVOUS SYSTEM ON THE HEART.

Over-sensitive individuals who are conscious of an extrasystole, or have been told they have a heart-murmur, are apt to become very alarmed about their hearts and to spend much time thinking about their symptoms which are apt consequently to be grossly exaggerated. Nervous women with exhaustion of the reserve force struggle on with their work and eventually the nervous system shares in the exhaustion and a breakdown takes place with an extreme development of the sensory phenomena. Very severe pain may be experienced across the chest and in the left arm. Such cases resemble angina pectoris, but they are not dangerous: they are evidence of exhaustion of the heart muscle. The attacks vanish when the cardiac reserve is restored.

Patients who have constant work night and day, such as is necessitated by crying children or a sick relative, lose sleep and become exhausted. They develop symptoms like angina pectoris from exhaustion of the heart muscle and nervous system. With suitable treatment the angina pectoris disappears entirely.

Removal/

Removal to the seaside, or other form of healthy open air holiday should be aimed at. Where this is impossible sleep at least must be assured by the help of another individual for night-work if available, if not, sleep must be assured with bromides even though this drug should produce languor during the day which is beneficial in so far as it induces idleness and restfulness.

Sedobrol is often useful and has no after-effects.

On the other hand there are patients who have exaggerated sensory phenomena and who have slowly progressing pathological changes taking place in the heart, and this has to be borne in mind, specially where there is a history of syphilis.

Where the myocardial condition is secondary to an over sensitive nervous system, it is essential to assure the patient that there is no organic disease, and the great mental relief experienced, once the patient is really assured of this, is the first step towards recovers. (Case of Mrs Forrest No.41). Where there is actual organic trouble patients may live in a constant state of fear, which greatly exaggerates the symptoms. It must then be pointed out that the symptoms are merely a warning-signal to prevent further exhaustion, and that with careful treatment/

treatment the suffering will largely disappear.

Extra-systoles often cause great alarm: I can well remember as a boy having an occasional extra-systole and wondering whether I would suddenly die. The patient must be reassured that they are of no import whatsoever and that they call for no diminution of work or exertion however severe.

Heart-tonics are not indicated in nervous cases, the overworked muscle requires rest rather than stimulation.

Diet is also important: tea, coffee, spirits are apt to be indulged in, instead of a simple dietary at frequent intervals.

Neurasthenic patients often have evanescent murmurs and the heart may be slightly dilated, sometimes the pulse is slow or it may be irregular, this is usually a respiratory irregularity, there may be extrasystoles. Heart failure never occurs however.

The symptoms are very varied. There may be stomach or bowel complaints such as dilatation of the stomach, visceral stasis or constipation. There may be irritability and peevish temper. Some patients are deeply concerned about their bodily or spiritual welfare.

The gynecologist diagnosis pelvic disorder.

The/

The surgeon removes the appendix and so on. The physician diagnosis cardiac, gastric, mental or renal neurasthenia.

The organic disease must first be treated, e.g. constipation; sleeplessness is avoided by means of bromides and the patient is encouraged to lead an open air life and try to forget himself by interesting himself in his duties. As a rule it is useless to tell neurasthenic patients not to think about themselves, it is better to advise active measures: to read an interesting novel, to work in the garden, paint, or otherwise remove his thoughts from himself. Golf is specially recommended as it breaks the train of thought and takes the patient's mind off himself. (Mott).

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4. THE TOXIC EFFECTS OF FEVER.

The heart is very sensitive to the effects of acute febrile attacks such as, influenza, pneumonia, typhoid fever, eyrsipelas, diphtheria, rheumatic fever and in septic infections by the specific organism. The heart may itself be the origin of the fever. Pericarditis, endocarditis or Myocarditis may be produced or all three may be involved together, only the latter of which I am concerned with here.

Everyone knows the importance of cardiac involvement in pneumonia, for example, and how a healthy young adult who would have combated the pneumonia is killed by the results of heart affection.

If the heart is auscultated during the course of a fever and a mitral regurgitant murmur is heart, it used to be concluded that an infection of the mitral valve had taken place: nothing could be more evident, it could be clearly demonstrated. After recovery of the patient however, the mitral regurgitation was often found to have entirely disappeared - the valve had fortunately recovered.

It is now recognised that in acute fevers the myocardium is profoundly affected by the toxin which destroys/

destroys the tonicity of the heart muscle causing an incompetence of the mitral orifice, and in this way producing the regurgitation which appeared to be due to endocarditis. In fact the toxin rarely infects the endocardium without infecting also the pericardium and the myocardium; and the symptoms which are often ascribed to the endo- or peri-carditis may be largely due to myocarditis: the murmur or the friction being so obvious as to lead to the symptoms being assigned accordingly, whereas the myocarditis is more liable to be overlooked. The heart sounds generally become faint, and soft murmurs appear at the mitral and tricuspid orifices. The size of the heart may be greatly increased.

Myocarditis is to be recognised by the rate and rythm of the heart. The rate is accelerated out of proportion to the increase of rate due to the rise of temperature alone. The sounds are soft and the intervals become equalised so that the heart assumes the foetal rythm.

The rythm is affected in various ways. Nervous arrythmias are generally abolished, it is interesting to note.

Ventricular beats are occasionally absent. This is evidence of damage to the auriculo-ventricular bundle/

bundle or weakness of the muscle whereby it fails to contract, no wave or a very small one only, reaching the arteries. This condition is of grave import.

Extra-systoles are rare. Cases No.7 and No.9.

Auricular fibrillation sometimes occurs and such cases rarely recover.

The profound significance of infection of the heart has only been recently recognised in cases of heart disease. Lewis refers to this at length in "The Soldier's Heart and the Effort Syndrome", page 33, see table. Not only so but the condition known as Soldiers Heart would appear to be largely due to an infection of one sort or another. The Soldiers Heart is common in civilian life and very often follows on febrile illness and is due to infection. The importance of infection as etiological factor will be recognised from Lewis' table: the remarkable similarity of the percentages will be noted.

TABLE./

Cause of Onset.	Soldiers Heart.		Heart Disease.	
Rheumatic Fever or Chorea	68		25	
P.U.O. or Influenza	28		1	
Dysentery	17		1	
Pleurisy and Bronchitis	15		0	
Pneumonia	10	33%	2	37%
Diarrhoea	10		0	
Enteric Fever.	6		1	
Other Infectious disease	27		7	
Wounds and Accidents	19		6	
Gassing	14		2	
Shell-Shock	13		2	
Under Bombardment	9	11%	0	12%
Sudden Strain	5		0	
Frost-bite and Inoculation.	3		2	
Total	558		101	

RHEUMATISM. The Rheumatic poison is the most important infection of the heart muscle and frequent observations will be made on it, but a brief account of the effects of acute rheumatic fever on the myocardium may be given here.

The heart may be infected with the Diplococcus Rheumaticus which has been isolated from the myocardium and vegitations on the valves and pericardium, so that it is evident the whole heart may be affected. In other cases the myocardial involvement is often due to its toxins. Fatty degeneration and breaking down of the muscle fibres are fairly common and cellular infiltration may take place, and the muscle may be permanently damaged. Congestion of the vessels, leucocyte infiltration and connective tissue swelling may be present.

If the toxaemia is severe, great dilatation of the heart may occur. The heart may become very weak and irritable, and these often persist for long after the absence of fever, necessitating rest which patients often find very trying. Adhesions, penetrating the myocardium may be found as the result of pericarditis. Slow cicatrisation may follow causing serious heart-trouble in after years.

The/

The auriculo-ventricular bundle is rarely involved interfering with the passage of stimuli to the ventricle.

Dilatation may be extreme. The pulse is rapid, soft, compressible and sometimes irregular.

Young people usually recover from a first attack but the middle-aged have less resistance. Syncopal attacks may prove fatal.

The significance of subsequent attacks depends on whether or not the myocardium is involved.
If the valves have been affected previously and the myocardium is not subsequently attacked, recovery is probable; but if the heart muscle is attacked the case becomes much more serious.

Most of the cases recorded have some connection with Rheumatism, e.g. Mrs Morrison - No.1, Peter Peat No.32, David Linton - No.33, Mrs David Kirkpatrick - No.31.

INFLUENZA. The far reaching effects of influenza on the heart are known to every practitioner. It may cause myocarditis, endocarditis and pericarditis: it may damage the auriculo-ventricular bundle as may also rheumatic fever,/

fever, diphtheria and septic infection (Mackenzie, p.280). Cases of Mrs Gray - No.9, Mrs C. - No.21, Miss Baird - No.22, and Mrs Stenhouse - No.23.

Myocarditis may lead to sudden death and functional disturbances are very common. Endocarditis and pericarditis are however extremely rare.

Lewis (The Soldier's Heart, p.37) describes how an influenzal infection broke out in one of his heart-wards and caused the death of two patients and jeopardised the lives of several others.

Influenza frequently causes the condition known as "Soldier's Heart" which is referred to at the end of this thesis.

PNEUMONIA. The importance of the heart muscle in pneumonia is recognised by everyone. At the outset, the pulse indicates the toxaemia of the heart. The rate may be 115 - 130, soft and compressible, the absence of sustained pressure always being a serious sign. The heart sounds are short and sharp at the outset and the second sound in the pulmonary area is accentuated. They become muffled and usually some dilatation occurs. Finally the pulse rate increases and irregularities appear./

appear. Acute dilatation may cause sudden death as exemplified in the case of a little boy Smith of Maddiston. He was exceedingly restless and the parents were warned of what might happen: though the parents took every care of him, one day he suddenly sat up when there chanced to be none at the bedside, and he fell back dead.

The causal organism may invade the heart or the heart may be only affected by the toxaemia. It is impossible to tell whether the heart has been directly invaded or not.

The effects of digitalis differs in the hands of different authorities. Sir Thomas Fraser was a great believer in the effects of the digitalis group while Sir James Mackenzie is opposed to its use.

I specially noted its effect in the case of James Rankine (aet. 16). He had a left sided lobar pneumonia followed by an empyaema (rib-resection) and then a right sided pneumonia from which he died. I tried him with varying doses all through his illness and I do not think it made any difference to his pulse rate. In other cases I have thought it beneficial but possibly it was not so. As an Edinburgh student one cannot readily accept the theory/

theory of its uselessness after having been taught by such an eminent authority as Sir Thomas Fraser, and I can find no one who declares that Digitalis does actual harm so I always employ it in Pneumonia in the hope that it may be beneficial.

DIPHTHERIA. The myocardium is particularly susceptible to the toxin of diphtheria and fatal syncope is frequent. Fatty degeneration may be severe.

TUBERCULOSIS. Fever in tuberculosis will profoundly affect the myocardium, but in cases without fever the toxic effects on the heart muscle are evinced by the rapid pulse rate. Riviere ("The Early Diagnosis of Tubercle", page 10) states that "a pulse persisting at 90 - 100 in absence of fever may arouse suspicion of early tubercular disease".

For some months I have been a member of the Medical Board at the Ministry of Pensions whose primary duty is the examination of cases of tubercular lung disease. Where the patient has not been improving there is a rapid pulse, and this evidence of toxaemia of the myocardium is a valuable indication of the man's condition.

SEPTIC INFECTIONS. These affect the heart either by toxaemia or the specific germ invading the heart, when a definite myocarditis becomes apparent with serious depression of the heart muscle. The organism can often be cultivated from the blood, e.g. Streptococcus, B. Influenzae, Pneumococcus, Gonococcus, Staphylococcus Aureus, etc.

Dilatation with a secondary mitral incompetence occurs.

In Pyaemia and Puerperal Septicaemia the heart often becomes very feeble and the pulse small and easily compressed. Actual abscesses may form in the myocardium (suppurative myocarditis).

The Prognosis depends on the amount of infection. After the active disease has subsided if the heart has resumed its normal size, rate and rythm, it may be presumed that the damage has not been extensive and the prognosis is good. But low inflammation may lead to fibrous changes when the prognosis is guarded.

Treatment is often most disappointing. Vaccine or serum therapy may be tried. In rheumatic fever the salicylates are always employed. Rest is of the/

the first importance. Sponging often diminishes the restlessness of the patient and so indirectly assists the heart. Sleeplessness must be prevented; if the milder hypnotics fail opium must be resorted to. Digitalis is often of the greatest benefit. Morphia has to be used with care: and never if there is any oedema of the lungs. (Case of Miss White).

Over-exertion must be avoided during convalescence. Anything that increases the heart' frequency must be avoided, no exertion being allowed so long as any dilatation remains: only after slight exertion fails to cause disagreeable sensations may light work be resumed. The patient's sensations are the best guide to this.

SYPHILIS. The most frequent effect of syphilis is to produce a cardio-sclerosis. In this way it may lead up to the Senile Heart. q.v. Gummata may form in the heart muscle and lead up to Heart Block, q.v. (Case No.30)

Syphilis generally attacks the vessels and in this way affects the aortic valves. A definite regurgitation at the aortic valve always implies myocardial involvement. (Case 20.)

5. HEART FAILURE.

The generally accepted view of heart failure may be instanced by a man with aortic incompetence. The left ventricle dilates and the left auricle has an increased strain thrown on it owing to the regurgitation of blood from the ventricle. Consequently the pulmonary circulation becomes congested and thus the right ventricle has an additional pressure to work against. Eventually tricuspid incompetence induces congestion of the whole venous system. (Case No.20)

Suppose, however, that the left ventricle hypertrophies sufficiently to cope with the regurgitating blood from the aorta then "the compensation" is said to be "good", and there is no evidence of heart failure. Should signs of heart-failure appear at a later date then the "compensation has broken down" and if he recovers the "compensation has been restored".

Case of Major _____ whom I knew in Sunderland for about three months. He had an aortic diastolic and mitral systolic murmur. He was able for active duties with his regiment, though in Category C.

I/

I sat on a Board examining him for possible service in India and he told me that at the innumerable Boards he had attended he had invariably been told that he had "remarkably good compensation".

As Mackenzie points out these views are due to the discovery of auscultation. The back-pressure theory did not result from observation of individuals passing through its various stages, but by assuming that the events happened as required by the theory.

As has been pointed out, heart failure is the result of impairment of the function of the heart muscle: the muscle may appear to be healthy, but even then there are changes, which quite possibly cannot be demonstrated; or the function may be impaired without pathological signs. Major ——— on the other hand is an example of healthy myocardium.

Here is a man with angina pectoris, and post-mortem there is found atheroma of the aorta and coronary arteries. These are always regarded as correlated, but what is the mechanism by which the anginous pain is produced? Presumably it is a condition of "cramp" similar to that brought about in the calf muscles in cases of Intermittent Claudication. Here is another man with a diseased valve and he suffers from shortness of breath and/

and dropsy: the actual valve lesion has not in itself produced the dyspnoea, but a functional derangement of the heart muscle which is found in all heart lesions and is the cause of the symptoms whether anginous or otherwise.

Thus one finds cases of cardiac murmur or cardiac irregularity in which the heart may be perfectly normal although these signs in other cases are of the gravest significance: and yet again both may be evidence of merely functional derangement.

So one comes to the conclusion that although there may appear abundance of healthy muscle at a post-mortem examination, the heart failure has been the outcome of functional derangement of the muscle although this cannot be proved.

According to Mackenzie (ibid. p.8):-

"A Murmur amy be the outcome of a disease process, or a functional impairment, yet it may be an expression of a perfectly normal heart.

An Irregular Heart may be evidence of a profound and fatal exhaustion, of an impaired organ, or it may be a perfectly normal sign.

Breathlessness may be a sign of great gravity, of an impaired organ, or it may occur with a normal heart."

6. CHRONIC MYOCARDITIS.

DIAGNOSIS. Myocardial disease may be taken for granted in every case of cardiac enlargement: if the enlargement is great the myocardial affection is greater than if the enlargement is not so marked.

Most authorities are agreed that cardiac enlargement is ascertained by percussion, but Lewis in "The Soldier's Heart", page 59, (Shaw & Sons, London, 1918) is emphatic: "Accurate delineation of the heart's size by percussion is not possible. The margin of the heart moves a centimetre in systole, often it moves more. It moves also with respiration. The impression of a sharp margin of dulness is largely fictitious; .....". page 55. "The area over which the heart's impulse is felt may be covered in most men by a shilling piece; and the outermost limit of this circumscribed area is the best clinical guide which we possess to the left border of the heart, and providing the heart is not displaced, to the size of the organ ..... in hearts of normal size or in hearts slightly or moderately enlarged."

(1)/

(1) Where the apex beat of the heart is displaced outwards, then, apart from deformities, effusions, etc., the myocardium may be taken as dilated or hypertrophied and diseased in consequence. No perfectly normal heart is hypertrophied. It is enough to find enlargement. Dilatation and hypertrophy are usually associated.

(2) The type of impulse of the apex beat is important. A diffuse apex-beat is generally found, though dilatation is not necessarily accompanied by a diffuse apex-beat. In hypertrophy of the left ventricle, for example, a diffuse apex-beat is generally associated: the thrust is systolic in time. If the thrust is diastolic in time, then a diffuse apex-beat indicates dilatation or hypertrophy of the right ventricle, unless the case be one of adherent pericardium.

(3) Where the heart's systole causes very definite movement of the ribs and sternum, specially where this movement is quite palpable, there may generally be assumed some cardiac enlargement, particularly where the lower end of the sternum is much moved. In this event adherent pericardium should be kept in mind.

(4)/

(4) A definite aortic regurgitation always implies disease of the myocardium. This particularly applies to syphilis where there is often also disease of the coronary vessels. In rheumatic cases the myocardium may be directly infected as well.

(5) A well developed, not an early, mitral stenosis also indicates disease of the heart muscle.

(6) Other conditions indicating myocardial involvement are:-

Chronic renal disease.

Persistent high blood pressure.

Thoracic aneurism.

Flutter or fibrillation of the auricles.

Heart block.

Pulsus alternans.

SYMPTOMS AND SIGNS. The first symptom generally noticed by patients suffering from myocarditis is breathlessness on exertion. This is often associated with pain. The patient easily tires and is glad to rest. The symptoms often come on so gradually that the patient does not allow himself to believe in them and either gradually reduces his daily exertion or else tries/

tries to "work it off" in which case the symptoms progress, and the patient seeks his doctor. But where the patient reduces his daily work it may be a long time before the doctor is called in. Case of Henry Cullen (No.20) and David Love (No.29).

These first symptoms are the indication that myocarditis is leading on to heart failure.

As time goes on the veins become distended and if the neck is examined the veins may be seen to be engorged, and the liver becomes enlarged. Congestion of the lungs may be diagnosed by means of the basal crepitations and accentuation of the pulmonary second sound. Cyanosis and diminution of the urine passed, lead up to ascites, swelling of the extremities. Albuminuria often appears and is not necessarily due to inflammation of the kidneys but may be due to chronic venous congestion. (Case No.20): the patient himself will sometimes be able to assist in describing previous attacks of Bright's disease, or otherwise (Case No.36).

In middle aged and elderly persons angina pectoris may make its appearance without almost any enlargement of the heart. If it is true angina the symptom is an indication of commencing heart failure.

PROGNOSIS./

PROGNOSIS. In every case of myocarditis the heart's function is not quite so efficient as in the healthy heart. This is scarcely noticed by the patient and he may allow the heart to get exhausted. Patients must be warned against commencing symptoms: so long as these are studied and suitable treatment adopted the heart muscle should last for many years. In progressive valvular lesions secondary fibrotic changes in the myocardium will almost certainly take place.

The prognosis is not so satisfactory because of the progressive element, and this must be retarded so far as possible by careful living.

Where myocarditis exists and the heart is infected with rheumatic fever or other acute infections, the attack probably leaves the myocardium in a less efficient condition. An acute, on the top of a chronic myocarditis always indicates a guarded prognosis.

Should syphilis be the leader in the train of events, suitable treatment may be of great benefit. The same applies to high-blood pressure and Bright's disease.

