THE REACTIONS TO ADRENA LIN AND ATROPINE CONTRASTED.
THE REACTIONS TO ADRENALIN AND ATROPINE CONTRASTED.

The results so far described are of some little interest in that they indicate a striking contrast between the actions of atropine and adrenalin on the idio-ventricular rate. In every instance in which these drugs were employed, some acceleration of the rate of the ventricles was induced. When the results are examined it is found that in each instance the response of the ventricles is determined by their initial rate. In spite of the fact that each drug induces acceleration, yet different laws determine the nature of the reactions, for at those initial rates, where adrenalin produces little or no acceleration, atropine has its most marked effect and vice versa.

The responses to adrenalin and atropine may be conveniently compared by graphic presentation of the related facts. In Chart 19 the increments observed for each drug have been plotted against their corresponding initial rates. Atropine, was injected intravenously in a dose of 1/30 grain, while adrenalin was administered subcutaneously 0.5/
0.5 c.c. each yielding the corresponding points inserted in the chart. The optimum response to adrenalin takes the form of a curve which is almost a perfect example of the decreasing exponential type.

If the atropine effect were the converse of that produced by adrenalin then an increasing exponential curve would be expected to express adequately the relation of the increment to the pre-existing rate. It has already been pointed out that an insufficient number of points are available for the constriction of a curve of the atropine response over the relatively wide range of initial rates studied. Nevertheless a theoretical curve is inserted in the chart which is exactly the opposite of that described by the adrenalin reactions. It will be observed that for low initial rates, the observed points (indicating the maximum gains in rate after atropine) fall around the suggested the theoretical value. This implies that for rates up to about 35 per minute, atropine produces about the same amount of acceleration, as adrenalin does at initial rates greater than 35 - the one being the mirror image of the other.

A divergence between the theoretical exponential
O = Observed response to 0.5 cc Adrenalin.
O = Optimum values for the Adrenalin response.
X = Observed response to 1/800 ATROPINE sulph.
O = Theoretical response to Atropine.
P-T = Probable curve of response to Atropine.
exponential response for atropine and the observed values occurs for initial rates in excess of 35 per minute. Unfortunately only three atropine observations were made at initial rates greater than 35, but each, when plotted in the diagram lies well to the left of the theoretical curve. Actually the curve P-T (as entered in Chart No. 19) might be taken to more closely represent the atropine response at different initial rates. Within the range of rates over 35 the atropine response cannot therefore be regarded as the perfect mirror image of adrenalin, for the actual increment is in excess of the theoretical. In other words the acceleration induced by atropine is apparently greater at rates over 35 per minute, than that which occurs after adrenalin at rates below 35.

The difference between the observed and the theoretical values for the atropine response are worthy of brief consideration. It is universally agreed that, in virtue of its power to paralyse the parasympathetic nerve endings, atropine induces acceleration of the heart. Similarly the adrenalin reaction occurs in response to an increase of sympathetic tone but the actual response in man is in some/
some measure distorted by an indefinite amount of reflex vagal inhibition. The recorded rate is therefore the product of two interacting forces – augmentation, and reflex inhibition. For this reason, of the two drugs, atropine produces a response, which by its nature more nearly represents a perfect and uncomplicated reaction.

In discussing the reaction of the auricular rate to adrenalin, the divergence between the auricular and ventricular responses received special attention. Emphasis was placed on the fact that with decreasing initial ventricular rates, the auricular response tended on the whole to lag more and more behind the ventricular. As an explanation for this phenomenon it was tentatively suggested that an increased degree of reflex vagal inhibition, consequent upon the free ventricular response, probably held the auricles in check.

It is attractive to speculate along similar lines regarding the problem at present under consideration. Consider the diagram (Chart 20) in which, for the sake of argument, the observed response to adrenalin for a given initial rate is represented by the straight line BX, and the observed response to atropine by the line AQ. Suppose that/
that atropine completely frees the ventricle from parasympathetic influences and that sympathetic tone remains constant (as it apparently does). This means that the line AQ may be taken as representing complete vagal paralysis. Now if atropine and adrenalin have reversed actions, then the line BP represents full accelerator tone. But BX is the actual response to adrenalin, which falls short of the theoretical line BP, by increasing amounts step by step with lower initial rates. In other words if the line BP represents full acceleration, as might be anticipated from the atropine response AQ, then the adrenalin reaction is retarded by a varying amount increasing up to the interval XP at an initial rate of 17. This suggests that the greater the ventricular response to adrenalin the more does some extraneous influence tend to limit the response. From analogy with the divergence observed between the auricular and ventricular reaction to adrenalin, it may be tentatively suggested that even in the presence of complete heart-block, the ventricles are also in some measure retarded by reflex vagal influences, just as the auricles are.