Any type of myocarditis indicates a careful prognosis because of the possibility of an acute infection.

TREATMENT. Dilatation generally considered, is an indication for the administration of digitalis, associated necessarily with rest in bed. Rest alone is enough in some cases, depending on the cause and condition. I treated Miss Inglis (Case 39) with rest in bed only, for six days by which time the heart had returned to its normal size, oedema had disappeared and she felt generally quite well.

Digitalis, also Strophanthus and Squills which belong to the same series, are certainly wonderful drugs for their effect. In some cases digitalis does not suit the idiosyncrasy of the patient when strophanthus or squills may be tried, but generally if digitalis fails the others are useless. I remember particularly a man Scott (Case No.27) with severe beart failure who did not benefit much from digitalis, so I tried him with strophanthus and he vomited after the first dose and continued vomiting for a whole day and nearly died.

The taste of strophanthus is a serious drawback, as it is extremely objectionable. The taste may be masked by giving tincture of capsicum in addition, and vomiting may be prevented by the administration of/

of a sixth of a grain of cocaine hydrochloride in pill form.

Nausea or vomiting during the administration of digitalis is generally an indication that enough of the drug has been given for the time being, and usually there will be found an accompanying slowing of the pulse. The case of Mr Love (No.29) was rather puzzling as he had attacks of retching before digitalis was started, probably due to chronic venous congestion of the stomach.

Other indications of overdosage of digitalis are various abnormal rythms such as bigeminal pulse, heart-block, extra systoles, auricular fibrillation, diminished urine which may be of a reddish-brown colour and dementia in old people (Case of Miss White) The same Scott, I overdosed with digitalis and he developed a bigeminal pulse which very soon disappeared when the drug was stopped.

It is in Auricular Fibrillation that digitalis has its most marked effect. I can well remember attending a Mrs Oliver (Case No.25) in Durham, as a Locum Tenens. Medicines were given from the doctor's surgery. I gave her Tr. Digitalis and later Infusion of digitalis and there was absolutely no change in her condition. I then ordered her Tincture of/

of digitalis from the best chemist in Durham and the effect was immediate, within a week all palpitation and oedema had disappeared and the patient expressed herself as "feeling quite different". The doctor's preparations were evidently old and I threw them away with a feeling of satisfaction.

I have found Nativelle's granules very satisfactory in several cases. The granules are prepared in two strengths, distinguished by their colour: the white granules contain 1/240 gr. of digitoxin and the red granules contain 1/600 gr. I used these in the case of Mrs David Reid (Case No.24) who had auricular fibrillation: she took one granule three times daily. In two days she felt infinitely easier and although the pulse was still very irregular it had greatly improved and become countable (120). Two days later it was 68 and the dose was reduced to one granule per diem.

The actual administration of digitalis is usually by means of the tincture of which three doses of twenty minims are given daily. This is continued until the pulse slows down sufficiently or other indications of slight poisoning are noticed, such as, nausea or diarrhoea. The drug may then be stopped for a couple of days. The infusion is the best diuretic preparation but has the disadvantage that it/

it requires to be prepared freshly every forty-eight hours as it does not keep.

It is quite possible to kill a patient with digitalis. This will not occur if the signs of digitalis poisoning are regarded, and the drug stopped for a couple of days.

Some patients continue to take digitalis continuously for years. The patients very soon know when they have had enough of it.

The associated conditions of heart failure require special attention. Oedema can be best counteracted in many cases by the effects of digitalis on the heart. It is a good plan I have found, to associate a drug such as calomel and to have the bowels well cleared daily. Guy's Pill is designed for this purpose: it contains Pulv. Scillae, Pulv. Digitalis and Pill Hydrarg, of each one grain.
Diuretic mixtures are often of assistance. I often start a patient with.-

R Pot. Acetatis ℥ss.
Spt. Aetheris Nitrosi ℥i
Tr. Scillae ♏x
Tr. Digitalis ♏xx
Infns Scoparii ad ℥ss

but/

but of course there is no end to the prescriptions that are used. Diuretin is often helpful. Salt free diet is of the greatest value, it having been demonstrated that retention of chlorides is always followed by retention of fluid. Southey's tubes have sometimes to be used, but I have never used them in general practice.

Ascites may require tapping as may hydrothorax, though much can be done to diminish these with rest in bed and diuretics and digitalis in sufficient quantity.

For oedema of the lungs breathing exercises are sometimes beneficial

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7. ACUTE MYOCARDITIS.

Acute myocardial involvement is certainly present in acute fevers, so much so that often enough the heart condition is of greater import than the fever itself.

The effects of the fevers is discussed under "The toxic effects of fever" at page 81.

8. SUPPURATIVE MYOCARDITIS.

This occurs in cases of pyaemia, puerperal sepsis and other generalised organismal infections.

9. SUBACUTE MYOCARDITIS.

This is a somewhat vague condition. If a mild infective condition attacks a patient so as to produce practically no symptoms one cannot be certain of anything. Suppose the myocardium so attacked, one would not be able to diagnose it. One sign there is which may become evident and that is produced by the auriculo-ventricular bundle being involved in addition. When this is so, it is probable that partial heart-block will be produced. But it should be/

be possible to have some myocarditis without the auriculo-ventricular bundle being attacked.

Subacute myocarditis is generally associated with muscular rheumatism, the heart muscle being attached in the same way as the skeletal, thus pain is one of the symptoms. Owing to the cardiac function not being normal, breathlessness, palpitation and violent beating of the heart are complained of. (Case of Peter Peat - No.32).

Etiology of Myocarditis.

The etiology of myocarditis is dealt with under the various sections. Generally speaking it is an infection: a mechanical defect may cause heart failure but infection of the myocardium has far more to do with the heart failure than has the mechanical defect.

The most important infection is undoubtedly Rheumatic in origin. (Rheumatic Fever, Chorea etc.) It is astonishing how frequently this is found to be the origin.

The various infectious fevers, influenza, dysentery, pneumonia, enteric fever, etc. are important etiological factors.

Various poisons have their share: tobacco, alcohol,/

alcohol, arsenic, etc. Acute Nephritis (Ernest Pearson) Urticaria (Mrs Foggo) Microbic infections of all sorts e.g. streptococcal, colon bacillus.

Infection is the key note of myocarditis.

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10. CHANGES IN THE MYOCARDIUM DUE TO CHANGES IN THE ENDOCARDIUM.

Valvular defects embarrass the heart by increasing the power required to force the blood through a narrowed orifice; or should there be a regurgitation, extra work is required to send the additional regurgitated blood on its course. An added strain is, in any case, thrown upon the heart muscle.

In order to meet the additional strain the heart-muscle hypertrophies and in this way the necessary work is carried out. The forcible thrust of the apex-beat is the best evidence of hypertrophy. But a hypertrophied heart is no longer a normal heart, so it follows that the field of response is always limited. It is an effort to keep up the hypertrophy. As time advances this becomes a serious consideration as the circulation may be just sufficient under normal conditions and the hypertrophied heart requires a little more blood than it received so that degeneration and dilatation follow. But patients with valve lesions may live comfortably for years without symptoms. Such patients are not easily discovered unless one happens to examine the chest for some reason/

reason or the patient himself gives the information as did Mrs Morrison (Case No.1) and Nos. 2 & 6.

With a damaged myocardium careful living is indicated. Over-exertion, drinking-bouts and other excesses entail an extra strain on the myocardium which was quite able to bear the average work required of it. Or some illness may supervene and upset the balance.

At first there are no symptoms, but when the heart muscle becomes exhausted symptoms of cardiac failure become apparent.

In some cases the valve lesion is a sclerotic one and is progressive in character and there may be associated advancing changes in the heart muscle which may cause shortening of the chordae tendineae or affect the auriculo-ventricular bundle, so that the conductivity of the bundle is interfered with or auricular fibrillation produced.

Cicatricial changes may also affect the auricle, so that the rythm of the heart is upset. This is liable to follow mitral stenosis.

Murmurs are not necessarily organic and this applies particularly to mitral and tricuspid murmurs. Impaired muscle tone may lead to regurgitant murmurs at/

at these orifices, sometimes merely fugitive after fatigue or excitement. Tricuspid regurgitation may occur without a murmur, only jugular and liver pulsation indicating the condition. The tricuspid valves according to Mackenzie (ibid. p.336) are barely able to close the orifice and very easily become incompetent.

Another murmur, which is present in some cases of chlorosis, and is not due to valvular disease is the pulmonary systolic. This is explained as follows by Russell (Brit. Med. Journal, June 24th, 1916):- "The right ventricle is lifted by dilatation of the left heart into close contact with the chest wall, so that its pulsations may be visible in the third, fourth and fifth left spaces; systole of the right ventricle, especially systole of that portion of it called the conus arteriosus, carries the origin of the pulmonary artery obliquely downwards and to the right; if you endeavour to simulate this action, you will find that the anterior and posterior parts of the artery at its origin are approximated, and that a relative narrowing is thereby produced. This narrowing, is to my mind, undoubtedly the mechanism by which the murmur is produced. It explains the disappearance/

disappearance of the murmur during inspiration, for with inspiration the heart has a freer forward movement and the kink is prevented."

Aortic valvular disease producing heart failure is generally the result of changes in the myocardium coincident with the valve changes, such as that caused by syphilis and arterio-sclerosis. I came across a man with a musical aortic diastolic murmur which certainly appeared to have been caused by rupture of the valve while the patient was lifting a heavy bale of goods at the docks in Dublin where he worked. But it is probable **** if rupture does take place, that there has pre-existed some disease of the valve.

Angina pectoris is often associated with aortic stenosis.

Aortic regurgitation is usually associated with stenosis of the valve. The combination of a hypertrophied heart with a powerful beat followed by reflux produces the characteristic "water-hammer," - or Corrigan's pulse.

Even with a double murmur there may be little or no dilatation and the patient may be able for a full day's work, proving that the disease of the valve has not spread to the muscle. Even with considerable/

considerable dilatation however the patient may be able for work, but he has to lead a careful life and is subject to attacks of heart failure.

The symptoms of cardiac failure generally commence with dyspnoea and palpitation on exertion. One cannot say from the symptoms alone what the cardiac condition may be as the symptoms are common to all valve lesions with heart failure. Sometimes the aspect gives one an idea of the condition as instanced by the pallor of aortic regurgitation and the redness or cyanosis of mitral disease.

As the heart becomes exhausted the usual signs and symptoms of heart failure appear, including many complications such as auricular fibrillation, paroxysmal tachycardia, angina pectoris, etc.

TREATMENT. If treatment is commenced early, the sooner will the patient improve and it is usual to recover entirely from the first and from many subsequent attacks and perhaps to continue for years, though the patient must live a careful life, avoiding excess in anything, e.g. exercise, drinking, smoking, etc.

On examining the patient there may be found some obvious cardiac defect such as a sclerosed valve. It/

It is evident that the valvular sclerosis cannot be removed, at least in the present state of knowledge. The aim then becomes one of endeavouring to get the patient's heart into such a condition that he will be able to go about his business with as little inconvenience as possible.

First it will be necessary to find out what cardiac reserve he has, since cardiac failure depends on exhaustion of the reserve of the heart. The reserve may be very far below normal, but the best must be made of what remains, particularly where the valvular disease is a progressive one and will gradually reduce the cardiac reserve whatever the treatment.

To estimate the cardiac reserve of the patient an inquiry may be made into his daily work. If he can perform a full day's work with very little exhaustion, careful regulation of his employment may be able to do away with the exhaustion. If he gets seriously exhausted his work will have to be drastically curtailed. Many men think that they can "work it off", as is exemplified in the case of Cullen: I explained repeatedly to both him and his wife that he would not benefit his condition by over-exercise but he persisted in his belief as long as he was able.

In/

In this way many men persist at their daily employment in the belief that one day they will begin to improve, and their decline is often so gradual as to be scarcely noticed by the men themselves. (Cases Nos. 20 and 29)

So long as a man has not a progressive lesion he should continue at his work provided the reserve is not over-exhausted, but he must not include any extra work, or particularly arduous work which causes him distress. (Case No.19)

When, however, a patient is unfit for regular daily work it will be a good plan for him to have some occupation which does not involve the necessity of working in the company of fit men who do a certain amount of work daily: he may be able to act as an Insurance Agent in a small district. In this way he will have some occupation to keep his mind off his infirmity and be able to regulate his work by his ability.

The next stage is the man who rarely gets out of the house; and finally the man who is confined to his bed. Even in these, muscular exercise is useful. It must be carefully regulated so as not to embarrass the heart. (Case No.20).

Thus exercise is beneficial in all cardiac conditions, except progressive cardiac disease and where the/

the rest-force is exhausted: excluding of course acute cardiac affections. The general indication being to continue exercise so long as there are no symptoms such as dyspnoea, pain, palpitation or a feeling of exhaustion.

Rest is however just as important as exercise and more important very often. In acute cardiac disease it is essential and exercise is contra-indicated.

Administration of digitalis is in reality a method of inducing rest for the heart, for by diminishing the rate of the heart, rest is assured.

If the heart is exercised up to the limit of the cardiac reserve, as for example in the case of Cullen, it is essential that a period of rest should follow and the condition of the patient will make this obvious to him. Cullen would walk till he could walk no further: if he had not reached his destination he would rest only until he could walk a little further, and so on. Eventually he had an hour or two of rest at the harvest fields and then gradually return home exhausted. A night's rest restored him a little and he started again.
He naturally went slowly down-hill.

The period of rest must be sufficient to restore the/

the reserve. Otherwise the heart is more and more easily exhausted. The patient soon learns how much rest he requires.

Some people need to be prevented from over-excitement as this quickly exhausts the heart. Such patients usually benefit by the administration of bromides, of which Mrs Forrest is an example. (Case No.40)

Feeding must be judicious, but the effect on the patient's mind must not be forgotten. Milk at frequent intervals is the best diet in cases of extreme heart failure, about a pint in the day should suffice if there is much dropsy. In milder cases salt should be eliminated as far as practicable. No excesses are permissible.

When there is great distension of the heart venesection causes relief, but its effects are only temporary so that it is seldom practised.

Baths. Special baths such as those of Nauheim have their advantages. The regular life probably does more good than the baths. Open sea bathing is probably the best kind of bath provided the patient enjoys it.

Digitalis is really one of the most satisfactory drugs to use. If administered in sufficient doses its/

its effect fills one with admiration. The digitalis group is described more fully under "Chronic Myocarditis".

Oxygen. I made a careful examination of the effects of oxygen in two soldiers I was treating in hospital in Italy who died of heart failure due primarily to valvular disease. Both benefited by the administrations but the beneficial effects were only temporary.

Often a holiday and a complete change of life is of great benefit in cheering up the patient and giving him a more hopeful outlook on life, especially in cases where the patient takes a morbid interest in his condition. Benefit may also follow from restrictions of indulgences in tea, coffee, alcohol, smoking, etc. Where the patient is sleepless or worried the bromides are often helpful as in the case of over-nervous individuals.

Besides the treatment of the cardiac condition a general investigation into the patient's mode of life will often prove that he has not been judicious in his living.

PROGNOSIS./

PROGNOSIS. This depends largely on whether the valvular affection is a progressive one. Healthy hearts may have a murmur, the presence of which is not to be allowed to condemn the patient to a restricted existence. The prognosis depends on the changes, or their absence, which have taken place in the heart muscle and particularly in the auriculo-ventricular bundle.

Consequently one may confidently assert that the condition of the myocardium is of greater significance than the valve lesions.

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11. CHANGES IN THE MYOCARDIUM DUE TO CHANGES IN THE PERICARDIUM.

The heart is surrounded by the fibrous pericardium beyond which it cannot dilate in the healthy individual.

Pericarditis is most commonly associated with rheumatism, although it may be caused by a variety of other infections such as the acute fevers, septic conditions and tuberculosis.

In rheumatic pericarditis the myocardium may be infected as well, and as simple pericarditis "perse" is not a serious condition the condition of the myocardium becomes of primary importance. It is myocardial involvement that renders this such a treacherous condition. When the condition goes on to adherent pericardium some hypertrophy of the heart takes place, but unless there is no adherence to the structures outside the pericardium the hypertrophy does not need to be great and there is little to indicate the condition.

The extra-work necessitated in overcoming the friction with the pericardium is of minor importance and is easily accomplished by the myocardium.
Many patients go about with pericarditis without ever knowing/

knowing they have it at all.

In the fevers, the myocardium is already poisoned. The extra work entailed by the pericarditis is only of secondary import.

In pericarditis with effusion the degree of embarrassment of the heart depends on the rate with which the fluid is poured out. If poured out slowly there is often remarkably little compression.

Tuberculosis of the Pericardium if early does not affect the myocardium. But it may go on to chronic adhesive pericarditis which causes the same train of events as adhesive mediastino-pericarditis.

Adhesive mediastino-pericarditis may be either tubercular or inflammatory. All the structures around the heart become matted together and the heart becomes fixed to the spinal column behind and to the ribs in front. There may be an associated proliferative peritonitis, perihepatitis and perisplenitis. As the posterior surface of the thorax is unyielding, the heart pulls on the ribs during systole, thus drawing them inwards and they return to the normal position during diastole.

As the condition gradually develops, the extra work necessitated to the heart produces gradual hypertrophy of the heart muscle which may become extreme,/

extreme, some of the largest hearts being caused by this condition. As the inflammation increases the heart becomes unable to cope with the work and dilatation ensues leading up to the usual signs of heart failure.

The prognosis is always of the worst.

Treatment is practically hopeless. Bower's cardiolysis is sometimes performed, being the resection of portions of the fourth, fifth and sixth ribs with a small piece of their costal cartilages. This gives the heart a little more space and affords some temporary relief.

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12. CHANGES IN THE MYOCARDIUM DUE TO CHANGES IN THE CORONARY ARTERIES.

Thickening of the coronary arteries necessarily leads to deficient blood supply to the heart muscle. The terminal branches of the coronary arteries are end-arteries. The anaemia gradually leads up to necrosis. The muscle fibres lose their fibril bundles and are replaced by connective tissue. The sclerosis may occur anywhere, but is most commonly seen at the apex of the left ventricle and in the septum, the area supplied by the anterior coronary artery. In this way a condition of Fibrous Myocarditis is set up. Hypertrophy is commonly associated with the degeneration. (Case No.36) The condition may lead up to aneurism of the heart.

Small wedge-shaped or irregularly shaped areas of necrosis may be produced which are equal to Infarcts. These may lead up to rupture of the heart.

Where a coronary artery is gradually completely obliterated the other coronary may be able to carry on the circulation. But if the vessel is suddenly occluded, for example by a thrombosis, sudden death is/

is caused, as the myocardium cannot work without its blood-supply.

Narrowing of the lumen with the consequent slowing of the circulation may lead up to thrombosis.

Such conditions as extra-systoles, auricular fibrillation, pulsus alternans or cardiac asthma may be induced, or simple heart-failure may be the first sign.

By far the most interesting condition caused by coronary disease is Angina Pectoris. The pain being produced by the extreme exhaustion of the heart muscle which is the result of diminished blood supply. Or the diminished blood supply may cause fibrous and fatty degeneration of the muscle, so that the amount of active muscle is so far reduced that the remainder is absolutely exhausted and the anginous pain is produced.

Angina pectoris is usually found in hearts which have for long struggled against various difficulties. Just as various symptoms such as breathlessness, etc. are produced when the reserve force is exhausted so anginous pain may be one of the symptoms. It indicates that the heart muscle is not able for the particular exercise which produced the anginous pain, and it is/

is a warning that the heart muscle is diseased as a rule, though the pain may be produced by overtaxing a healthy heart.

False angina pectoris is produced in some cases by mental excitement inducing the heart to beat so rapidly that it becomes exhausted. In such cases the myocardium will entirely recover.

The Prognosis of Angina Pectoris depends entirely on the condition of the myocardium. In young persons who have over-exerted themselves, a period of gradually increasing exercises will restore the reserve of the heart.

In the middle aged, by living his life at a lower level no further attacks will be experienced except when the patient indulges in over exertion of some sort.

In the elderly, with thickened arteries, and a high blood pressure the outlook is very grave, as the myocardium is badly nourished and probably considerably degenerated. The condition will slowly progress.

Treatment of Angina Pectoris consists in improving the condition of the myocardium where this is possible. Where the patient is sleepless or worried or overindulgent, these must be put an end to. Where over-exertion induces the attack the work must/

must be modified accordingly.

In order to permit the heart to regain its reserve force a period of rest is advisable, all work is avoided and the patient lives a very quiet life for a short time. The strain should be further reduced by the administration of vaso-dilators.

Where the heart has been exhausted a complete rest in bed will be essential to restore the heart as far as possible.

Otherwise the treatment is the same as detailed under Myocarditis.

Bate (Alabama Medical Journal. July 1909) states with reference to the use of Pituitrine.-
"The angina of arterio-sclerosis has quickly yielded in two cases coming under my observation". I have not heard of Pituitrin being used in this country.

ETIOLOGY. Arterio-sclerosis is an accompaniment of old age. It is often hereditary but is usually due to "wear and tear" such as overindulgence in Alcohol, overeating and over-working.

Toxaemias such as lead, gout, acute infections play their part.

High blood-pressure and Bright's disease are often associated.

Syphilis is a frequent cause, specially in aortitis.

TREATMENT. Sclerosis of the coronary arteries is to be treated in much the same way. A quiet well-regulated life with avoidance of all excesses is indicated. Where the history is syphilitic, the iodide of potassium is particularly of use and it is of assistance in all cases. Where the blood pressure is high, sodium nitrite and nitroglycerine may be given.

The Heart in cases of High Blood Pressure resulting from Bright's Disease, etc.

The blood pressure depends on the peripheral resistance and the force of ventricular systole. In accordance with the increased resistance, the heart hypertrophies enormously, particularly the left ventricle. The chambers may dilate a little. The apex beat is very marked to palpation and may be outside the nipple line. The aortic second sound is accentuated.

Patients live for long periods in this condition and the end is very various, cerebral haemorrhage, and a variety of like conditions causing death. Fibrous/

Fibrous myocarditis is generally associated, and as the condition advances the heart becomes prematurely senile. The exercise tolerance is reduced and cardiac failure eventually sets in with or without fibrillation of the auricle.

The Prognosis and Treatment are confined to the arterio-sclerosis. (Case No.36)

V. DEGENERATIONS OF THE MYOCARDIUM.

1. THE SENILE HEART.

Generally speaking, degeneration of the heart muscle due to advancing years consists of fibrous and of fatty changes. Thickening of the arteries always takes place as age advances, particularly where there is kidney disease, syphilis, over-indulgence in alcohol, over-work, etc. The semilunar valves are often involved in the fibrous changes.

As the blood-supply diminishes the muscle fibres degenerate and at the same time the heart has an extra force to contend with in the raised blood-pressure due to the narrowing of the arteries throughout the body. (Cases No.10 - 14)

A sure result of the degeneration is a diminution of the reserve force of the heart. The patient gradually finds himself unfit for his daily occupation and later for any exertion.

The heart may be regular to the end as in the case of David Winton (Case No.10) or various irregularities may be set up and heart-block if the auriculo/

auriculo-ventricular bundle is involved. (Case No.30)

Sooner or later the heart dilates, ushering in the signs of cardiac failure. Brown atrophy is common.

PROGNOSIS. The Prognosis depends on the condition of the myocardium, and whether the condition is progressive. Where this is not extensively involved a quiet existence may be carried on, but where there is little or no response to treatment the condition is very unfavourable.

TREATMENT. The treatment in early cases is limited to avoidance of over-exertion and over-indulgence of every sort. Exercise is enjoined and a change of surroundings is often beneficial. The best use must be made of the remaining healthy muscle fibres and their reserve force may be increased by careful treatment.

Sleepless nights are to be avoided as they tire the patient and exhaust the heart. Some light food at bed-time is often sufficient to induce sleep: in other cases bromides, chloral, veronal or sulphonal have to be resorted to. Morphia is better avoided. (Case of Miss White). I have found Dial very satisfactory.

Vasodilators are often used in order to diminish the resistabce against which the heart has to work. They are in no way curative, but one is sometimes driven to use them in desperation, as in the case of Mrs Lillie (Case No.32) whose blood-pressure is generally about 270 m.m. and often over 300 m.m. When given over long periods their administration is not very satisfactory.

David Winton suffered from a senile heart.

2. FATTY DEGENERATION OF THE MYOCARDIUM.

This condition can scarcely be dissociated from The Senile Heart in many cases. Fatty degeneration usually follows from sclerosis of the coronary arteries, and the diminution of the blood supply leads to a fatty degeneration of the muscle fibres.

Fatty degeneration is however found in wasting diseases and cachetic conditions.

It occurs in prolonged infectious fevers, especially diphtheria. Fatty degeneration may be associated with parenchymatous degeneration.
It is very marked in cases of phosphorous poisoning and pernicious anaemia.

Where/

Where the fatty degeneration is superficial in the heart muscle there is often pericarditis.

In the hypertrophied heart fatty degeneration of the muscle is assured, sooner or later, provided some other condition does not carry away the patient before the degeneration has started.

The heart muscle, owing to its constant activity, shows this degeneration before it appears in other muscle. Oxygen must be supplied in sufficient quantity and when it fails degeneration sets in rapidly. The left ventricle is most commonly attacked.

Fatty degeneration cannot always be diagnosed as such with certainty. It may be present in extreme degree with a full pulse and a regular heart. The heart's function is not seriously interfered with and so long as marked dilatation does not occur there may be no symptoms. As dilatation commences shortness of breath, palpitation, irregularity, weak action, lead up to cardiac failure. Rupture of the heart is a special danger and may be brought on by strain of any kind, particularly by straining at stool. The commonest site for rupture is near the apex of the right ventricle.

The treatment is limited necessarily to that of heart failure.

Fatty Infiltration of the Myocardium.

Fatty Infiltration is an accompaniment of general obesity and cannot be diagnosed without it. It generally occurs in the middle-aged and elderly. It is a simple excess of the normal sub-pericardial fat.

The fat insinuates itself between the muscle-fibres, in some cases as far as the endocardium, particularly in the right ventricle. In some cases even the papillary muscles show evidence of fat, but this is unusual. The muscle fibres often show marked fatty degeneration.

No symptoms are produced until dilatation takes place from weakening of the heart muscle. The heart sounds are weaker and muffled and sudden death may take place from syncope or rupture of the heart.

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3. THE FIBROID HEART.

This is a condition which one cannot diagnose with any certainty. There are however always indications when the sclerosis of the heart is well marked and these are the signs and symptoms of cardiac failure.

The aetiology is frequently associated with sclerosis of the coronary artery, but syphilis or rheumatism of the heart may lead up to fibrosis.

The heart's action is often irregular, though not always. The pulse is slow and feeble. Angina pectoris may be present if there is associated coronary disease.

The Diagnosis is difficult and will have to depend on the weak and muffled heart sounds even though the position of the maximal impulse is quite apparent.

The Prognosis is grave as the condition is generally not discovered until the sclerosis is well advanced.

The Treatment is that of myocarditis.

4. OTHER DEGENERATIONS OF THE MYOCARDIUM.

There are a few other degenerations which are of little clinical interest, such as Brown Atrophy, Calcareous Degeneration, Amyloid Degeneration, and the Hyaline Transformation of Zenker.

Brown Atrophy is frequently found in senile hearts and chronic valvular lesions. The heart muscle becomes a dark brown colour. The muscle fibres when examined microscopically have some yellowish-brown colouring matter around the nuclei.

VI. IRREGULAR ACTION OF THE HEART.

1. SINUS IRREGULARITY.

This irregularity depends on the stimulations passed to the heart from the Sino-Auricular Node. This is the area where stimuli normally originate and if the stimuli are regular, as they generally are, the pulse is regular. But sometimes the stimuli from the sino-auricular node are not regular.

The vagus nerve exercises an inhibition of the heart in the normal subject and should the vagus centre be easily excited the excitement will influence the heart through the vagus, the effect being a lengthening of the diastole of the heart.

Thus respiration may excite the vagus and thus change the rate of the heart.

In animals where there is a sinus irregularity section of the vagus causes the disappearance of the irregularity.

Sinus irregularities disappear when the pulse-rate is increased, e.g. by exercise or during fever.

When the pulse is examined the rate changes with respiration though the individual beats are equal in strength.

The/

The heart sounds indicate that the irregularity is in the length of the diastole for the interval between the first and second sounds is constant. Tracings of the jugular pulse indicate that the auricle is affected.

The slowing may be frequent or rare and it may affect only a few beats.

The myocardium is not responsible for the irregularity and the heart muscle is perfectly healthy.

No treatment is called for.

2. EXTRA-SYSTOLES.

The occurrence of extra-systoles is a fairly common condition and is usually easy to recognise. (Cases 6 - 9). While the pulse is being palpated there suddenly occurs a rather smaller beat before its time. Immediately following this, there is a pause lengthened roughly in accordance with the time that the beat was premature, so that the next beat occurs at its normal time. Following the extra-systole, the heart is in a "refractory phase", as indeed it is after every beat, and during this period it does not respond to stimuli, or only to very marked/

marked stimuli. Thus the normal stimulus which follows close on the extra-systole, finds the heart in the "refractory period" and no contraction takes place until the next normal stimulus reaches the heart; in this way the long pause is explained.

Auscultation may enable one to be fairly certain of the condition, for during the long pause while the ventricle is awaiting the next stimulus, there is no systole, no heart sounds. Extra-systoles can generally be distinguished by the weak contraction, premature in time and followed by a powerful systole. It may also be possible to diagnose the condition as is done in complete heart-block by visual examination of the jugular waves and auscultation at the apex.

But besides the occasional extra-systole there are patients who have an extra-systole after every two, three or four beats.

Sometimes the extra-systole is so weak that it is not palpable at the wrist.

I have often found the occasional extra-systole very difficult to record. In the case of William Garvey I decided to take a tracing of his pulse as he had plenty of extra-systoles, but when I visited him two days later the irregularity had practically disappeared.

Mrs/

Mrs Scott whom I attended recently during an attack of influenza had a few extra-systoles, and though I took a good many tracings with a Dudgeon's Sphygmograph I never happened to record an extra-beat.

Miss Robertson (Case No.8) always has a few extra-systoles: one day I visited her and found two extra-systoles following each normal beat as regularly as clockwork. I hastened home for a sphygmograph but when I returned about five minutes later the regular irregularity had entirely disappeared.

I myself have occasional extra-systoles; for a week or two I do not notice a single example, and then in one evening I am conscious of one extra beat every hour or so.

When the extra-systole is not palpable at the wrist, it can be diagnosed with certainty by examining the apex-beat: during the pause in the pulse a small beat will be observed to take place at the apex, or it may be auscultated where the apex-beat is not palpable or visible. In heart-block there would be no beat visible or auscultable.

The commonest extra-systole is the ventricular, as has been described. Sometimes this extra systole is followed by the usual contraction instead of by a pause, the auricular stimulus stimulating the ventricle/

ventricle to contract again. The auricular contraction may take place at the same time as the ventricular extra-systole, in which case the auricle empties its blood into the jugular vein. Extra systoles originating in the auricle cannot be differentiated from ventricular extra systoles without special tracings. Extra-systoles originating in the auriculo-ventricular node cause the auricle and ventricle to contract before their normal time, but to contract together. The electro-cardiogram of Hush shows an auricle extra systole followed by a ventricular, also on the same electro-cardiogram, an auricular extra systole not followed by a ventricular (heart-block) (Case No.30).

The extra-systole is an example of an abnormality occurring in a perfectly healthy myocardium, in many cases. I myself have extra-systoles occasionally and yet my heart is perfectly fit. Extra-systoles are more common in elderly subjects. Many patients are not conscious of their presence - Mrs Foggo (Case 7), but Mrs Scott was very conscious of them. I myself notice them very markedly: I feel the short beat followed by a long pause practically simultaneously, and I then anxiously await the expected thumping beat which follows.

ETIOLOGY./

ETIOLOGY. In many cases various indulgences such as tobacco, alcohol, tea, coffee, a heavy meal, induce extra-systoles. In myself over-indulgence in tobacco (particularly in John Cotton's "Smyrna Mixture") is responsible: when I smoke in moderation I do not have extra-systoles. I always notice them when sitting quietly reading, never during exertion.

Various toxaemias are often responsible (Case 7) such as intestinal toxaemia, the metabolic poisons, rheumatic affection of the heart after the acute stage, infectious disease, etc. Over-excitable people are also subject. Mrs Foggo's was an interesting case, extra-systoles being very frequent and appearing along with an attack of urticaria. In less than twelve hours the urticaria disappeared and there were no more extra-systoles.

High blood-pressure and organic heart disease such as dilatation, myocarditis, coronary disease, etc. also lead up to extra-systole.

PROGNOSIS. Apart from organic disease of the heart prognosis is excellent. If a cause can be found, such as tobacco, a different sort of tobacco or a diminution in the amount smoked will probably cure the condition.

If there is no apparant cause after the patient has been advised as to possible causes, no notice should be taken of the irregularity provided there are no other symptoms or signs. The patient is advised to indulge in football or other exercise just as if there were no irregularity present.

Where there are other signs, such as heart-failure, the prognosis depends not on the irregularity but on the condition of the myocardium and the signs produced.

TREATMENT. The patient who comes to the doctor complaining of extra-systoles can generally give a clear account of his sensations when questioned. But I can well remember as a youth asking my doctor about my heart which he auscultated and as no extra-systole occurred during the auscultation, I was told my heart was perfectly/

perfectly normal. I went away unrelieved as I knew the irregularity was present and feared one day the heart would stop entirely. It was not till I studied medicine that I recognised I suffered from extra-systoles.

Consequently I am now always careful to get a description from the patient of his sensations and I have found the condition is sometimes easily diagnosed without any extra-systoles being heard during an interview.

To relieve the patient's mind is the first step and then to point out the possible predisposing causes.

In excitable people who are often much troubled when in bed, bromides are useful. Digitalis should not be used as it may induce rather than prevent extra-beats.

A healthy open-air life is often of assistance.

3. HEART BLOCK.

As has been detailed the normal contraction commences in the sino-auricular node and after the auricle contracts the stimulus passes along the auriculo-ventricular bundle to the ventricle. Clearly then if the stimulus cannot pass along the auriculo-ventricular bundle, the ventricular systole will not take place in the usual sequence of the cardiac cycle.

The stimulus may be only delayed in passing through the bundle: it may pass through the bundle occasionally but not always, or it may be entirely prevented from crossing, in which case the ventricle sets up a rhythm of its own, which is heart-block proper.

Tracings of the jugular vein are of great assistance. The auricular wave (a) is followed by a short period during which the stimulus is passing to the ventricle and then the ventricle contracts and a small wave (c) is produced by the carotid pulse. This a - c interval, according to Mackenzie (ibid. p.262) "is fairly constant lasting usually one-fifth of a second". If the a-c interval is prolonged this/

this must be due to loss of time between the auricular and the ventricular systole.

If the waves in the jugular vein which are visualised,are much faster than the rate of the ventricular systoles which are observed by auscultating at the apex, the two are not synchronising. The auricular waves may be two, three or four times as fast, one ventricular systole following a certain number of auricular beats, or they may be quite independent of the auricle.

A mere delay in the passage of the wave to the ventricle can scarcely be diagnosed without simultaneous tracings of the radial pulse and the jugular vein.

ETIOLOGY. The auriculo-ventricular bundle may be affected by inflammation acute or chronic, or there may be an actual growth in the bundle, e.g. a gumma.

As has been mentioned, rheumatic affections of the heart may lead to deposits of cells in the muscle, and this may occur in the auriculo-ventricular bundle to such an extent as to interfere with its function of conductivity.

In sclerotic changes of the heart, if the auriculo-/

auriculo-ventricular bundle is affected the conduction will be lessened in accordance with the amount of sclerosis.

Heart-block due to gummata may be entirely cured by suitable anti-syphilitic treatment. James Hush was supposed to have a gumma in the auriculo-ventricular bundle. His Wassermann was positive (Case 30).

SYMPTOMS. Patients with complete heart-block are not so incapacitated as to be unfit for mild exertion. The pulse may be only 40 and yet the patient be able to go about slowly. The field of cardiac response is of course limited and must not be over-taxed.

During the period between complete and incomplete heart block the ventricle may continue in diastole as if awaiting another stimulus from the auriculo-ventricular bundle. At such times and after the establishment of independent ventricular rhythm, the temporary stand-still of the ventricle may produce cerebral anaemia causing syncopal and epileptiform attacks.

When independent ventricular rhythm is established anything/

anything which causes rapid action of the heart, such as excitement, will only affect the auricles, the ventricles are either very slightly or not at all affected.

PROGNOSIS. Heart-block indicates that the myocardium is being invaded by disease.

The prognosis depends on the extent of the myocardial degeneration. This is guaged by the functional efficiency of the heart.

Cases with slow pulse (40) may live for many years, and in them one can be sure that the myocardium as a whole has escaped invasion, for partial heart-block does not in itself seriously affect the cardiac efficiency.

Where complete heart-block commences in an already impaired heart, the prognosis is of the worst.

During the change from incomplete to complete heart block, the tendency to syncopal attacks will not seriously endanger the patient with a good myocardium, but where this is degenerated the heart may not recover from an attack.

The whole prognosis depends on the condition of the myocardium.

Heart-block may occur in acute and subacute conditions and the patient may show no evidence of this after recovery.

TREATMENT. Just as the prognosis depends on the myocardial condition so the treatment turns on the same point. The heart-muscle must be given every chance to perform its functions and all strains avoided. Where the myocardium as a whole is degenerated, treatment is of little avail.

During the period of syncopal attacks the patient must be hept at rest in bed, the least additional strain on the heart being avoided.

The most hopeful cases are those of gummata in the auriculo-ventricular bundle and in all cases where the Wassermann is positive, anti-syphilitic treatment is advisable.

Where the patient is able to go about he must live within the limits of his strength.

Heart-block inducing cardiac failure calls for the usual method of treatment, digitalis not being contraindicated for it strengthens the myocardium and does not slow the ventricle.

4. PULSUS ALTERNANS.

This is a condition where every second ventricular beat is smaller than the previous beat, and produces a corresponding variation in the pulse. Indeed it may be present and not be detected by the palpation of the pulse at all. Its presence is ascertained by means of a pulse-tracing which shows the strength of the ventricular systoles. Even auscultation may fail to convince one that the systoles are not all of equal force.

The rhythm is perfectly regular, so that even should an extra-systole occur after every beat, the two conditions could not be confused since there would be a longer pause after the extra-systole than before it; while in pulsus alternans the diastolic periods are all equal. Where both conditions are present together, pulsus alternans becomes more marked after an extra-systole.

Apart from pulsus alternans per se, the condition appears in cases of auricular flutter and paroxysmal tachycardia, and where the pulsus alternans is found it is well to suspect auricular flutter.

ETIOLOGY AND PROGNOSIS. The pulsus alternans generally occurs in persons well advanced in years, and it is indicative of a degeneration of the myocardium, and herein lies its true significance.

When it is found in younger subjects the issue is usually fatal, as it occurs only in marked exhaustion of the heart.

When it occurs in paroxysmal tachycardia, the myocardium being healthy, the prognosis is quite satisfactory. But when it occurs with a normally active heart it indicates exhaustion which is the result of degenerative changes in the myocardium with resulting enfeeblement of the heart.

TREATMENT. Where the case is discovered early, before much degeneration of the myocardium, treatment may be satisfactory and the patient may be enabled to live for several years.

Rest is the great essential since the condition is really one of exhaustion. As the heart improves, the patient may be able to assume some activity, but this must always be carefully regulated or a rapidly fatal issue may ensue.