It would seem definitely established that atropine/
atropine at an appropriate initial rate (say 45) produces a greater gain in rate than adrenalin given at a corresponding rate, say 25. The natural implication is that the ventricular rate in complete heart-block is retarded to some extent by reflex vagal tone induced by the rise of blood pressure which is an essential feature of the response to adrenalin. Further the degree of vagal tone varies with the magnitude of the response. In so far as vagal tone is measured by change in rate, the degree of tone must vary with the rate at the moment but not necessarily in a direct linear way. In future work it would seem desirable to study the effect of small doses of adrenalin (say 0.25 c.c.) at various times after the intravenous injection of 1/30 gr. atropine. This might be compared with the effect of atropinisation at the height of the adrenalin reaction. The results would give valuable information regarding the extrinsic nervous control of the ventricles in complete block.

It has already been suggested that the response to atropine is probably best represented by a curve rather than by a straight line formula. The exact nature of the curve cannot be accurately determined/
determined from the available figures, but by plotting the log of the initial rate \((x)\) against the log of the increment \((y)\) the points, such as they are, fall along an almost perfectly straight line. If this relationship be true throughout the whole of the available range, then the law expressing the reaction of the ventricles to atropine may be represented by \(\log y = \log K + n \log x\). The observed response to adrenalin is apparently best represented by a decreasing exponential curve. The difference between these two equations may be taken to represent the amount of vagal tone acting on the ventricles at various rates in complete heart-block.

**SUMMARY.**

From a comparison of the effects of atropine and adrenalin at given initial rates of the ventricle in complete heart-block, it is suggested that reflex vagal tone inhibits in some measure the natural tendency of the ventricles to develop full acceleration after adrenalin.

Taken all over the response to adrenalin is less complete than that of atropine. The auricular gain in rate is distinctly limited after adrenalin, and in comparison with atropine at appropriate/
appropriate (but different) initial rates the ventricular response is probably also retarded for the same reason.

This interpretation of the reaction to atropine and adrenalin suggests that the vagus can and generally does influence the ventricles in complete block. A mathematical method of calculating the degree of vagal tone acting on the ventricles is suggested. Quantitative comparisons between the degree of vagal tone existing in the auricles and in the ventricles, is rendered complex by the natural differences in their inherent rhythmicity.
THE RESPONSE TO EPHEDRINE.
THE RESPONSE TO EPHEDRINE.

The alkaloid ephedrine, obtained from the herb Ma Huang possesses sympathomimetic actions very similar to those of adrenalin, which it also resembles in its chemical structure. Since 1924, following the experimental work of CHEN & SCHMIDT, (27) an extensive literature has accumulated regarding its pharmacological and therapeutic properties.

It would appear that, while ephedrine in large doses may act as a depressant on the mammalian heart, yet the cardiac stimulant effects of smaller quantities are quite well marked in the case of the human heart. The effects of ephedrine in therapeutic doses are less intense than those of adrenalin, but persist for a longer time. The heart rate tends to quicken, but inhibitory influences consequent upon a rise in arterial pressure, check the response and may indeed be so far increased as to induce slowing of the beat. It has been suggested (Chen & Schmidt) that ephedrine not only acts on the myoneural junctions of the accelerator system, but also upon the sympathetic ganglia, thus differing slightly from adrenalin in the locations of its action.

For these reasons it might be confidently anticipated/
anticipated, that at least under experimental conditions, ephedrine would augment the rate of the idio-ventricular rhythm in complete heart-block, artificially induced. So far as can be discovered, the literature contains no record of such an investigation. There are available, however, isolated reports of the action of ephedrine in complete heart-block in man. MILLER (158) was the first to use ephedrine in a case of complete heart-block. After a subcutaneous dose of 100 mgms., electrocardiograms demonstrated an increase in the auricular rate from 110 to 125 per minute, in the ventricular rate from 33 to 55 per minute, and an associated rise in the blood-pressure. Some variation in the shape of the ventricular complexes was observed, similar to that recorded by GILCHRIST & COHN (87) and as noted by DE GRAFF & WEISS (90) after adrenalin. There were no Stokes-Adams seizures in Miller's patient.