5. PAROXYSMAL TACHYCARDIA.

Mackenzie (Diseases of the Heart, 1918, p.251) defines paroxysmal tachycardia as "a term used to indicate the starting of the heart's contraction, in an abnormal place, with a sudden increase in the rate of the heart, and a sudden cessation of the rapidity on resumption of the normal rhythm".

The interesting part of paroxysmal tachycardia is just this suddenness of commencement and cessation, for herein it differs entirely from palpitation.

The cause of paroxysmal tachycardia is the sudden domination of the cardiac cycle by a series of abnormal contractions rapidly fired off from some part of the heart muscle other than the sino-auricular node.

Persons attacked may have a continuous attack for many days or even weeks, and yet sometimes it only lasts a very short time.

Short paroxysms last only a few minutes and may occur daily or several times a day. Long paroxysms last hours or days and are repeated possibly once a week or once a month.

The pulse rate is 150-200 or even more, and the/

the rate does not alter even on exertion, nor yet after rest.

When the attack commences the patient feels a curious fluttering sensation in the region of the heart. This fluttering consists of "soft and gentle movements, not rhythmical, but varying softly in intensity" (ibid) Apart from these sensations the patient soon finds himself quite unable for the slightest exertion, and very soon the signs of cardiac distress become apparent to everyone.

The astounding "cure" that takes place is the most remarkable feature of this disease. The dyspnoea and all the signs of cardiac failure rapidly disappear, the oedema, lividity and dilatation vanish like a mist and the patient becomes transformed into a different individual in the space of a few hours.

PROGNOSIS. Many people suffer from attacks of paroxysmal tachycardia for long periods, while others die in the first attack. Suppose the attack to commence in a heart which is already struggling to overcome the difficulty of a valvular lesion: the cardiac dilatation and heart failure are too much for the myocardium to cope with and the patient succumbs.

Should it however attack a healthy myocardium,/

myocardium, it can struggle through and the patient is none the worse. In such cases the heart does not dilate and herein lies the difference. Sometimes during an attack a pulse rate of 200 per minute may be kept going for as long as fourteen days night and day. In such cases the heart dilates and the patient suffers from cyanosis, the liver enlarges, etc. Yet when the paroxysm stops the heart quickly returns to normal and is just what it was before the paroxysm, the patient being again fit for a full day's work. Everything depends on the condition of the heart-muscle which is attacked.

Paroxysmal tachycardia may become permanent, in which case the duration of the life of the patient depends again on the condition of the myocardium, the better it is the longer will it be able to withstand the strain, though once the condition has become permanent the patient must succumb.

Where the attacks do not produce dilatation and the attacks do not last long, the patient may live for years. Unfortunately treatment does not prevent the recurrence of the attacks, so that the patient lives in constant fear of the next one.

Where cardiac dilatation and the signs of heart failure appear, such as dropsy and enlargement of the liver, the prognosis is not good.

TREATMENT. Obviously rest is indicated, and the patient does not need to be told this in well marked attacks. Curiously enough, rest has no effect in reducing the rate of the heart beat.

Cardiac dilatation has to be dealt with in the usual way.

The treatment may become that of auricular fibrillation.

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6. AURICULAR FIBRILLATION.

It is only within comparatively recent years that the condition of Fibrillation has been recognised, and one cannot but be filled with respect by the extensive studies of Sir James Mackenzie on this subject and with thankfulness for their wonderful results in relief of symptoms.

As a medical student I was taught nothing about auricular fibrillation and in Osler's "Principles and Practice of Medicine" of 1911 which I used, the subject is not even mentioned.

It was not till after the War, when I had the honour of being Professor Russell's Resident in the Infirmary that I met cases of auricular fibrillation and first became interested in the heart and began to recognise the vast importance of the heart muscle. It was not until a year after this that I got Mackenzie's "Diseases of the Heart" and discovered that the whole teaching points to the prior import of the heart muscle.

Mackenzie emphasises the great importance of auricular fibrillation with the statement that

"60/

"60 or 70 per cent of all cases of serious heart failure met with in practice owe the failure directly to this condition, or have the failure aggravated by its presence".

For a long time what took place in the heart during auricular fibrillation was not understood, but it is now recognised that instead of the auricle beating as a unit, i.e. all the muscle fibres contracting at one and the same time, the fibres contract according to their own pleasure, as it were, each one taking no interest in the time the others contract so that no auricular contraction takes place at all. The auricle remains in a relaxed condition though the individual fibres are constantly contracting.

The rate is greatly altered: it is usually very rapid, but in some rare cases it is slow: it varies from 40 to 130.

The rhythm is markedly affected. One would expect that no stimuli would pass down the auriculo-ventricular bundle and that the ventricle would set up its own rhythm, in this way heart-block being produced. But though the rate may be under 40 there is no evidence of heart-block, possibly because an occasional stimulus passes down the auriculo-ventricular bundle.

Thus/

Thus auricular-fibrillation is suspected when the finger touches the pulse. The extraordinary irregularity of the pulse frequently indicates at once what is taking place. The intervals between the beats are best recorded on a tracing where there is doubt. I thought that Case No.29 suffered from extra systoles until I took a tracing and found the intervals did not correspond with extra-systole intervals, though even the sphygmograph at first glance is like a series of extra-systoles.

Where there are many extra-systoles the two conditions are easily confused, but the characteristics of extra-systoles will soon become distinct from the great irregularity of fibrillation where no two beats are of exactly the same strength and no two intervals are the same.

The symptoms are those of a fluttering in the chest and a consciousness of the irregularity interspersed with violent beats.

ETIOLOGY./

ETIOLOGY. The causes of auricular fibrillation are particularly rheumatism, e.g. case of William Scott, and diminished nutrition of the heart muscles, so that it frequently affects the senile. Case of Mrs Rutherford (No.26) where the arterial-sclerosis was due to syphilis, and case of Mrs Reid (No.24) with some arterio-sclerosis and moderately high blood-pressure. (first attack). But apart from these definite pathological conditions it may occur after violent exertion, in which case it will be only temporary if it occurs in a healthy muscle. This is unusual, however, and generally speaking, auricular fibrillation only occurs in an inflamed, sclerosed or badly nourished myocardium. But Mackenzie (p.217) cites a most unusual case of a medical man who "at the age of fifty ran two hundred yards and was seized with an attack of auricular fibrillation which lasted two hours. This was ten years ago, and he is still well and actively engaged in his work".

It consequently follows, as these changes are not of a temporary nature, that fibrillation accompanies the patient throughout his lifetime although it may not be always present. At first it may last only for a minute or so, but the attacks lengthen/

lengthen in duration until the auricles fibrillate all the time, thus leading up to heart failure.

When fibrillation commences in an already embarrassed heart, e.g. a case of mitral-stenosis, heart failure will occur correspondingly sooner. (Case 29).

Since fibrillation commences in a damaged myocardium, the first effect will be to impair the ventricular efficiency still further, for with an irregularly acting ventricle the efficiency can never be equal to the normal condition. The more the ventricles themselves are affected by the original change, be it sclerosis or otherwise, the greater will be the impairment of the cardiac efficiency, and the less the ventricular myocardium is involved the fewer will be the symptoms and the more efficient will be the ability of the patient for his daily work.

DIAGNOSIS. In some cases the diagnosis is easy, e.g. the case of Mrs Reid (Case 24). As in her case, the extraordinary irregularity combined with the symptoms were quite characteristic.

But in slight cases the diagnosis is not so easy and/

and it is satisfactory to be able to make a certain diagnosis by means of the jugular tracing or the electrocardiogram. In the former, auricular waves are absent and the jugular pulse is of the ventricular type: the irregularity is also shown up. Little strings of small waves produced by the auricle are sometimes present. In the electrocardiogram the wave P due to the auricle is lacking.

In cases of mitral stenosis complicated by auricular fibrillation a presystolic murmur which may previously have been present, will disappear as the contractile power of the auricle is no longer strong enough to drive the blood through the narrowed valvular orifice. In these cases the suction action of the ventricle is the only force by which it is filled, thus the presystolic often becomes replaced by an early diastolic murmur.

PROGNOSIS. The prognosis in auricular fibrillation depends on the condition of the myocardium. The myocardium is always diseased, except for those rare cases where auricular fibrillation is brought on by overexertion and where the myocardium is healthy.

Consequently the first test will be to find out/

out how the heart responds to effort. In patients able to go about and even perform their daily work, the myocardium will be regarded as comparatively healthy and the prognosis will be good.
How well some patients respond to careful treatment is shown by the case of James Hush who suffered from auricular fibrillation. Though he suffered from both a pulmonary infarct and a pleurisy with effusion he made a good recovery. Mrs Pettigrew (Case No.28) is able to go about and do most of her own house-work.

There are many patients who while quite able to go about and do a little work, are not able to do what they previously did without symptoms. It is with such cases that one often experiences difficulty, as the patients are willing to undergo the discomfort of symptoms, and they continue to tax the reserve of their hearts to the utmost. Such cases will soon show signs of cardiac failure, probably the sooner the better, as then such patients realise that care is essential and willingly undergo what treatment is advised, and if the over-exertion has not been persisted in too long, the prognosis may yet be a favourable one. (Case of Mrs Oliver - No.25).

Patients who show signs of early heart failure are suffering from a diseased myocardium and the prognosis/

prognosis depends on the extent of that disease. This can only be ascertained by putting the patient under stringent treatment and time will show how he responds to this and his future will depend accordingly.

Patients whose hearts are attacked with fibrillation after their hearts have long been struggling with a serious valve lesion have little to look forward to but an early death. (Case 29). The very rapidity of the ventricular action will in itself diminish the efficiency of the heart and all that can be done is to reduce this rapidity so as to give the heart every possible chance: fortunately with that invaluable drug digitalis this is quite possible.

It might be said that the slower the heart the better the prognosis, as the tendency to dilatation increases with the rapidity of the heart.

Generally speaking the pronosis depends on the amount of response to treatment.

TREATMENT. As the heart is obviously struggling under difficulties the first line of treatment will be to reduce the work of the heart as far as possible by rest. Often patients continue to struggle along until signs of heart failure/

failure compel them to seek rest. In such cases the need also of rest in bed is recognised even by the patient.

But this in itself does not slow the action of the heart and this can be readily induced by the action of digitalis.

Digitalis used to be regarded as a drug to be given in small doses of five minims or so, but provided the effects of the drug are watched, very much larger doses may safely be given. I overdosed William Scott who suffered from auricular fibrillation until his pulse fell to 44 and became bigeminal in type, but as the drug was then stopped he quickly recovered from the poisoning. He received only fifteen minim doses four hourly, but for fourteen days continuously. He ought to have had his digitalis stopped for a day or two much sooner, but one is so delighted and amazed by its effect that the tendency often is to continue its use beyond the required amount.

Four daily doses of fifteen minims will generally give a decided result in a few days, but twice this amount is sometimes required and well stood.

I used Nativelle's Granules in the case of Mrs Reid and they proved most satisfactory.

When/

When the drug has to be continued over long periods the patient himself is generally the best judge of how much of the drug is required.

The reaction of fibrillating hearts to digitalis is wonderful.

7. AURICULAR FLUTTER.

Auricular flutter is best regarded as a further development of paroxysmal tachycardia. It is more or less uninteresting to the general practitioner since it can only be diagnosed with certainty by means of the electrocardiograph: for clinical purposes, however, it is assumed that any heart whose rate is more than two hundred beats per minute is in a state of auricular flutter.

In this condition the auricle contracts so rapidly that the ventricle is unable to keep pace with it, and although there is no disease of the band of His, a condition resembling that of heart-block is often shown in polygraphic tracings.
The auricular contraction may be as rapid as 350 per minute while the ventricle, although working at its utmost speed can seldom attain more than about 180 contractions per minute.

VII. FUNCTIONAL DISORDERS OF THE MYOCARDIUM.

1. TACHYCARDIA.

Everyone is aware of the effect of mental excitement on the heart. One is driving a car fairly fast, another car appears round a corner and an accident is narrowly averted: one becomes aware of the increased frequency of the heart-beat at once. "One's heart is in one's mouth" is a common expression to cover this phenomenon.

Examination of recruits for admission to the army, at Medical Boards, life-insurance examinations and the like often induce rapid pulse from excitement. The mere entrance of the doctor to examine a patient may cause a tachycardia, and this may be very persistent.

In such cases it is best to estimate the heart's efficiency by learning from the patient what duties he can perform and what hard work he is able to do.

This is in reality a normal condition exaggerated; more is required of the heart and it does more work.

Should however slight exertion bring on the same rapid cardiac action and dyspnoea which one would expect/

expect only after violent exertion, then it becomes apparent that the heart is deviating from the normal: the field of response of the heart to effort is distinctly reduced by the rapidity of the heart beat. The greater the amount of exercise the more rapid becomes the pulse and the more inefficient becomes the circulation, the patient meanwhile experiencing distress.

The causes of such a condition are enfeeblement of the myocardium from wasting diseases, and convalescence after serious illness; malnutrition of the heart in the various anaemias; affections of the myocardium and organic disease of the heart where the myocardium is so taxed that it can barely carry on a sufficient circulation.

Apart from periodic rapidity of the pulse there are conditions which induce a permanently rapid heart beat. The heart not only responds to effort by increased frequency of action, but beats rapidly while at rest in addition.

This condition is found in degenerations of the myocardium so that its power is impaired.

There too must be included the condition known as "soldier's heart" (see end of thesis) and other like conditions due often to times of great physical exertion and endurance.

The heart may be the seat of valvular disease where the myocardium has been fighting against an organic deficiency and has become exhausted in its efforts.

Other conditions where this phenomenon is elicited are pregnancy, exophthalmic goitre, exhausting diseases, neurotic cases and various poisons such as alcohol, toxaemia, etc.

The tachycardia in some cases comes in irregular paroxysmal attacks, such as attacks of palpitation and paroxysmal tachycardia.

Cases of palpitation have a pulse rate up to 150 or 160 per minute, the rhythm is usually regular and the attack gradually diminishes.

Cases of paroxysmal tachycardia have a pulse rate of perhaps 250 or 300, the rhythm is often irregular and the attacks stop very suddenly.

Palpitation occurs in every sort of heart condition from nervous excitement to organic disease of the heart. It is commonly found in the healthy hearts of women and is induced by fright, or reflexly from various organs in the body. Such attacks are generally of no significance though if they are very frequent they may easily exhaust the heart.

PROGNOSIS. Where the myocardium is healthy tachycardia is of small significance unless the attacks are very frequent, in which case exhaustion may ensue. The condition of the myocardium is estimated by the response to effort: where the reserve force is satisfactory the prognosis is good.

Where outside causes induce the tachycardia the prognosis depends on the condition of these causes, e.g. tuberculosis.

Cases of rapid tachycardia with organic heart disease usually suffer from exhaustion of the myocardium and the prognosis is very grave.

TREATMENT. Where there is nervous excitement the information given that there is no serious heart-disease will be a great mental relief to the patient: bromides may have to be used to act as sedative during day and induce sleep at night which is essential. In case No.34 all treatment was without avail.

Regular hours and regular exercise which must not be of too strenuous a nature will be of assistance. Rest must be taken during several hours of the day. Good/

Good nourishing diet with tepid or cold baths and avoidance of all excesses must be adhered to. A steady regular life is advisable. Tonics may be of assistance.

Tachycardia due to specific conditions such as oxophthalmic goitre and the like devolves into treatment of the causal condition.

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2. BRADYCARDIA.

Bradycardia is an uncommon condition but people with very slow heart rhythm (35 to 50) may live for years and be none the worse.

Often enough people who imagine they have bradycardia because the pulse is slow really suffer from some serious myocardial condition such as heart-block. I can well remember a very old man once telling me that he had a very slow pulse and that he expected to live so much the longer; but I was not surprised to hear a month or two later that he had died. It appears to be a popular belief that an exceptionally slow pulse is regarded as a natural compensation for an unusually active brain!

ETIOLOGY. Neurasthenia, Bright's disease with high blood-pressure, gout or arterial degeneration may be accompanied by bradycardia. Progressive slowing of the heart rate is notably seen in cases of diphtheria and often precedes a sudden fatal syncope.

PROGNOSIS. The condition of the myocardium indicates the prognosis. If this is healthy in every respect the rate of the ventricular rhythm/

rhythm matters little.

Where the cause is doubtful the response of the heart to exercise must be examined. Where the reserve force is satisfactory in all respects the prognosis is satisfactory.

TREATMENT.

The treatment in that of the etiological factor.

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3. HEART STRAIN.

The duty of the heart is to supply blood to the body. During exercise the heart is accelerated and the blood pressure rises: these are the natural results of exertion during which more blood is required than under conditions of rest, and the regulating mechanism increases the frequency of the heart, and the heart by its extra exertion raises the blood pressure.

The reserve force of the heart, as has been previously described, is enormous, and it is built to withstand the strains put upon it. Should the strain be excessive the heart becomes unable to cope with its duty, as described, and the result is not that the heart is strained, but that the blood-supply to the body fails.

The heart may be acutely dilated by anyone who holds his breath and at the same time endeavours to take a deep breath. This acute dilatation may be safely induced over and over again, and yet the heart is not strained.

If the heart dilates during exercise the dilatation is physiological and dangerous dilatation is prevented/

prevented by the fibrous pericardium.

Lewis (The Soldier's Heart, p.32) boldly declares, "In cases of so-called acute dilatation of the heart, which I have been called in to see and in whom I could find dilatation, the basal condition has always been an unrecognised paroxysm of tachycardia".

"Of thousands of patients we have drilled, a few score have fainted while at work, a largernnumber has fallen out exhausted; not a single man examined has shown dilatation of the heart either at the time or afterwards; not one has been the worse for his experience."

"If strain of the healthy heart exists it is no more than a curiosity".

Should there be some defect in the heart however, then an extra amount of very hard work will make an extra call on the reserve force of the heart and the unnoticed symptoms become exaggerated; the heart is said to have been strained by the extra effort.

Thus one might say that heart strain can only occur with an unhealthy myocardium, for it cannot occur with a healthy one.

Where then does dilatation take place?
"In a heart invaded by infective organisms or their products, with a consequent weakening of the myocardium"/

myocardium" (ibid p.37). It is this poisoning of the myocardium that is the all important factor in heart conditions. If a patient like Mrs C. or Mrs Morrison with a definite murmur and a healthy myocardium gets the myocardium infected, then the valve lesion becomes a serious embarrassment and danger.

Where a perfectly normal heart is infected, the myocardium is weakened, and then the heart will dilate: the dilatation is commonly ascribed to heart strain.

In the same way a continuously high blood pressure, as in the case of Mrs Lillie who was a bed-patient, will not embarrass the heart so long as the myocardium remains healthy, though Mrs Lillie latterly began to show signs of cardiac weakness owing to sclerosis of the valves and heart.

Often the cause of dilatation is difficult to find out. I never found the cause in the case of Miss Inglis (No.39)

4. THE "SOLDIER'S HEART".

The term "Soldier's Heart" has become known to most of us since the War, but the phrase "the Irritable Heart of Soldiers" was used long before the War and indicates the same condition.

The condition known as "Soldier's Heart" is perfectly common in civil life: it might be termed "general ill health". In other words, although the patient may complain of breathlessness on slight exertion, precordial pain, palpitation, giddiness or faintness, it does not follow that the heart is the main organ at fault. These symptoms might quite well be induced by pulmonary tuberculosis.
At a Medical Board in Atholl Crescent a pensioner, William Hastie, with marked physical signs of pulmonary tuberculosis came up for examination: before his lungs had been examined by an expert he had for long been classified as a case of D.A.H. (disordered action of the heart.)

Thus the "Soldier's Heart" being a somewhat indefinite term and not being confined to soldier patients, Lewis has adopted the phrase "the Effort Syndrome" to cover the symptoms and signs known as the/

the "soldier's heart".

"When I use the term "effort syndrome", I have in mind the symptoms and signs which follow exercise in health: but I believe that I recognise the same or a very similar group of symptoms and signs in a large class of patients in ill-health. In patients of this class, if no signs of disease are anywhere discovered, I say that they suffer from the "effort syndrome". (The Soldier's Heart and the Effort Syndrome. 1918. Thos. Lewis, page 4).

Patients with the "soldier's heart" suffer from the "effort syndrome". This phrase has the advantage that it stands for a group of symptoms and signs only, while the "soldier's heart", "irritable heart", "D.A.H.", "neuro-muscular asthenia", etc., all of which are more or less synonymous, appear to have established a diagnosis. There are never any signs of structural disease of the heart.

S Y M P T O M S.

The symptoms associated with the "soldier's heart" are those usually associated with heart disease.

BREATHLESSNESS./

Breathlessness. The breathlessness associated with the soldier's heart is not accompanied by cyanosis and the rapid breathing is a nervous phenomenon: it is not persistent during sleep: sometimes it may be due to a relative acidity of the blood. (Murray. B.M.J. Dec. 14, 1918, p.651) or to an excessive irritability of the respiratory reflex (Mackenzie. B.M.J. April, 1920, p.533).

Pain. This is often precordial but may be in small areas here and there over the chest or abdomen. Mackenzie describes how a stimulus arising in a carious tooth produces pain, not only in the tooth but in the skin of the cheek, which may be used to explain these areas of hyperaesthesia of the skin and muscles. (The Soldier's Heart and War Neurosis. B.M.J. April 10th and 17th, 1920, p.491.)
Murray explains the pain by a relative acidosis of the blood. "The cardiac pain, whether accompanied by hyperaesthesia or not, is merely the distress signal of an organ compelled to work in spite of a food supply, at least altered chemically". (ibid p.652)

Fatigue and Exhaustion. This is exceptionally easily induced, very light exertion being enough. Sometimes there is tremor of the hands and shaking of the legs.

Palpitation. This is felt most during exercise and is also often brought on by excitement. It does not indicate any myocardial change but is merely an increase of the normal rhythm.

Murray explains this by the "changed character of the body fluids interfering with function" Again "We know from physiological and from clinical experience in different diseases that certain compounds stimulate one or other divisions of the autonomic nervous system. The case I review in this paper is one instance where the vagus is under such control. The rule, as we know, is for stimulation of the sympathetic. I only point out that such is the action of the various abnormal circulating fluids". (Ibid. B.M.J. p.652).

Mackenzie explains the increased frequency by increased irritability of the sympathetic reflex just as/

as a carious tooth induces pain not only in the tooth but in certain skin areas as well. (Ibid.)

Fainting. This is fairly common in "soldier's heart" cases and occurs while the man is standing still or during slight exertion. Usually the patient can give no indication of the cause. The blood pressure is reduced and the pulse rate is slowed.

The aetiology is a vagus nerve stimulation and may be explained in the same way as the sympathetic nerve stimulation.

The prognosis is quite satisfactory. The patient is given a few days from his work after which there will probably be no more fainting fits.

Giddiness. This usually occurs after exercise and is due to the blood pressure which has been raised by the exercise falling rapidly on cessation of work, cerebral anaemia being produced.

The prognosis is satisfactory.

Other Symptoms. An infinity of other symptoms are met with such as myotactic irritability of the chest muscles, excessive/

excessive sweating, headache, mottling of the hands, numbness of the fingers, etc.

Pulse rate. It is the pulse rate that is usually used as an indication of the "soldier's heart". The rate is 90 to 100 and may be even faster.

Not only is the rate rapid but exertion raises the rate more than it would in a normal individual. Various exercises may be used such as running up a flight of steps, etc. The exercise always used at the Military Pensions Boards in Atholl Crescent is to get the patient to extend his arms above the head and then to bend down and touch the toes with the knees extended. I generally try to get men to do this ten times very rapidly. In myself this raises the pulse from 72 to 88 and the rate returns to normal in less than one minute. In soldier's heart cases the rate starting at 90 will be raised to 120 or more, and the original rate is not resumed in less than a minute: it may take two or three minutes before the pulse falls to 90 again.

Breathlessness is quite common even after quite slight exertion. The combination of increased frequency with breathlessness is described in the phrase,/

phrase, diminished exercise tolerance.

Various alterations in rhythm are found, e.g. Pensioner George Anderson's pulse was 120. After slight exertion it rose to 144 and after a minute it fell to 100.

Blood Pressure.

The blood pressure rises excessively just as the pulse rate does.

Thus the symptoms of the "soldier's heart" are those of an "exaggerated physiological response to exercise, and the degree of effort needed to call them forth is an inverse measure of Morbidity". (Lewis "The Soldier's Heart", p.26).

There is nothing remarkable in the symptoms: they are the result of great effort found in a healthy man.

AETIOLOGY OF THE SOLDIER'S HEART.

"In a careful inquiry into the origin of ill health in over 2000 soldiers I found that in the case of over 80 per cent the first onset of their illness began/

began with some complaint of an infectious nature, such as measles, influenza, trench-fever, typhoid fever, malaria, dysentery, or P.U.O. In other cases one could gather that the onset was due to an ill-defined illness suspiciously like an infection". (The Soldier's Heart and War Neurosis. B.M.J. April 17, 1920. Sir James Mackenzie.)

"The dominant etiological factor in the clinical histories of soldiers complaining of the "effort syndrome" is infection of one kind or another. Rheumatic fever easily occupies the first place, pyrexia of unknown origin or influenza occupies the second place and dysentery comes third". (The Soldier's Heart and the Effort Syndrome", page 33. Thomas Lewis). See table page 84.

There should be no doubt that an infective agent of some sort is the cause of the condition generally known as the "soldier's heart". When the heart is invaded by such an agent the myocardium is weakened. The whole contention of this thesis is that the weakening of the myocardium is of the first import.

Heart strain has often been said to be the cause of the "soldier's heart". This has already been discussed. The reason is in many cases at least, not difficult to find. Men had adopted in civil life/

life sedentary occupations because they did not find themselves fit for heavy work. They did not know how to increase their cardiac reserve (see III) and adopted light occupations. When the War necessitated their enlistment, and drills were commenced, they showed evidence of the "effort syndrome".

There are some other etiological factors which come into play but these are small, they include syphilis, tobacco, alcohol, hyperthyroidism, gas poisoning, nervous derangements. The effects of these have been dealt with elsewhere, their etiology need only be mentioned as causing the symptoms of the "soldier's heart" where there is no structural heart change.

As has been mentioned Murray emphasises the importance of relative acidosis of the blood. (The Common Factor in Disordered Action of the Heart. L.C. Murray. B.M.J. Dec.14, 1918, p.650). Exhaustion he described as being synonymous with malnutrition of the cells owing to the chemical alteration of their necessary food. He also considers supra-renal insufficiency as a possible factor.

Mackenzie/

Mackenzie in two long and very interesting papers in the B.M.J. (April 10 and 17, 1920) on "The Soldier's Heart and War Neurosis" points out how stimuli from an organ are felt not only at that organ but elsewhere as well, e.g. certain diseases of abdominal organs produce an alteration in the sensibility of the skin. In the same way, "In the limitation of the response to effort (shown by breathlessness easily produced) and in the increased rate of the heart we get evidences of increased susceptibility to stimulation on the part of the organ. A heart in this condition of undue excitability, compelled to do the same amount of work as it did when the health was good, will become sooner or later exhausted, and so we will find a series of symptoms arise which are the outcome of exhaustion".

"While the over-excitability of the heart is brought about mainly by a disturbance affecting its nervous mechanism, in all probability the same injurious influence affects the myocardium itself, so that the efficiency of the heart may be impaired". (page 533).

This puts the condition of the "soldier's heart" in a nutshell. It is an "undue excitability", a condition of general ill-health where the excitability of the heart is well marked.

PROGNOSIS. The prognosis depends on the exercise tolerance of the individual. This cannot satisfactorily be tested by a few bending exercises lasting perhaps half a minute. A series of exercises or drills should be resorted to, commencing with exercises of a few minutes duration and gradually extending the length of these and their severity. In general practice this is difficult but the results may be estimated by the distance walked along a road at an average walking speed. An examination of the patient should be made after the exercise, for example, by the doctor immediately the patient reaches his house after a walk of the prescribed distance.

The symptoms have to be largely discarded, and definite evidence only taken, such as breathlessness, fatigue, giddiness, etc.

The cases with a history of rheumatic fever are the most disappointing.

The general tendency is towards recovery however.

TREATMENT. A regular, well ordered life is the first indication with open air exercise, regular meal-hours and cheerful surroundings.

Definite/

Definite exercises in the open air are begun and generally extended so that the patient sees the improvement and is encouraged.

Over-indulgence of all sorts is avoided, constipation, dental caries and the like, are attended to. Tonics often help the patient but digitalis is avoided.

VIII. CONCLUSION.

This thesis does not purport to bring to light any startling new theories or indeed any new matter at all. It endeavours to examine all heart conditions from the standpoint of the heart muscle and it aims at emphasising the great importance of the myocardium in cardiac disease.

Modern research has shown conclusively that whatever kind of heart-lesion may be present, the real factor of importance is the state of the heart muscle. A healthy myocardium can compensate for every defect.

The actual condition of the heart muscle is the one guide to treatment and the only sure index in prognosis.

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1. A case of disease of the mitral valve with a healthy myocardium.

Mrs Morrison, (aet.35)
Lilybank, Currie.

This patient came to consult me about some rheumatic pains on 27th November, 1920.

During the course of the examination I asked her whether her heart ever troubled her. She replied in the negative but added that she had always been told that she had "heart disease".

The moment the stethoscope touched the chest one was conscious of a loud very definite mitral systolic bruit, indeed it is audible all over the chest.

History.

When nine or ten years of age, she had a very serious illness which her doctor called "influenza". She remembers being in bed for about three months with this and that she did not return to school for six months at least. She remembers also wearing a porous plaster over her heart for a year. She is certain it was never called rheumatic fever.

She/

She returned to see me a few days later and said she remembered having a very expensive medicine during her illness. I got her to write to her mother about this medicine, and the mother wrote saying the medicine was salicin.

This history certainly points towards a rheumatic fever rather than influenza.

In April 1919, she had a quinsy which was "very bad". The abscess did not burst for fourteen days and she was severely ill. Ever since childhood she has had "sore throats", though this was her first quinsy. Her tonsils had apparently always been very large, for she says she was advised by every doctor who saw them, to have the tonsils enucleated. When they inflamed even slightly they "almost choked" her. She feared the operation and states she had them needled, treated with electricity, caustic applications and various other methods of treatment without avail. The quinsy however gave her a fright and in July 1919 she had the tonsils removed at last, and the operation has been a very satisfactory one, for there is now no evidence of any tonsillar tissue remaining and her general health has been much improved.

Heart. Apart from the loud mitral systolic bruit there is nothing to note about the cordiac condition. There are no symptoms whatever. She can walk up hills with dyspnoca. She has climbed the Black Hill (Pentlands) and was not dyspnoeic. She does all her own house-work and washing and never considers her heart at all.

I made her run up a steep flight up stairs thrice running and there was no dyspnoca palpitation or other symptom: the pulse rate before this exercise was 80, after 96.

I examined her exercise tolerance on other occasions and always found the pulse very slightly accelerated.

The blood pressure varied from 120 to 150 m.m.

The urine had no abnormal constitutents.

When the patient lay down, the bruit became remarkably musical in character.

2. Case of valvular disease associated with a healthy myocardium.

Mr C -

This gentleman, a well known Princes Street personality came to me one day saying he felt perfectly fit, that he had been passed as a first class life by more than one Life Insurance Company, but that he wanted an "overhaul" for his own satisfaction.

I expected to find him perfectly fit, for he told me something of his habits of life. He does a complete set of Müller's exercises daily and is a great believer in these: he is quite certain that they have kept him as fit as he is at present. He takes a walk every morning for the sake of his dog. He is careful about his diet and in every way leads a regular life, always thinking of his fitness before everying: e.g. while walking he told me he often holds his breath and sees how far he can walk without taking a breath and he thinks this exercise is good for him by increasing the chest capacity.

I started the examination by taking his blood pressure and was astonished to find it 250 m.m. It varied/

varied round about this figure and 210 m.m.

So I examined his heart and found a mitral systolic mumur not influenced by position with however no dilatation: apex in normal position. There was no aortic bruit.

His exercise tolerance was excellent as would be expected in a man who exercised himself as Mr C. always has done.

I was assured by the patient that there could be nothing wrong with his heart, and I told him nothing about the bruit, but described how his blood pressure was above normal. I advised him not to hold his breath while walking and to avoid meat as far as possible.

He came back to me a month later, complaining of a "cold in the head" for which I gave him a nasal irrigator and glycothymoline.

His B.P. was 210 so I gave him a course of Pot Iodidi gr.X t.i.d. and after four or five days the B.P. was 190 so I advised him to stop the bottle as it was having very little effect.

The heart condition was as before. Vessels somewhat thickened and the exercise tolerance excellent. He was still enjoying his exercises and feeling very well except for the "cold in the head".

The urine was normal in every respect.

3. Case of mitral and aortic diseases with a healthy myocardium.

A.J. Black. (aet. 18)

This youth was an inmate of the Borstall Institution (Prison) for boys.

He was a source of considerable annoyance to the staff for it was known that he had valvular disease of the heart and whenever he felt inclined for a rest he would report sick with a pain over the heart or some such complaint and immediately be relieved of his duties.

He had loud and definite murmurs which were recorded in all his papers, viz:- presystolic and systolic mitral and aortic systolic murmurs.

He was however perfectly fit and played football and indulged in all the activities along with the other boys.

4. A case of mitral regurgitation with a healthy myocardium.

Mrs Anderson, (aet. 36)
Lower Polmont.

I attended this women for a cough she complained of, and found a few fine ronchi from which she rapidly recovered.

There was a very definite mitral systolic murmur so I asked her whether she had ever had rheumatic fever or had ever been troubled with her heart. She replied that she "had had none o' thae things" and that her "heart was right enough".

She was a stout, good-natured woman with a large family and a rather lazy husband. She did the entire work of the family, including the washing of all the clothes. A remarkably hard-working woman, she was kept at her work all day and had no difficulty.

She told me that she "had never been ill" and that the others"took so much looking after that she had no time to be ill".

I attended various members of the family for some seven months, but she was always well and hearty.

5. Case of healthy myocardium associated with a mitral diastolic murmur.

Henry Fowler. (aet. 31)
Polmont.

Patient's mother has a "weak heart" but she is 72. One of his brothers is occasionally short of breath, but patient does not know whether it is due to his heart or not. Four brothers and two sisters are all well.

I first saw patient in June 1920 when I was struck by his healthy appearance. He was well nourished and had walked a mile and a half to the Surgery very fast as he was anxious to catch a certain train.

I was astonished to find a long murmur, my notes liken it to "water rushing over a fall, rising in intensity until systole takes place: very loud and interesting to listen to". I could make out no systolic bruit.

The heart was hypertrophied, the left border lying along the nipple line. The apex beat was forcible and hit against one's hand when palpating the/

the chest, but no aortic nor mitral systolic murmur was ever made out.

The interesting point however, is that a man with a long diastole murmur should have such a healthy myocardium. His occupation was a Railway Relief Agent which meant that he acted as temporary station master at various stations and did a lot of odd work of a more or less skilled nature. He had to go in to Falkirk from Polmont daily and he states that he was generally late for the train and had a full mile of uphill walking to get to the station. He could do this perfectly well. He had walked a mile and a half when I saw him first and was as fit as anyone. He stated he could walk three miles fast without symptoms as he had frequently to do this, as he often lost his train at Polmont and had to walk to Falkirk.

6. A Case of extra-systoles with a healthy myocardium.

H. Maitland Moir. (aet. 32) (the author)

Extra-systoles have always been present so far as can be remembered, certainly since the age of nineteen.

A doctor was once asked about them. He listened to a few beats and of course heard no extra-systole as they were very occasional. He said there was nothing the matter with the heart and the patient went away dissatisfied.

While studying medicine later, the condition of extra-systoles was recognised. No further notice was taken of them.

Undoubtedly tobacco in excess increases the number of extra-systoles, particularly John Cottons "Smyrna" mixture an Egyptian blend.

Normally an extra-systole is noticed about once a week, on some days, two or three or more might be noticed, but this is exceptional. There might be a period of several weeks without any being noticed.

An extra-systole commonly occurs while sitting still reading or working at books, etc. Immediately a/

a blank is noticed as if something were going to burst in the chest - my attention is very markedly attracted. Then follows the well known "thump" and everything goes along normally again.

It is evidently the extra-beat that attracts my attention with a feeling as if something had happened.

There are no symptoms. Heart normal in size and function. All exercises can be performed perfectly. Healthy open air life is the routine commenced with a few physical exercises used more to produce physical warmth prior to a cold bath in the morning.

B.P. 130 no vessel thickening.

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7. Case of temporary extra-systoles associated with an attack of Urticaria.

Mrs William Foggo,
13 Kinauld.

On February 27th I was called out after midnight to see a patient who was stated to be recovering from influenza and who had suddenly developed a rash on the legs.

I found the patient looking very well and very alarmed about the rash, which was a simple urticaria all round the thighs and extending half way up the abdomen. She had well marked dermographia.

On listening at the apex I was astonished to hear an extra-systole as I placed the stethoscope on the skin. I looked up at the patient but she appeared quite unconscious of it. After another two or three beats there was another and I asked her sharply if she felt it. She was astonished and asked what I meant. I explained to her, but she said she had never felt any extra-beats.

The/

The extra-systoles occurred quite irregularly sometimes after two beats, sometimes after four or five. There was never more than one at a time. They were not followed by a very violent beat as the pause was fairly short, the pulse being fairly rapid. (98) T. 97·5.

As it was after midnight I did not feel inclined to return and take a pulse tracing. I gave her Dover's powder gr. 12 and said I would call next day.

On the following morning I found the urticaria entirely absent - the patient had had a sound sleep and felt quite recovered.

Before starting with the pulse tracing I auscultated the apex and was astonished to find the extra-systoles quite absent. I listened for a long time and did not hear a single extra-beat.

On subsequent visits no extra-systoles were ever heard.

8. A Case of extra-systoles with an only moderately healthy myocardium whose reserve could be improved.

Miss Robertson. (aet. 65)

I first attended this lady in 1919 when she had an attack of influenza which lasted a long time. The heart was little affected. She made a good recovery.

Her father died of heart disease and she had always had a great fear of it as she knew her heart was affected in someway as she could feel it beating irregularly. She first noticed this irregularity in "middle age."

She feels at times " as if she were going to collapse" and occasionally she has "giddy turns". When she feels unpleasant sensations such as these, she sits down and after a while feels better and continues her house-work.

Dyspnoea troubles her considerably on exertion. If she goes upstairs or if she walks up a hill the breathlessness comes on.

Palpitation/

Palpitation comes on after exertion - the "heart begins to go". A sudden interruption or a fright produced, for example, by the door bell suddenly ringing loudly, or a sudden noise, bring on palpitation.

She describes minutely how on two widely separated dates she took a very hot bath. On both occasions she became very ill and was certain she was going to die. She remembers her feelings than very acutely and will never forget them. The sensation was exactly the same on both occasions and was described by herself as being "exactly like birds fluttering with their wings going on as hard as they could inside her". She felt that she could not lie down and scarcely knew what she did at the time.

The symptoms seem to point to fibrillation of the auricles. It is interesting to note that a second hot bath on a subsequent occasion produced precisely similar symptoms. She did not think that the same would happen again, but she will never take another hot bath.

Her occupation consists of house-work and she can do this quite easily apart from the occasional attacks of palpitation and giddiness.

She scarcely ever goes out of doors, as she does not/

not like the trouble of getting her out-door things on and prefers the comfort of her kitchen.