HOLLINGSWORTH'S (114) patient suffered from completed block complicated by repeated Stokes-Adams seizures, which ephedrine, in daily doses of 50 mgms. by mouth, successfully abolished. The attacks recurred within 48 hours of stopping the administration of the drug. STECHER (205) reported a similar case. He observed that, not only were the seizures abolished, but that the auricular and ventricu/
ventricular rate increased from 80 and 30 respectively to 88 and 40 as a result of the administration of ephedrine. Using a synthetic preparation of the drug, PARADE & VOIT (166) obtained similar results.

From the similarity in their pharmacological actions, supplemented by the scanty clinical reports concerning the use of ephedrine in complete heart-block, it is probably right to conclude that both drugs, adrenalin and ephedrine, are capable of increasing the idio-ventricular rate in man. Of the two, ephedrine is perhaps the more serviceable as a therapeutic remedy in the prevention of Stokes-Adams attacks, on account of the fact that it is equally efficacious when administered orally.

RESPONSE OF THE AURICLES AND VENTRICLES TO EPHEDRINE.

The effect of the oral administration of ephedrine has been tested in seven patients suffering from complete heart-block (Case 1, 2, 4, 5, 9, 10, 14). The drug was generally given three times a day in the form of tablets and the rate of the pulse was counted hourly or two hourly by the nurse in attendance. The rates were checked by daily electrocardiographic/
electrocardiographic examinations and were compared with those existing before and after the experimental period. It seemed desirable to test the efficiency of the drug in this way, rather than by subcutaneous injection, for it is largely in virtue of its alleged efficacy by oral administration that the ephedrine is entitled to a place in therapeutics, particularly in view of the claim that it is successful in warding off Stokes-Adams attacks.

In Case 1, two doses of \( \frac{1}{2} \text{ gr.} \) were given daily over a period of 11 days. No effect on the ventricular rate was observed. The pulse was counted at four hourly intervals, and the minor fluctuations in rate were found to be of the same range and extent as those observed before and after the drug's administration. Electrocardiograms did not display any alteration in the form of the waves. The T waves were unaltered. The blood-pressure was unchanged.

In Case 2, the drug was given after a course of digitalis had failed to relieve an advanced degree of congestive heart failure. For 6 days, 1 grain of ephedrine was given thrice daily. No definite effect was observed.

In case 4, the dose employed was \( \frac{1}{2} \text{ grain} \) t.i.d. for 8 days. The effect was indecisive. No alteration/
alteration was observed in the form of the electrocardiogram.

In Case 5, ephedrine produced a definite acceleration of the ventricles. The course of treatment commenced on 13th March 1932, and ended on the 23rd, as shown in Chart 21. On the first day, the patient received two doses of \( \frac{1}{2} \) grain. For the preceding six days, the rate varied from a minimum of 32 to 38. Throughout the whole period depicted in the Chart, the pulse rate was accurately counted for a full minute at intervals of 4 hours. The maximum and minimum daily rates are recorded in the diagram where they can be conveniently compared with the amount of ephedrine taken. The highest rate noted was 46, representing a gain of at least 8 beats per minute.

After the cessation of this treatment, the rate gradually returned over a period of 10 days, to the pre-existing level. Increasing the dose, within the range employed, apparently increased the response, which, measured in this rather crude way, takes quite an appreciable time to develop. The systolic blood-pressure increased by about 20 to 25 mm. at the height of the reaction. The electrocardiogram remained unaltered, save for the increase in rate, which also affected the/
the auricles.

An acceleration of the pulse was induced in Case 10, by the administration of 1 grain of ephedrine 4 times a day for 4 days. This woman's pulse ranged from a rate of 28 to 32 per minute during the control period. Ephedrine in \( \frac{1}{2} \) grain doses 4 times a day for 3 days produced no appreciable effect on the rate of the ventricles, but when the dose was doubled, definite quickening of the ventricular beat was recorded. The rate ranged from 32 to 36 during the four days on which the larger dose was used. On discontinuing the drug, the rate returned to its pre-existing level within 36 hours. Extrasystoles were not observed. The blood-pressure and electrocardiograms were not significantly altered.