I told her that I thought her heart could be improved by a little regular exercise in the open air, commencing gradually and increasing the amount regularly and slowly. She said she was quite content as she was: that her heart did not trouble her enough to warrant the bother required in going out or in exercises. She is averse to taking medicines and says she would rather do without them.

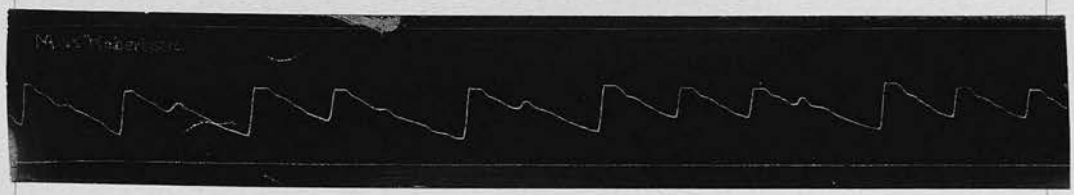
The personal history has been a good one. She remembers as a child having bronchitis every winter and thinks that she grew very fast, but there is no history of "growing-pains". She had measles and whooping-cough together and apart from these she has never had a doctor, as the bronchitis never troubled her after the age of 20. I remember when I visited her at the influenzal attack, she said I was the first doctor she had had for many many years.

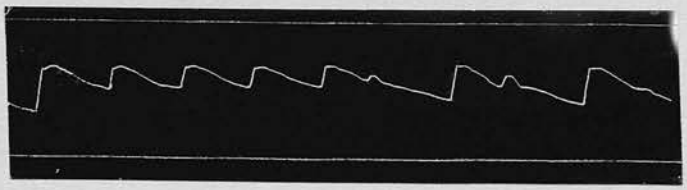
The extra-systoles are always present and occur quite irregularly. I remember visiting her one evening and I found two extra systoles occuring quite continuously after each normal beat. She lives not far from my house so I rushed back after listening at the apex for a minute or so, got a sphygomograph and was back in Miss Robertston's house in a few minutes./

minutes. The extra-systoles had however become quite irregular again in the interval.

Her pulse generally runs about 70 or 80. After a bending exercise test of moderate severity the rate rises to 90 or 95 without any dyspnoea or other symptoms.

I am convinced that Miss Robertson could greatly benefit her heart if she would take some graduated exercises. She walks about in the kitchen and to all intents and purposes lives an absolutely sedentary life, so that she never increases her cardiac reserve in the least.





9. A case of extra-systoles first experienced after an attack of influenza.

Mrs Gray, (aet. 36)
Shieldhill.

I first attended Mrs Gray during an attack of influenza which was a protracted illness. She was in bed for three weeks and for a long time afterwards she was very unfit.

From various notes I have gathered the following together:-

Well after the influenza the heart condition was as follows:- no dilitation: no murmurs: occasional extra-systoles. She was very conscious of the extra-beats and said very often there were a string of little beats followed by a thumping over the heart.

She complained of dyspnoea on exertion. Pre-cordial pain, Giddiness and faintness. Pulse was generally about 100-120. When asked to bend a few times to touch her toes she managed to bend down five times but became so dyspnoeic that she had to sit down and felt faint. Pulse rose about 20 beats or rather more after very slight exertion.

She/

She is very anemic and loses a lot of blood during menstruation. I managed to reduce this somewhat and improved her condition a little.

Her occupation was house-work: she seldom went out or took any exercise in the open, not feeling fit for it.

The interesting point is that she is very conscious of the extra-systoles and that she had never had these before the influenzal attack.

I tried her with rest, with graduated exercises, with wine, with various medicines etc. She slowly improved on the whole. The mixture which did her most good she states, is one containing Tincture of Digitalis m.v. Ferri et Quin Cit gr.v. and Tr. Nuc. Vom. m.v.

I left off attending her at her own request as she did not want to pay a large doctor's bill: she was improved but not recovered.

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10. Case of Senile Heart.

David Winton. (aet. 80) Malleny Mill.

I first saw Winton in September 1920. He was then confined to bed although he had been walking about not long before. He suffered from marked rheumatoid arthritis which fortunately did not pain him at all. He was unfit for any exertion and had to be helped in all his movements, becoming dyspnoeic if he exerted himself.

Although there was no bronchitis there was always a lot of secretion in the trachea and large tubes which he coughed up, although this tired him very much and he preferred to let it gurgle away in his chest to the exertion of spitting it out. This noise made ausculation of the chest very difficult.

He was an extraordinarily cheerful old man and had always a smile and a word of thanks or praise for his doctor.

In October for the first time he had difficulty in urinating, and later, for two or three weeks his son passed catheters: fortunately the son is an intelligent man/

man and was scrupulously aseptic in his methods. The prostate was enlarged: a large rubber catheter was easily passed. After a few weeks of catheter life, Winton passed his urine voluntarily. The urine was frequently examined and contained no abnormal constituents.

In his younger days patient had been in the Regular Army and had always been healthy and after leaving the army, a hark-working good-living man.

I never found the heart dilated although it may have been so, for there was emphysema and I never felt the apex beat all the time I attended him. The musculature was always weak.

Ausculation was a difficult matter as a rule, owing to the aforementioned gingling but on occasions this was less. I never heard any bruits in the heart.

The heart sounds were always very feeble. It was always perfectly regular.

The pulse was generally 80 - 90. The vessels were not very thickened or tortuous.

The blood-pressure ranged from 120 - 150 taken on many occasions.

Towards the end, the pulse became very weak, and I took up a sphygmograph on two or three occasions and could not get a single extra-systole. The heart sounds became inaudible and he eventually died quietly on January 16th, 1921.

11. A Case of Senile Heart almost without symptoms.

William Culbert. (aet.69) Currie.

Excellent Family History. States he has never been ill except that occasionally he had not "such a good appetite".

Heart: No dilitation so far as can be made out, but the chest is fairly emphesematous. No endocarditis or murmurs. Heart sounds feeble.

Pulse 120 and very dyspnoeic after walking a mile to the Surgery. Respirations 40. He will not recognise that he is not fit to walk so far and persists in coming occasionally for his insurance certificate, a distance of nearly a mile. Vessels markedly thickened and tortuous. B.P. 120.

Stated on one occasion that he had spat bloot and asked if sputum could be examined! No tubercle bacilli were found.

Urine contains no albumin or other abnormal constituents. He gets up once at night to pass urine./

urine.

He spends most of his time sitting by the fire-side and resents the idea of taking a little daily exercise. He likes to have a good walk when he can be bothered to leave his house. He gets easily tired, and when he walks far he has to sit down every little while for a rest. He is convinced this does him more good than a little exercise regularly.

I have not given him any medicines except on one occasion when he complained of cough, when I gave him a stimulant expectorant(although there was no bronchitis) and Pot Iodidi.

12. A Case of Senile Heart with extra-systoles.

Patrick Garvie (aet. 69) Polmont district.

This old man looked more like 79 than 69, but was of cheerful disposition.

His only complaint was of not being able to get up sputum, first thingin the morning. There was no bronchitis.

He was a feeble old man, fairly frail and thin but took his food well. He was out of bed several hours each day as a rule, though unfit for any exertion.

There was no definite cardiac dilatation nor any definite bruits, but he had extra-systoles which did not trouble him at all, he had one about every five or six beats.

His vessels were markedly thickened and tortuous. The blood pressure was usually about 200. The urine contained no abnormal constituents.

13. A Case of Senile Heart with valvular disease.

John Liddle. (aet. 71) Polmont district.

This old man kept house for himself but he was not fit for exertion and took life very easily. He managed to be out bed most of the day however.

His vessels were thickened and the blood pressure was usually about 180.

His heart had aortic systolic and mitral systolic murmurs. It was difficult to tell whether the heart was dilated as he had bronchitis and emphysema. He managed to get about with marvellously few symptoms.

14. Case of senile heart with chronic myocarditis and heart-failure.

Mr Goseman. (aet. 80) Polmont.

This was a remarkably bright-spirited and contented old man who looked as if he could live for several ********* years. He was a Parish patient and never wanted to bother his doctor.

I found him suffering from very marked dropsy both upper and lower limbs being remarkably swollen. He was quite accustomed to the swelling and I think would almost have regretted its removal.

I first visited him in August 1920 and found he had been at work up to the previous autumn. He enjoyed life though almost entirely bed-ridden. He invariably slept well.

He had mitral systolic and aortic systolic murmurs with cardiac dilatation. The vessels were greatly thickened and tortuous.

He suffered from all the signs of heart failure.

This case is merely mentioned as a contrast.

15. A case of dilatation due to toxaemia of the myocardium associated with nephritis.

Ernest Pearson. (aet. 6) Kinauld, Currie.

Mrs Pearson brought along her daughter to see me on January 9th, 1921, saying that she was not well, but without definite symptoms. I noticed the eyes a little puffy and found a trace of albumin in the urine and a little oedema of the legs and feet. She made a rapid recovery.

On February 1st, just as his sister was recovering, Ernest Pearson developed an oedema of face and legs. I found five grms of albumin per litre. The chart shows how greatly the albumin increased.

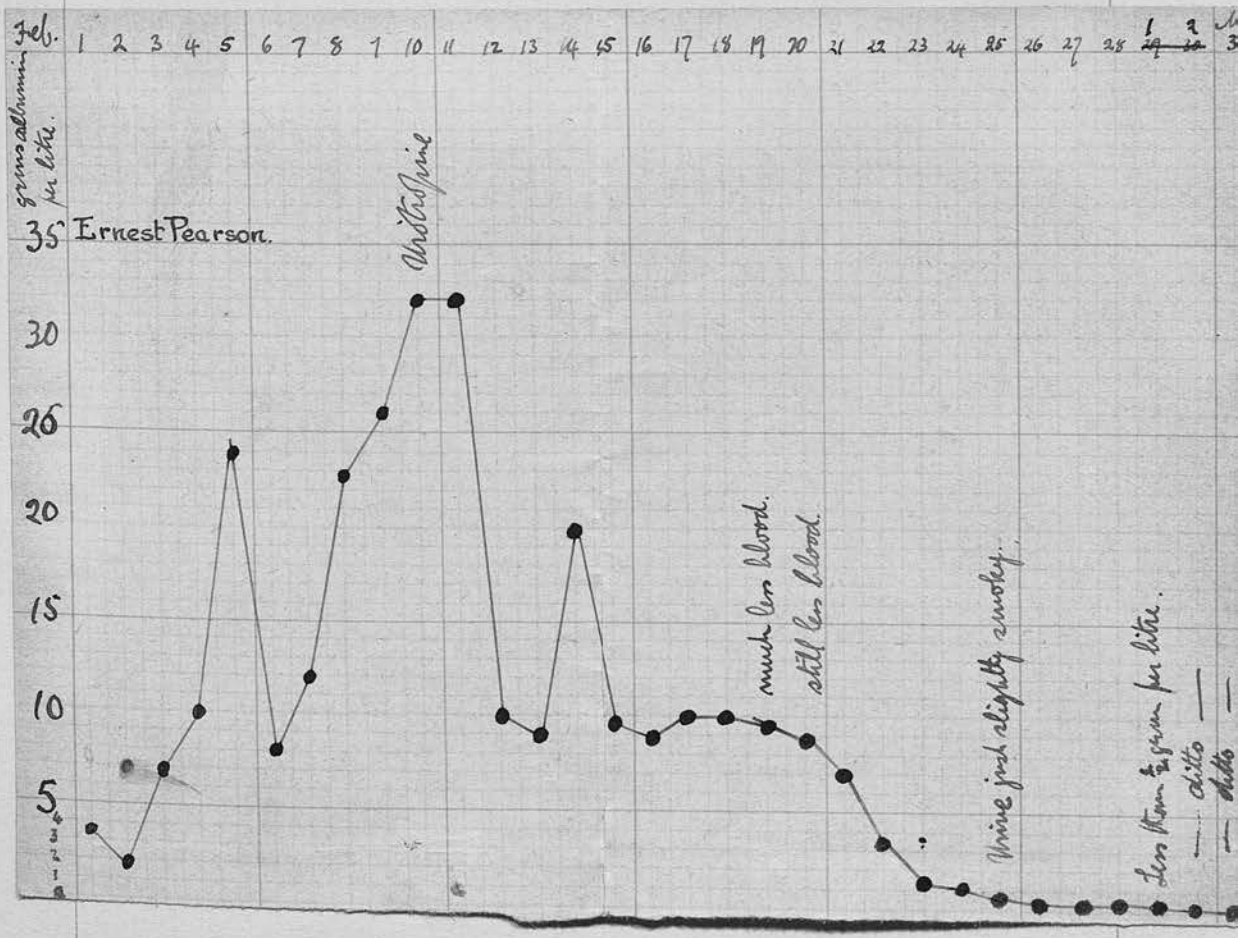
On Feb. 9th, I feared he would certainly die, and as the albumin was increasing daily I took him off the Pot Citratis mixture and gave him Hexamine with Acid Sod. Phos. This was given on the evening of the 9th and throughout the 10th and stopped on the 11th and the Pot. Citratis reverted to. The albumin fell with an extraordinary rush. All this time the urine was so deeply blood-coloured that it might have been half urine/

urine and half blood to all appearance.

Heart: He developed a mitral regurgitant bruit, a bruit evidently indicating a toxaemia of the heart muscle. The apex was at first an inch outside the nipple line. The pulse was never very rapid. The regurgitant bruit still persists.

Treatment was entirely for his urinary condition, hot packs and pilocarpine proved very satisfactory. He was very patient but only made a slow and fair recovery.

Time abolished the mitral bruit.



16. Case of dilatation of the heart of a child.

James Forrester. (aet. 8) Polmont.

This patient's father brought him to me one evening saying the child was not well, that he would not play as formerly and he did not know what to make of him.

I examined his lungs and then the heart which I found dilated three quarters of an inch beyond the nipple-line.

The father could give no suggestion as to the etiology.

He was put to bed and in a couple of days the dilatation had disappeared and the boy was very anxious to be allowed out of bed. I foolishly allowed him up next day and when I visited the following day the heart was again dilated.

This time I was much more careful and he was kept in bed ten days, although latterly he jumped about in bed and got very tired of his confinement. He was eventually allowed up for fifteen minutes and the time was gradually extended until he was allowed to/

to run about outside and to return to school.

I examined his heart every second day and made certain that the dilatation did not reappear.

I saw his father repeatedly for some couple of months after this and he said the boy was giving no further trouble and is as active as ever.

17. A case of cardiac dilatation due to chlorosis.

Elisabeth Harper. (aet. 15) 4 Kinauld Buildings.

Chlorosis - Cardiac Dilatation - Mitral and Aortic systolic murmurs.

Had Scarlet Fever and Measles.

Works in the finishing dept. of the Tannery. Constanting standing, polishing and staining of skins.

End of December 1920 her father brought her: paleness and dyspnoea on exertion: slight cardiac dilatation mitral and aortic systolic.

Continued at her work until holidays. Then kept in bed for eight days by which time the dilatation had gone. Pulse generally about 100 when she walked sharply, acclerated by bending and takes some little time to return to normal.

Bruits now absent. Heart in normal position.

19.1.21. Looks ever so much better now. P104 after bending 6 times 125. Returns to 102 within a minute. No dilatation. No bruits. Still very pale.

Was/

Was having cod liver oil with malt at outset. Given a tonic for a while with good result.

I examined her again in March and found the aortic and mitral systolic murmurs still present, but faint and only definite after exercise. I made out no dilatation, the apex-beat was easily palpable a full inch inside the nipple line.

Pulse after walking to Surgery 100. After bending to touch the toes ten times, rapidly, pulse rose to 120 without dyspnoea.

It is difficult to get her parents to give her a satisfactory course of treatment as they are not too well off and require the money their daughter makes at the tannery. They think that so long as she is able to work that she ought to do so.

It is disappointing to find the murmurs have returned after their being abolished by treatment.

18. Case of chronic myocarditis.

John Young. (aet.63) Maddiston.

This was a big powerful man with a healthy appetite and a tendency to adiposity.

He was a miner and at first I though him rather a lazy one though he always maintained he was anxious to work.

His one complaint was giddiness and I rather scouted his idea that this was serious.

After I had attended him for a week or so without taking much interest in him, I got a telephone message from the local chemist to say that Young was receiving medicine from another doctor as well as myself. I went to see Young next day and he told me he had called the other doctor in two days before as he was not benefiting under my treatment, and he really wanted to get well.

I decided to examine him thoroughly and started with his heart which to my astonishment and chagrin I found/

found dilated to the nipple-line, with a faint mitral systolic murmur. Pulse 90 while at rest in bed. Blood pressure 150.

I decided to take him more seriously. I treated him with Nativelles granules and with Tincture of Digitalis, with various tonics, with rest in bed over periods of a couple of weeks and with some open air exercise, when this failed and I could not reduce the dilatation of the heart.

He began to complain of headaches and occasionally had "faint turns" and precordial pain was added to his giddiness which persisted throughout. I found that trinitrin tablets, one hundredth of a grain relieved his headaches.

I must have attended Young for four months and I left him on the return of his own doctor as I found him on my first visit.

I repeatedly urged both the patient and his wife to agree to hospital treatment, but they both refused persistently. He was not so very ill as to make them afraid.

The urine was repeatedlt examined and I never found albumin or other abnormal constituents.

19. Case of chronic myocardial disease with valvular disease and failure of reserve followed by improvement after graduated exercise.

Henry Smith. (aet. 26) Wiers' Buildings, Currie.

Loud blowing systolic murmur with a rough pre-systolic murmur.

Heart in 5th Interspace just outside nipple-line.

He has never had Rheumatic Fever. He had no diseases except measles when a child. Always been healthy and used to be a football player before the War.

At Peebles while in the Army, he was racing and thinks he strained his heart as he has "never been right since".

Last November he spat blood and was admitted to the R.I.E. (for three weeks). Haemoptysis lasted 24 hours. He left his work at the mill.

Parents, brother and sisters etc., healthy: no heart trouble in the family.

He was discharged from Army in April 1916 with V.D.H. He is now quite unfit for his former occup-

ation/

occupation at the Mill and has built a shed where he proposes to do boot-repairing etc.

Lungs: Some congestion L. Base, none R.

Urine: No abnormal constituents.

Blood vessels not thickened. B.P. 120.

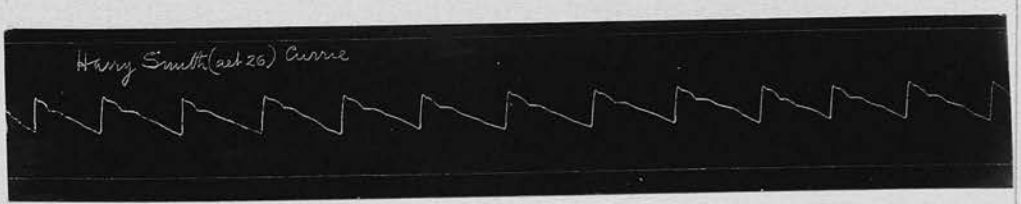
Pulse 64.

Walked here tonight and had to stop three times for a rest (in one mile). When he arrived he was dyspnoeic and after resting 10 minutes still took long deep breaths.

Advised to take $\frac{1}{4}$ mile walks with a rest half way. No med. To return in a week.

This man is now doing well. He is an example of a careful liver, not anxious to over-strain his heart by excessive exercise. He has started the occupation of boot-repairing which suits him very well and is gradually increasing his cardiac reserve. He takes regular exercise and can now do much more than formerly.

One trembles to think of the result of an attack of pneumonia or some other infection attacking a myocardium so burdened as this.



20. Case of valvular disease associated with myocarditis aggravated by persistent over-exertion.

Henry Cullen. (aet. 39) Labourer,
Main Street, Balerno.

I only had charge of this man for a week or two during the absence of his doctor, but can well remember the astonishment with which I saw him one day nearly a mile from his house when I imagined he was strictly confined to the house.