Case 14 presents some unusual features. This patient had been under observation suffering from partial block of the 2 to 1 type without prolongation of the P-R interval for two years. She was admitted to hospital in April 1932, by which time the block had become complete with some irregularity of the ventricular rhythm. Daily electrocardiograms over a period of one month demonstrated no improvement in conduction and it was then decided to test the effect of ephedrine. An ample control period,
period during which the patient was confined to bed, demonstrated that spontaneous fluctuations in the ventricular rate allowed of a natural variation ranging from 26 to 32 beats per minute. Ephedrine ½ gr. 4 times a day was commenced on 26th May, on which date she received a total of 1 gr. as shown in the accompanying Chart, No 22. The ventricular rate increased from 30 to 36 and electrocardiograms demonstrated, four days after commencing the drug, that complete heart-block had been replaced by a regular 3 to 1 rhythm, the P-R interval being 0.395 sec. The systolic blood-pressure fell from 250 mm. during the complete block to 210 during 3 to 1 rhythm. In other words, the increase of rate under influence of ephedrine was accompanied by a decline of at least 40 mm. in systolic pressure. The administration of the drug was stopped on the night of 31st May.

A remarkable series of symptoms arose within twelve hours of discontinuing the treatment with ephedrine. In the early hours of the following day (1st June) the patient complained of headache, pains in the back, and suffered from two faint turns. Both of these lasted no more than a few seconds. At 11.55 a.m. she complained of feeling weak, and it/
it was observed that the pulse rate had fallen to 10 beats a minute. This was confirmed by electrocardiograms as shown in PLATE 4, which also demonstrate that the ventricular rhythm is not perfectly regular, the intervals in lead 2 between successive ventricular cycles being 5.680, 5.625, 5.710, 7.805, 6.605, 5.780 and 6.675 secs. This yields an average ventricular rate of 9.57 beats per minute. The auricular rate (in lead 2) is 75.3. She remained quite conscious, complained of some headache and general weakness, but was sufficiently well to ask for nourishment. At 12.35 p.m., shortly after taking food, she complained of feeling sick and immediately afterwards had a fit, with twichings of the face, arms and legs. The eyes became dull and glassy, with upward deviation and the pupils dilated. She was unconscious for about 1½ minutes. Throughout the whole of the seizure, the pulse could be felt at the wrist, but the rate declined to 7.5 per minute. Immediately before regaining consciousness, the heart quickened to 10 or 15 beats per minute. Between 1 and 2 p.m. she had further attacks, each of which resembled the first and in none of which was there a period of complete ventricular arrest. A subcutaneous injection of 0.5 c.c adrenalin, by producing 4 to 1 block appeared to have the effect/
effect of abolishing the attacks. Two further doses of adrenalin were given during the same evening. The following day (2nd June), ephedrine was recommenced, $\frac{1}{2}$ grain, 4 doses daily by mouth. No further seizures occurred. On the 3rd June, the midday dose of ephedrine was omitted, and the patient kept under careful observation. At 3.45 p.m., she had a slight but definite Stokes-Adams seizure. Ephedrine was recommenced at 4.10 p.m., and no further attacks were recorded. On the 5th June, she complained of giddiness and weak feelings about 8 a.m. without the development of seizures. The dose of ephedrine was increased to a total of 3 gr. a day, conduction being restored to 3 to 1 rhythm. No further attacks occurred. She was discharged from hospital the following day and advised to continue taking ephedrine. This she has done and to date has had no further seizures. When last seen (December 1932) the block was again of the complete variety, and she has kept in very good health.

To summarise this case briefly, it would seem justifiable to conclude that the administration of ephedrine was the means of restoring conduction between auricle and ventricle. Complete block was replaced by 3 to 1 and the ventricular rate increased.
On abrupt withdrawal of the drug, the ventricular rate declined to an alarming extent. At a rate of 7.5 per minute, Stokes-Adams seizures occurred. At a rate of 9.75 or thereabouts, the patient was sufficiently conscious to engage in conversation, but all her actions were slowed and she complained of headache and weakness. The block remained complete for two days, but a further course of ephedrine again restored the 3 to 1 rhythm, which persisted until her discharge from hospital.