His heart showed the following murmurs:-
Mitral diastolic and short presystolic murmurs.
Long blowing systolic mitral, propagated into the axilla.
Short aortic systolic.
Long blowing aortic diastolic propagated down the sternum.

There was oedema of the feet and legs, but this was not well marked when I first saw him. There was oedema of both bases and enlargement of the liver. He spat a little blood occasionally.

The pulse while patient was at rest in bed was 84.

The/

The vessels were not markedly thickened and the blood pressure was 125 m.m.

Urine 1014 acid Albumin was generally about half grm. to the litre.

His only complaint was the dyspnoea which he found very trying and a feeling of weakness. He never had any pain or giddiness.

One can imagine my surprise when I saw this man in the afternoon nearly a mile from his house where he had walked and he had also to walk back again.

I reasoned with him next day and explained that he must not do so much walking. But it was impossible to convince him. He believed that he only suffered from weakness and that he would soon "work this off" and that he would get worse is he stayed in the house.

I used to spend some time explaining to both him and his wife that he was getting worse, but Cullen did not believe it and his wife and I could not convince him. It was most aggravating to see the man going steadily down hill and he was sent to the Infirmary where he eventually died not very long after.

His family history was excellent and the patient himself stated that he had never had a doctor attending to him in his life before. He attributed his condition to hard work. His previously healthy life made him impatient with illness.

21. Case of post-influenzal toxaemia of the myocardium.

Mrs C. Currie.

This lady had an influenzal attack of mild severity of which she took little notice. I was not called in to attend her.

After the attack she went for a two mile walk and was tired on getting back. Suddenly everything went round and round and things disappeared before her. She knew she was about to faint and at once sat down and felt better.

She got up and almost at once felt she was about to faint again: she called to a maid who happened to be at hand and was assisted by the maid who was alarmed about her and got her to bed at once.

I arrived soon after and found the heart dilated to the nipple line and a definite mitral systolic bruit audible. I thought this was probably an organic murmur as she said she had had a "bad heart".

I told her she must stay in bed.

Some hours later I found the dilatation less obvious/

obvious but the systolic bruit as before.

She made a good recovery and the mitral bruit disappeared entirely.

For a month subsequently she felt easily tired.

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22. Case of post-influenzal myocarditis.

Miss Baird. (aet. 42) Craigs, Polmont.

I attended Miss Baird's brother in June 1919: he had an attack of influenzal pneumonia. As he and his sister lived in a small one-roomed house with a very small window, it was inevitable that Miss Baird should contract influenza: she also developed pneumonia. She made a good recovery though her brother nearly died.

As Miss Baird was a club patient in a large and busy practice I left off attending her much too soon, telling her to leave a message at the Call-house near by if she wanted me any day. I unfortunately did not look her up again.

About a month later I was in the Polmont district again and visited Miss Baird. I found her in a wretched condition. She said she had never got over the pneumonia and that she was getting worse. Ever since her illness she had been short of breath on the slightest/

slightest exertion and very giddy. She had had many syncopal attacks and she complained of pain over her heart.

I examined her heart and was not astonished to find it dilated: the apex being half an inch outside the nipple-line. I could make out no mitral or other bruit. Pulse at rest 100. B.P. 115.

I told her she must rest in bed, but she refused absolutely to stay in bed all day, saying she would rest as much as she could. She said there was no one else to attend to her brother's food.

I gave her digitalis with a "tonic bottle" with Nux Vomica and Ferri et Quin. Cit.

Two days later the heart was in the same position but she stated she felt much better and that she was resting as much as she could. The pulse was 88.

Unfortunately I left Polmont at this time.

The finger-nails all showed a very marked ridge which corresponded with the date of the pneumonia.

Ever since the pneumonia her hair had come out in handfuls - she showed me a bag the size of a football packed with the hair that had come out.

I look back on this case with regret, as I fear I was responsible for the condition in which I found her. I handed her over to her own doctor who would doubtless get her well again in time.

23. A case of myocarditis due to influenza.

Mrs Alexander Stenhouse. (aet. 37) Currie.

This patient contracted influenza and called in no doctor. While confined to bed she thought more of the dirty condition of her house than of herself and consequently she got up and started to clean up the house after two days in bed. She felt very weak and ill but got up again next day and felt short of breath and had to go back to bed.

I visited her and found the temperature 99°2. I kept her in bed for four days and she got up for a little on the fifth, but was glad to get back to bed after being up only about twenty minutes.

The next day I examined her and found the heart dilated nearly to the nipple-line with a pulse rate of 72 quite regular. She bent slowly up and down ten times and the pulse rose to 100 and she became very dyspnoeic. The pulse was thin and weak. There were no cardiac bruits. She complained of palpitation if anything frightened her and that she got dyspnoeic on exertion.

I/

I explained to her that she was not fit to attend to the house-work, but she said she could not be in bed and see the house dirty, but agreed to take as much rest as possible.

I visited her a few days later and found the apex an inch outside the nipple-line: there were no murmurs, but the second sound was accentrated.

The pulse 100 at rest. She managed to bend slowly up and down eight times, by which time she was very dyspnoeic. The heart was going about the rate of 150 per min, but was slowed down and I counted 150 beats in half a minute. After a minute of interval the pulse had slowed down to 63 per minute.

I told her most emphatically that she really must stay in bed, but I am sure she never did it.

So her condition dragged on and she slowly improved in spite of herself, though the heart remained dilated. Later, as she saw she was not improving she went to her mother's house, and was kept in bed for about ten days. After this she made a good recovery.

24. A first attack of auricular fibrillation followed by a satisfactory recovery.

Mrs David Reid (aet 53) Redding Muirhead, Polmont.

I was called in to see this patient on the 31st of August 1920 when she told me she had been suddenly attacked with severe dyspnoea, palpitation and pain over the heart, also a curious feeling of fluttering inside the chest. She could suggest nothing that might have brought on the attack except that she drank a lot of tea and was generally fairly constipated.

Her previous health had been excellent until quite recently when she began to feel "done" and have headaches, which symptoms alarmed her very much, she had been so accustomed to good health.

The attack had come on suddenly on August 27th in the morning when she got out of bed. The "heart suddenly went flying like a wee hammer inside her" and she experienced the symptoms mentioned.
She improved a little during the next two days but called me in on the 31st.

I found the heart dilated but there were no signs of heart failure. The pulse was quite uncountable/

uncountable and utterly irregular. Auscultation at the apex and examination of the jugular veins confirmed the diagnosis which palpation of the radial had suggested. The vessels were thickened and the blood pressure 150 mm.

Dr Hunter of Falkirk had been telling me of Nativelle's Granules which he had found very satisfactory and I decided to use them in this case which would prove their efficacy quite soon, or otherwise. As this was the first time I had used these I started with one three times a day.

2. XI. Appears very much better. Heart beats about 120 and pulse still very irregular. Patient expressed herself as feeling infinitely easier.

4. XI. Heart and pulse regular, and reduced to 68 per minute. "Granules" reduced to one per diem. Patient expressed herself as "still feeling very "done" if she exerted herself" but she was quite obviously a different woman to what she had been four days ago and was very conscious of the fact.

5. XI. Walked nearly quarter of a mile to the Surgery saying that she felt so much better she had decided to pay me a visit. She experienced no discomfort. The B.P. was 165, Pulse 65.

7. XI. Walked to Surgery. B.P. 135. Pulse 60, so the "granules" were stopped entirely.

9. XI. Walked as before. B.P. 160. Pulse 64 so I told her she would probable need no more of the "granules" but always to have some near by in case of emergency.

10. XI. Still improving and walking further without discomfort. Pulse 65.

11. XI. Walked two miles. Expressed herself as feeling so well that she would not require any further medical attention.

I visited this woman occasionally later and she did well and had no subsequent attacks.

25. A case of auricular fibrillation.

Mrs Oliver (aet 45) 85 Claypath, Durham.

Family History.

Father died 62 (cause unknown) M. 75 and well.
2 Brothers 2 Sisters all well. No children.
No abortions. Been married 12 years.
Husband healthy.

History. She worked in Y.M.C.A. hut in Durham for 3½ years during the War from 8 a.m. till 10 p.m. "pretty hard work", constant work, washing dishes and floors and generally keeping the place clean. She thinks she worked far too hard and has never got over it.

Symptoms. She had, a year ago, dyspnoea on ascending stairs and as this got worse, she gave up the Y.M.C.A. work much against her will soon after. Palpitation came on later. She first noticed swelling of the feet 3 weeks ago.

When I first saw her she was in a wretched condition, trying to do the house work and utterly unfit for it. There was very marked oedema of the legs and/

and feet. I told her she must stay in bed and she very reluctantly consented, saying that she would not stay more than a couple of days at most.

It was absolutely impossible to count the pulse, so fast and irregular was it.

I was acting as Locum Tenens for a doctor in Durham and all medicines were given from the doctor's surgery. I gave her ten minim doses of tincture of digitalis to be taken every four hours night and day.

When I called next morning I found her pulse practically as bad as ever, though she had taken five doses of the bottle I gave her.

I went back to the Surgery, poured out the Tincture of digitalis and made up a bottle with infusion of digitalis: next day she was little better so I poured that away too and ordered fresh tincture from Messrs Sarsfield near by.

Next day the change was remarkable. The pulse was much steadier and could be counted quite well - rate 140 and she told me she felt so much better that she was going to get up and attend to the house whatever I said about it.

Two days later I found her polishing the grate. P. 120 partly due to the exertion. She stated she was able to do a full day's work and that she had no dyspnoea on exertion at all. Oedema practically absent.

Unfortunately I left Durham at this time and was not able to follow her subsequent history.

26. A case of auricular fibrillation due to syphilitic thickening of the coronary arteries.

Mrs Margaret Wood Rutherford, 18 Viewcraig Row.

Age 64.

Occupation: Housework.

Complaint: Painful tightening in the region of the heart.

General. Patient has a drawn, worried expression. Face cyanosed. She is very restless and moves hands nervously: markedly short of breath. While lying in bed sits up in bed propped with pillows. Number of respirations 40 but by next morning reduced to 36 and to 32 by the evening. It took five days treatment to reduce resps. to 20.

Marked oedema.

Family History. Husband died at 51 in Morningside Asylum of "Paralysis of the Brain". Patient had 9 children, three of whom are dead - two of pneumonia in infancy, the other lately in childbirth. The remaining 3 boys and 3 girls/

girls are healthy except for the eldest son who suffers from "heart disease". Patient's mother died of "consumption": father of "shock".

Previous Health. Had Rheumatic Fever about 11 years ago. Since then she has always been troubled with her heart. She has been in the Infirmary several times - the heart-pain is very great at times, then patient may be quite well for weeks until the pain recurs. She was in the Infirmary in June and was sent to the Convalescent Home, Corstorphine, in the beginning of August: then she had another "bad turn" and was sent back to the Infirmary.

Wassermann Reaction is positive.

Circulatory System. Pulse on admission very rapid and irregular. Soon after admission she became very breathless and was given Three Spirits at 10.10 p.m., and a hypo. of Strophanthus 1/100 gr. at 10.15 p.m. and was relieved for half an hour. The Strophanthus had to be repeated at 2.15 a.m. and 6.30 and patient became much easier. After this she received several strophanthus injections.

Heart/

Heart dilated.

Loud blowing mitral systolic.

Aortic Systolic murmur.

Vessels very thickened: B.P. 180.

Rate 60-80 under digitalis.

Rhythm very irregular: a great many beats do not reach the pulse.

This was a case of auricular fibrillation confirmed by electro-cardiogram. She was in Professor Russell's Ward while I happened to be house physician and the notes were taken at that time.

The origin of the fibrillation was undoubtedly syphilis having produced a thickening of the vessels and in this way affecting the heart.

I gave her intravenous injections.

Digitalis reduced the pulse rate from an extremely rapid and irregular one to 42, and it then became fairly but not quite regular.

Throughout her stay in the Infirmary there were pus and albumin in the urine.

She was discharged "relieved".

27. A case of auricular fibrillation with myocarditis and valvular disease.

William Scott. (Aet 52) 12 East Thomas St., Edin.

Complaint: shortness of breath and palpitation.

Family History: Father died of Rheumatic Fever at 46. Mother died of "Senile Decay" at 86. Patient is married. He has 9 children - seven are alive, two dead, one at the age of three, of appendicitis, the other at the age of 10 days.

Social Condition: Patient lives with his wife and family in a three roomed house, five of his children are working so that he has plenty to live upon. He smokes regularly - about an ounce per week. He is a total abstainer now.

Previous Health. Had measles when a child. He had "rheumatism" in 1913. He remembers being 22 weeks in bed.

General./

General. Patient was previously under my care as Resident in Professor Russell's Wards (28) for ten weeks. I had a Wassermann test done which was negative. An electro-cardiograph showed marked auricular fibrillation.

Patient on admission to Ward 26 (Prof. Meakin's) in which I was Resident, was very dyspnoeic. Resps.42. He could not lie down but had to be propped up in bed, he gasped for breath. Colour: pale with marked cyanosis of the nose and lips. He coughed a good deal.

Circulatory System. Complaint: dyspnoea, occasional palpitation. No pain.

On admission the pulse at the wrist gave no indication of the heart speed. The irregularity was exceedingly marked, whole strings of little beats following each other in rapid succession. The electrocardiograph showed some heart-block.

Marked oedema of feet and legs and scrotum and lower part of back and anterior abdominal wall. Some fluid in the abdomen.

Heart. Apex beat not palpable, but is situated in the 6th and 7th spaces in the anterior axillary line.

Markedly irregular as indicated. Sounds distant. First sound feeble - second accentuated.

B.P. 120.

Urine. - Albumin $\frac{1}{8}$ - 1 grm. per litre.

Respiratory System. Cheyne-Stokes breathing. On admission there was pain in the left axilla and coarse friction was audible over this area. The chest is well-formed and moves well with respiration.

Some crepitations in both lungs - no dulness.

Alimentary System. Tongue moist but furred. Teeth in filthy condition.

He was so oedematous that the first aim was the reduction of this. He was given:-

Pot. Acetatis ℥ss
Tr. Digitalis ℥ss
Diuretin ℥ss
Decoc Scoparii ad ℥vi
℥ss. four hourly.

On this mixture he passed on successive days 50, 100 and 146 ounces of urine per diem and the oedema diminished.

The pulse on admission was charted as being 116 but this was only a rough estimate as the irregularity was extreme and the rate so fast that it was impossible/

impossible to count the pulse.

In fourteen days the pulse was regular and the rate was 44 per min. The digitalis was stopped. As the rate rose again the digitalis was re-commenced two days later and in five days it became bigeminal and the digitalis was at once stopped. After two days it was commenced again in five minim doses.

As the pulse rate rose whenever the digitalis was stopped I decided to try him with strophanthus. He was given five minims of the Tincture (not the 1914 pharmacopoea) and he started to vomit and continued vomiting the whole day: I thought he would certainly die, but he recovered and the digitalis was resumed again, but his pulse rate was not kept down by five minim doses, so he was increased to ten minim doses.

He existed for a week or two after this though his coughing and breathing were a trial to the whole ward.

28. A case of auricular fibrillation with myocarditis.

Mrs Pettigrew (aet 66) Balerno.

Family History. Patient's mother and one brother both died of heart disease. Patient has never had rheumatic fever or rheumatism. She had two attacks of pneumonia; one, thirty years ago and the second twenty years ago. Otherwise she has enjoyed excellent health.

Two years ago a blood clot caused gangrene of the foot and the limb was amputated above the knee. It healed quickly. She walks with crutches, not being able to afford an artificial limb. There was no apparent cause for the blood clotting.

Her heart has troubled her "for years", she does not know when the trouble commenced.

The present symptoms are mainly shortness of breath on exertion. She says she cannot stoop at all as this brings on dyspnoea at once. She moves slowly about the house on crutches and is able only for the very slightest exertion. Occasionally she walks to her daughter's house which is about fifty yards distant, and this produces dyspnoea and she always "feels very shaky for a while after", but this passes/

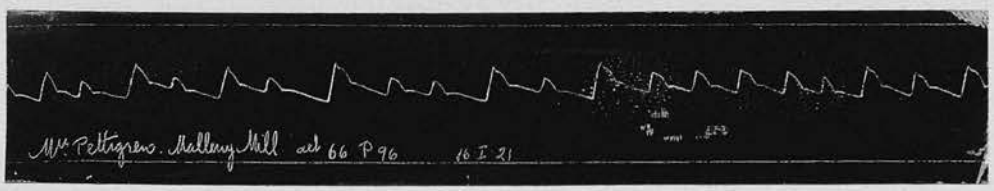
passes off after a rest.

She manages to do some house work and sleeps fairly well.

The apex was situated a full inch outside the nipple line. There is just a suspicion of an aortic systolic murmur. The pulse varied somewhat, I have taken it after patient has sat still for a while and found it 135, but on other days it has been as low as 90.

The sphygmograph indicates the condition of the pulse perhaps better than I can describe it. When I first examined her I thought she had extra-systoles and when I first examined the sphygmograph I thought she had a bigeminal pulse, but the irregularity can only be that of auricular fibrillation.

She refuses to take digitalis as she says she can get along well enough without it!



29. Case of auricular fibrillation with myocardial degeneration following on valvular disease, where the heart had been persistently over-exerted.

Mr Love. (aet 85) Dean Park Farm, Balerno.

This was the case of a remarkable old farmer: his pulse was so irregular that at first I thought he must be suffering from auricular fibrillation, and I discovered he had strings of extra-systoles, sometimes as many as eight at a time, rather difficult to count, and I thought the condition one of extra-systoles until the sphygmograph showed they were not such.

History. He had always been a particularly healthy man stated never to have had any serious illness. He always had spoken of his heart as being an irregular one, so far back as he could remember: he called it "the dot and carry one" pulse.

He had an attack of pneumonia in March 1920 from which he was not expected to recover. He was kept reluctantly in the house for six months.

In August 1921 he sailed from Leith to Aberdeen and went to the Agricultural Show, coming back by train./

train.

On February 19th he went to the Market in Edinburgh and to the Veterinary Hospital where he had a horse recovering from an operation. He walked about a good deal, travelling by train from Balerno to Edinburgh and back.

He was ill next day and in a week he was to all intents and purposes dead, with Cheyne-Stokes respirations, oedema, pulmonary congestion, etc. He existed for nearly a week in this condition.

This illustrates the marvellous activity of a man unwilling to give in, although not fit to do what he did.

When I saw him he had marked oedema, dyspnoea, pulmonary congestion, albuminuria, an enormous and dilated heart. The valvular condition was difficult to make out as the dyspnoea made it difficult for him to hold his breath, but I subsequently made certain that he had double aortic and double mitral murmurs. It is scarcely credible that a man in this condition should have been walking all over Edinburgh less than two days before. For long his family had endeavoured to get him to reduce his exertions, but he was a headstrong old man and would take advice from none.

He sank rapidly. In less than a week I was called up at midnight as he could not be got to bed. He/

He imagined he was away from home and wanted, or rather demanded, to get home at once. I got him to bed by telling him I had to listen to his heart and had to examine his feet. He at once agreed and took off all his clothes and got into bed. I examined him and went off well-pleased. Just as I was leaving some one came down to say he had all his clothes on again! I gave him gr. 18 of Pot. Bromide which had little or no effect and a restless night was passed.

Next night I gave him Pot. Brom. gr.x, Sod. Brom. gr.x and Syr. Chloral ℥ℓ . It was of no avail. Next night I gave him two tablets of Dial. which did splendidly and he sleps quite well and was rather better next day.

Each day one expected him to live only a few hours and yet he managed to live on. Curiously, the oedema disappeared practically entirely from his feet towards the end. For days he lived in a semi-conscious condition, latterly being unable to swallow. He also developed hydro-thorax. The respirations were Cheyne-Stokes in type.