This is, no doubt, an exceptional reaction, but it serves to emphasise a danger associated with the administration of ephedrine - at least in the presence of certain forms of complete block. It is not proposed to discuss this case in full, but the available evidence suggested that the block had not been long of the complete variety when the patient came under observation in hospital. Ephedrine appeared in the first instance to have a stimulant action on the ventricle, facilitating conduction over a few remaining strands of the A-V bundle, but when the drug was withdrawn, conduction failed, and, probably as a result of exhaustion from over stimulation, the natural rhythmicity of the ventricular centre declined. Stokes-Adams seizures then occurred.
Certain of the phenomena described above, might be interpreted in a number of ways, e.g. according to the theory of parasystole, or to "block-in-block". It is not proposed to discuss, at the present time, the theoretical aspects of the nature of the block in this woman, nor to enter into detail regarding the action of ephedrine in the conducting tract. The case is here recorded with the desire to draw attention to a possible danger in the indiscriminate administration of ephedrine to patients suffering from certain forms of complete heart-block.

THE USE OF EPHEDRINE IN THE PREVENTION OF STOKES-ADAMS SEIZURES.

In but few patients do Stokes-Adams seizures occur with sufficient regularity to enable one to assess the efficacy of any particular method of prevention or treatment. It has been previously pointed out that the majority of the individuals in the present series of cases, suffered from seizures, of greater or less severity at one time or another. There is every reason to believe that attacks of the form described by Adams & by Stokes tend to herald the onset of complete block. Repeated or single attacks may then occur, and the idio-ventricular rhythm becoming firmly established, the patient may live for years,
years, never to experience another fainting fit. On the other hand, seizures are occasionally repeated at long intervals of months or years, despite the persistence of chronic complete heart-block. When attacks occur at rare and infrequent intervals, the effect of treatment aimed at their prevention is difficult to assess. Even in those patients, who suffer from repeated attacks, perhaps at short intervals, care must be taken in drawing conclusion, for Stokes-Adams attacks are always irregular in their time incidence. As the precipitating cause of attacks doubtless varies from case to case, and even from time to time in the same individual, it is essential that a certain restraint should characterise all therapeutic deductions regarding such a peculiar and varying group of symptoms as the Stokes-Adams syndrome. It is probable that the cardiac mechanism responsible for the onset of periods of ventricular asystole varies from seizure to seizure, even in the same individual, and it might therefore be anticipated that the successful prevention of cardiac arrest by such a drug as ephedrine, will depend on a number of factors.

The probability is that, as the mechanism of the heart's action varies in different attacks, not/
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<table>
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Days: 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21
not only during the arrest of the circulation, but also before and after the seizure, so rational and successful therapy will depend upon further detailed knowledge regarding the precise nature of the Stokes-Adams attack. Certain distinct varieties are already known.

It is perhaps too much to hope that ephedrine will prove equally effective in all cases under all circumstances.

In Case 9, ephedrine was also employed, but the effect observed is difficult of interpretation. This patient suffered from intermittent complete heart-block with repeated Stokes-Adams seizures. The day after commencing treatment with ephedrine, the ventricular rate greatly increased, the block disappeared and the Stokes-Adams seizures ceased. Electrocardiograms from this patient are shown in FIGS. 9 and 10. On ceasing the administration, the pulse slowed and some weeks later Stokes-Adams attacks recurred. See Chart 23. It is not possible to say that ephedrine was directly responsible for the cause of events in this patient. To date there has been no opportunity to repeat the test in this patient.

As an example of repeated seizures, Case 10 is of some interest. This woman suffered from
(Case 9 - 20th. June 1932.)

FIGURE 9.

FIGURE 10.

(Case 9 - 22nd. June 1932.)
occurred. The drug's administration has now been discontinued for two months and at the time of writing no attacks have followed its withdrawal. No precise conclusions can therefore be drawn from such a test, unless it be that ephedrine in the dose employed, apparently did no harm.

Seizures were likewise of fairly common occurrence in Case 13. This woman suffered from attacks at the rate of two or three a fortnight with intervals of two or three months' freedom. Yet she had none during her stay in hospital, and only one in the four months since her discharge from the institution. Her response to ephedrine was not tested. No particular reason can be advanced for her symptomatic improvement. She received no drug treatment.