This case was difficult to treat efficiently as he had attacks of retching immediately after the Digitalis was started. He had had attacks of the same sort before and probably the retching was not entirely due to the Digitalis but to chronic venous congestion. I gave him mx of the Tincture three times/

times daily. I increased this and the retching increased so I did not feel justified in giving him more. After a week it was stopped entirely for a day. The digitalis reduced the number of little beats at first, though later they became just as frequent in spite of digitalis. His apex came in to four and three-quarter inches from the mid sternal line.

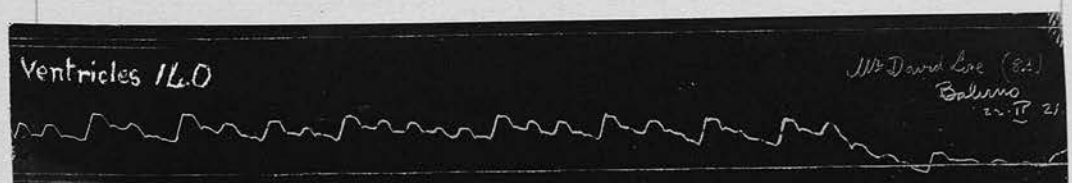
When I saw the urine on the first day it was very acid and loaded with urates. Large doses of Pot. Citratis put an end to this in twenty-four hours and I think he was relieved, certainly mentally, for he examined his urine.

Influsion of Scoparium seemed to help his oedema somewhat, as the oedema of the feet diminished, but the pulmonary oedema increased.

There can be no doubt that he would have lived much longer had he been more judicious in his living.

His Blood-pressure was always about 130-150.

He must have had aortic stenosis for a long time to account for the enlargement, for the apex at first was two and a half inches beyond the nipple-line. He certainly had cardio-sclerosis as indicated by the aortic and mitral valves. His blood vessels were not so thickened as one would have expected.



30. A case of partial Heart-block probably due to Syphilis.

James Hush. (aet.64) 68 Grassmarket, Edinburgh.
Occupation: Carter.

Complaint. Dyspnoea.

History of Present Illness. About April 1917 patient began to get breathless on exertion and had a "choked up" feeling in his throat. The breathlessness came on fairly suddenly and patient attributed it to his heavy working. He thinks that he strained his heart while lifting some heavy biscuit boxes. He did not however feel anything happen to his heart at the time.

In April 1919 he first experienced precordial pain.

In Sept. 1919 patient was admitted to Ward 28 (Prof. Russell) where he remained a month. He was only out of hospital a short time when he was re-admitted, to Ward 26.

Circulatory System. Pulse 50-70 after digitalis. B.P. 118. Vessels tortuous but not markedly thickened. No varicose veins. Well marked oedema of feet and legs. No clubbing of finger. No venous pulse in/

in neck. Epigastric pulsation well marked.

Cardiac. Apex beat cannot be seen but can be felt below the nipple 4½" from the middle line in the fifth interspace.

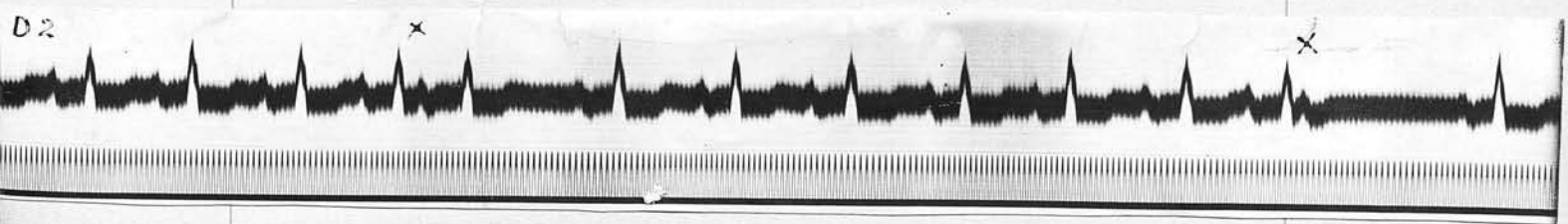
Percussion. Right border one inch to right of middle line at 3rd rib. Left border 4½ inches from middle line.

Auscultation. Sounds difficult to hear at apex, first sound replaced by a systolic murmur. No presystolic. No aortic bruit.

Later he developed a pulmonary Infarct and Pleurisy with effusion, from which he entirely recovered. He also recovered from his cardiac condition sufficiently to be sent to the convalescent home, though his future prospects were not very bright.

Hush had a positive Wassermann reaction and this is of interest in so far that the electrocardiograph shows an auricular extra-systole followed by a ventricular, and near the end of the graph an auricular extra-systole not followed by a ventricular.

I treated his syphilis with novarsenobenzol suspended in a preparation of Burroughs Wellcome & Co., and it was injected deep into the gluteal muscles. He complained of little pain following the injections.



31. A case of healthy myocardium following rheumatic fever.

Mrs David Kirkpatrick (aet 36) Balerno.

Mrs Kirkpatrick had a definite attack of rheumatic fever when 24 years of age. She had very severe pain in all her joints. She was "all wrapped up in cotton wool" and had "salicylates to take". She states that she was very ill and that she was in bed for a month or two.

Following on the attack and for at least two years she had very severe pain in all her joints, particularly the knees. Now she has no pain in her joints.

Heart:- The apex is $2\frac{3}{4}$ inches from the midsternal line. Pulse 70 at rest, increased to 90 after severe bending exercise rapidly carried out.

She can do anything in the way of exercise. She bicycles a lot and can cover long distances and cycle up hill without dyspnoea. She never has symptoms connected with her heart.

This was a particularly fit looking young woman. It is satisfactory to meet a case of completely healthy myocardium after rheumatic fever.

32. Case of Subacute Rheumatism.

26.11.21. Peter Peat (aet 19). Kinauld, Currie.

Family history is free of rheumatism.

When about 14 he had rheumatic fever and St Vitus Dance which lasted about six months.

In Jan. 1921 he had an attack of Influenza which laid him up a week or ten days. Since then he has always had some pains in the joints and back. He went to his work and worked for a month and began to feel less and less fit. If he lifted anything with his left hand"he seemed to forget all about it and drop the article". Pains in the joints and back got rather worse. One day he was much worse and had to stay in bed. I found him suffering from severe pains in the joints and the pulse 85 without obvious dilatation. After a couple of days in bed he was allowed up.

Pulse about 98 increased to 132 on bending eight times with some dyspnoea.

Apex 5th space - no dilatation. No evident dilatation after bending eight times.

Mitral systolic bruit.

He feels unfit to work and gets dyspnoeic on slight exertion. No thickening of vessels. He has occasional/

occasional palpitation which alarms him very much.

1.III.21. Feels distinctly better after the Salicylate gr x t.i.d. More able to walk about, still dyspnoeic on exertion.
P. 88-112 after bending ten times. Dyspnoea present but not marked. (B.P. 140, no thickening of vessels.) Still has pains in both shoulders and both knees, wrists and ankles (not hip joints). Still has occasional pain over the heart.

3.III.21. P.68. Mitral systolic still present.
P.100 after 10 bends. Pain in joints very much less. Sod. Sul. gr x. Bicarb. gr x t.i.d.

A few days later the joint pains were improved. The systolic bruit was much less evident, indeed could only be made out definitely after exercise. Exercise tolerance about the same.

33. A case of chronic myocarditis due to infection following rheumatic fever.

David Linton. (Aet 25) Juniper Green.

Family History. His father died of heart disease in 1920. There is no available information as to the origin but he always was an extreme suffered from rheumatism and had always been delicate.

His mother is well (aet 64).

His sister is affected with rheumatism so badly that she cannot walk although not absolutely bedridden: when helped and given two sticks she can shuffle along "all bent up". She never leaves the house. Her age is 41.

Personal History. In 1914, when 19, he contracted rheumatic fever. He was confined to the house for three months. Every joint in his body was affected and the pain was so severe that he used to cry out with it.

He eventually recovered.

Present/

Present History. (1920)

When I first saw him he was an exceedingly nervous man: he was so nervous that if anything fell on the floor he would jump and his "heart went off at a great rate:.

He is at present greatly hampered by severe pain in his joints and states he can predict the weather like a barometer. He has had the pains for about fifteen weeks now.

He is very stiff and cannot bend down and touch his toes. He cannot tie his boot-laces. The right hand meta-carpo phalangeal joints are all swollen and deformed. He cannot abduct the right arm from the side at all, and cannot move his forearm much at all: his right shoulder is particularly painful. Both his knees are very sore and he has myalgia of the thighs.

Heart. The heart was dilated and he suffered from pain which he said was in his heart. No murmurs could be made out: the heart sounds were weak and distant and rapid in action. It was difficult to get him to do any exercise as his joints were all so painful, but I got him to stand up and sit down six times which he had to do slowly, and I suspected a mitral systolic murmur but was never very certain/

certain of it. This small amount of exertion made him very breathless and he did not like to bother about exertion in consequence. No apex beat was palpable or visible but epigastric pulsation was noticable. No signs of heart failure.

He made a slow recovery: he was not long under my charge but I advised him to give up his work of loading carts at Kinleith Mill, which he agreed he was quite unfit for and to start a grocer's shop.

I got permission from his doctor to visit him six months later. He now manages a grocer's shop which suits him very well.

He still gets dyspnoeic on exertion and has occasional attacks of palpitation. Pulse 96 at rest, increased to 122 after exercise. No dilatation of the heart: no murmurs.

His joints had immensely improved though he still felt stiff and feels the weather changes.

34. A case of Tachycardia.

Mrs John Hope. (aet 49) Redding.

The family history was a fairly good one and the personal history was excellent except for an attack of scarlet fever which was followed by some kidney trouble apparently.

One of Mrs Hope's daughters died of influenzal pneumonia when twenty years of age, after less than a week's illness. This gave Mrs Hope a great fright: she took to bed and has never been up since the beginning of 1919. I first saw Mrs Hope in January 1920.

Mrs Hope states that she used to take epileptic fits at the age of 30 and that for a while she had two fits daily, but she has had none for twenty years now.

She suffers from menorrhagia, she says.

Heart: Presystolic thrill and murmur and systolic murmur at mitral area. No signs of heart-failure.

Patient suffers from constant dyspnoea even though she is confined to bed and never gets out of bed. The combination of tachycardia and breathlessness make her unfit for anything.

Patient is a large fat woman and has sugar in the/

the urine - no albuminurea.

The pulse rate was anything from 120 to 150 and quite regular. The blood pressure varied from 140 to 190 mm.

Jan. 17	Pulse 120	Tr. Digitalis	mx tid.
20	140	"	"
22	140	"	mxx tid.
24	95	"	"
27	145	"	"
29	99	"	mxxx tid.
31	140	"	"
Feb. 3	144	Digitalis stopped.	
5	140	"	"
7	105	"	"
9	140	"	"
12	128	"	"
14	124	"	"

The extract will show that digitalis had no effect on her pulse rate so at the end of February I sent her in to Professor Gulland's Ward in the Royal Infirmary.

The resident physician writing about a month after her admission says that the electro cardiograph tracing showed "normal rhythm as regards the synchronising/

synchronising of the auricles and ventricles, thus it is not a case of flutter and seems to be a case of tachycardia pure and simple".

In May Professor Gulland writes:-

"We return Mrs Hope, with the diagnosis, I am afraid, just as doubtful as ever - neither cardiogram nor anything else gave any explanation of her tachycardia - but she is a good deal better in general condition".

I saw her again in the end of August when the pulse was 120 with the patient lying in bed. She got up for a little most days, but was still troubled with breathlessness and not fit for any exertion.

35. Case of an exceptionally heavy drinker with a fairly satisfactory myocardium.

Mr Forsyth. (aet 70). Maddiston.

I had attended Mrs Forsyth for a long time and never took much interest in Mr Forsyth until after his wife died from cerebral haemorrhage.

He had apparently been devoted to his wife and was most terribly affected by his loss. He refused to eat almost anything and it was a pathetic sight to visit him. He would not get out of bed and took no interest in anything. His bowels did not act for long periods - at least a week - and he told me he did not care, that he wished to die.

He was a remarkably spare man with scarcely any fat at all and ate practically nothing.

I feared for his life and was talking to a neighbour one day who told me that for months if not years Forsyth had drunk enormous quantities of whisky, and that latterly he had seen cases of whisky repeatedly being sent into the shop, in the back premises of which, Forsyth lived.

I taxed Forsyth with this fact and he agreed that he had repeatedly taken a bottle of whisky in

a/

a day and that then he was drinking as much as he could, about two bottles per diem! as he wished to die soon, having no further interest in life.

I had often examined his heart and never found it dilated. His pulse was always regular, there were never any extra-systoles nor bruits, though he stated he had occasionally felt as if his heart stopped. Latterly he had some giddiness but this was probably due to the effect of the alcohol on the brain.

I heard recently from his daughter that he lived for three months after I last saw him.

37. Case of a heart struggling against a high blood pressure. (chronic interstitial nephritis.)

Mrs Lillie (aet 52).

She states she had always been quite well until the winter of 1919 when she began to have palpitation. This stopped during the summer and recommenced the following winter when I first visited her.

I found her practically aphasic, the history being that she had had a shock a week previously; she could not speak at all for four or five days: she could make sounds but not articulate: she fully understood all that was said to her. By the time I saw her she could speak a little and managed to make herself understood.

There had been no paralysis.

The urine contained no abnormal constituents. S.G. 1015 on the 18th Sept. The B.P. was 260, a couple of days later it was 350. I had to estimate the 50 as my instrument only reads up to 300. I gave her Trinitrin tablets, one every four hours. Two days later the B.P. was 250.

Heart: regular: no dilatation: no murmurs:

no/

no symptoms. Vessels very thickened and tortuous.

21. IX B.P. 330

23 " " 265

25 " " 240

On November 26th she had a cerebral haemorrhage following on the lowest blood-pressure during the past few days. I found the pupils inequal, the right being wider than the left, and the whole of the left side apparently paralysed. She was completely unconscious. I expected her to die but she recovered. The left side turned out to be only partially paralysed. She became aphasic again. After about two months she gained quite fair control over her left side.

On the 28th and 29th the B.P. readings were respectively 240 and 230.

Nothing worthy of note happened until the beginning of January when I found her B.P. 350 again. I put her on Pot. Iodidi, grains 10 and Liquor Trinitrini, one minim.

January 3. B.P. 350

8 270

18 290

24 310 at this date she entreated to be allowed to stop the K.I. and Liquor Trinitrini mixture, and as it did not appear to have kept

the/

the B.P. down much I allowed this.

25 B.P. 260. It went down to my astonishment, but her bowels had acted very well the evening before.

26 250 Lower still.

27 240 so I decided to start the medicine again.

28 250

31 310 P 120. Patient stated she "felt very done" and as she was constipated she got castor oil which acted in the evening about 7 o'clock. While sitting at stool she fell over and was unconscious her husband states. They got her into bed and she fell asleep, but woke up two hours later and had another attack. The husband was half drunk and could give no description. I arrived about 10 p.m. She was restless and moaned incoherently, uttering words without meaning in which the word "doctor" occurred repeatedly. She was not unconscious but understood nothing said to her. She would gaze with wide staring eyes at one of us, then sit up in bed, burst into a string of indistinct words and throw herself back on the bed still muttering.

Face a little twisted. No paralysis.
The pupils reacted to light, but the R was larger than the L. - it has been so ever since 26th Nov.
I gave ¼ morphia and she went quietly to sleep.
Evening pulse 140: B.P. 310.

On/

On examining the heart I made out perfectly definite Mitral Systolic and Aortic Systolic murmurs. I had at repeated examinations suspected a Mitral systolic but could never be sure of it. But I had never suspected an aortic systolic though I had often searched for its presence as I had expected its appearance sooner or later. There was also a tricuspid systolic. Apex just on nipple-line. Definite venous pulsation in neck.

Feb. 1st. Next morning she felt much better, having slept a good sound sleep. She remembered nothing about the previous evening and had I not made some remark about it to the husband she would never have known she had had it at all.

Apex just outside nipple line. Mitral and Aortic systolics just audible. No aphasia. Can talk quite normally. Pulse perfectly regular.

The Pot Iodidi and Liquor Trinitrini was re-commenced.

She was sent to the Royal Infirmary for further treatment.

38. Development of a bruits, with dilatation, after exercise.

One is repeatedly struck with the fact that, no bruit being audible, it becomes quite definite after exercise.

Numerous examples could be given of this.

In the case of Pensioner George Alexander Pearson of 7 Paten Place, Tillicoultry, it so chanced that all three medical examiners listened to his heart and heard no murmur. It had previously been recorded in his papers that murmurs were present.

After exercise tests however we all satisfied ourselves that the bruits were quite definite, viz. a mitral systolic and a pulmonary systolic.

He was classified as a case of D.A.H.

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39. An indefinite case of possible heart-strain.

Jessie Inglis (aet 20) Clerkess, Polmont.

This patient was at a dance on Jan. 14th, 1920. She agreed that she had got very breathless and progressively so. But she had enjoyed the dance thoroughly nevertheless.

I was called in two days later and found the feet and legs oedematous and that she was feeling very unwell and easily tired.

There was very slight cardiac dilatation, no murmurs, but as there had elapsed an interval of thirty hours, this may have been quite definite the day before. This interval also negatived the physical examination and made the case less definite.

I certainly concluded that the heart had not been fit for the exertion of the dance, that it had dilated and oedema had ensued.

There was no swelling under the eyes nor albuminurea. The B.P. was 120. There was no vessel thickening.

She was a particularly healthy looking girl and resented very much being confined to bed as she felt almost quite well by the time I visited her.

She/

She was kept in bed six days during which period the oedema disappeared and she soon after resumed her occupation.

I either saw her or her mother occasionally during the next seven months and there was no further heart trouble of any sort, nor any other illness.

She is said to have had a "shock" when three years old and lost her speech entirely.

She had had scarlet fever when ten years of age and influenza when 19. She was told she had had a weak heart after the scarlet fever, but she had never had oedema nor any trouble with her heart that she knows of.

She recovered under the influence of rest alone: I gave her no medicines of any sort.

40. A case of congenital heart disease.

May Forrest. (aet 9) Hannahfield.

This child was one of Professor Wyllie's "blue babies". From birth onwards for a month or two she was always "black" the patents state. And now at periods she turns quite blue - they know of no cause for these "blue turns", they simply come and go.

I have seen this child very often and never had any idea she turned blue: she always appeared to me to be the picture of health. I only heart about the attacks when I examined her heart during an attack of mumps and I asked the patents about her birth and was told the above story.

There are no symptoms. The girl can run about and play with other children and never evinces any symptoms whatever. She is active and healthy - no signs of debility or infantilism. There is no clubbing of the fingers.

Pulse generally about 80. Murmurs all soft in quality as follows:- short sharp mitral systolic: soft aortic and pulmonary systolic and occasionally a tricuspid systolic murmur which is long and drawn out. Apex three inches from middle line which is a quarter inch outside nipple line: left border of heart half inch from middle line.

41. Case of a palpitation benefited by bromides.

Mrs Forrest, Hannahfield, Balerno.

This patient has a very nervous temperament: the slamming of a door sends her heart off with a rush she states. The unexpected entrance of a stranger or any sudden event excites her greatly.

On scores of occasions she has described heart attacks which she has: these commence with a trembling of her limbs as a rule, but sometimes the attacks commence without this, or the trembling of her limbs continues without the heart attack.

I always ask her to send for me during an attack: she replied that they come on during the night so I agreed to come at once when sent for. One night she sent a boy on a bicycle and I was at her house in fifteen minutes after his leaving for me, but the attack had as usual passed off. I always suspected that I would never see her in an attack and am sure I never will. I think they are mere feelings of palpitation.

On the night when I was called to see her I certainly did find that I could produce clonus of the ankle quite easily but there were no other signs, and although I took a sphygnograph, the pulse

was/