The preceding cases of complete heart-block complicated by fairly frequent seizures, illustrate the difficulty in assessing the value of ephedrine in the prevention of the attacks. The administration of small doses of the drug appeared to have the effect of preventing seizures on one individual (Case 4) but, from the scanty facts available, the efficacy of the drug cannot be definitely established in this type of case, or by this method of administration. Nevertheless, the fact that the drug possesses the power (at least in certain cases) to hasten the ventricular rate/
rate, coupled with its simple method of administration, makes it desirable that its potential usefulness in the prevention of the Stokes-Adams seizures should be widely known and that it should be given an extended trial when opportunity occurs.

While no positive conclusion can be reached regarding the value of ephedrine in the prevention of Stokes-Adams attacks occurring intermittently during the course of chronic complete block, yet very suggestive results were obtained in one patient who suffered from repeated periods of ventricular arrest in the course of a partial block of the 2 to 1 or 3 to 1 type. This patient (Case 14) was admitted to hospital in June 1931 on account of repeated weak turns and faintness, coming on without warning. Reference has already been made to this patient. On a second occasion (June 1932) after a second course of ephedrine treatment, extreme slowing of the ventricular beat occurred in association with Stokes-Adams seizures. In June 1931, she also suffered from seizures, but of minor severity, frequent periods of ventricular arrest lasting up to 10 or 12 seconds, being recorded electrocardiographically in the course of a 2 to 1 block. An example is shown in the continuous film record, in PLATE 5. During the particular arrest/
arrest recorded in the figure, the patient did not lose consciousness nor suffer from epileptiform attack. She merely complained of a transient weak feeling. The electrocardiogram shows that the period of ventricular arrest lasted for 9.76 seconds.

Before the attack, the block was of the 2 to 1 type, successive P-R intervals being 0.180, 0.175, 0.180, and 0.210 seconds for the last conducted beat. On the same period the ventricular rate was 29.91 per minute. During the arrest of the ventricular beat it is interesting to note that the auricular rate quickens, the interval between successive P waves declining from 0.970 seconds to 0.785 seconds, representing a rate over all of 67.8 auricular beats per minute. After the period of arrest, the ventricles resumed beating in response to every third auricular impulse. It is of interest to note that both before and after the period of ventricular asystole, the auricular beats are irregular in a peculiar way. The interval between those auricular contractions which are separated by a ventricular response, is shorter than those in which no ventricular systole occurs. The same phenomenon frequently occurs during complete heart-block, but it is less commonly seen in partial. (WENCKEBACH & WINTERBERG (222) WILSON & ROBINSON (232)).
The irregularity of the auricles, which often accompanies ventricular bradycardia, cannot be entirely attributed to vagal influences (as has been suggested), for HECHT (102) has shown that it may persist, though to a lesser degree, after the injection of atropine. ZEISLER (235) attributes the peculiar auricular arrhythmia to abrupt improvements in auricular blood-flow. With an improved circulation, the S-A node builds up and discharges an impulse more promptly. This patient provides an excellent example of this auricular arrhythmia during partial block. That it bears a definite relation to the ventricular contraction is supported by the fact that, during the period of asystole, the alternate quickening and slowing of auricular cycles disappears and is replaced by a progressive quickening. (anoxaemia). When the beat of the ventricles is resumed, the fluctuations in auricular rhythm again appear.

She was admitted to the ward for observation on the 25th June and at 10.30 p.m. that evening several "fainting attacks" occurred in succession, but though the patient became pale and complained of weakness, consciousness was maintained and no fits occurred. On the 26th, 29th and 30th June, similar
single attacks were noted. On the 1st July, there were about half-a-dozen attacks, and the following day they increased greatly in frequency. The electrocardiogram recorded on the morning of the 2nd July (PLATE 5) depicts one of the many seizures (about 20 in all) which occurred in the space of an hour or two. The patient was so alarmed and apprehensive that it was thought right to administer 0.5 c.c. adrenalin subcutaneously at 2 p.m. She had no further attacks until the evening between 5 p.m. and 6 p.m., when she again fainted twice. Ephedrine was commenced in doses of half a grain, 4 times a day at 6 p.m. on 2nd July.

No seizures were observed or complained of on the 3rd, 4th, or 5th July. The administering of ephedrine was stopped after the evening dose on the 5th, and at 6 a.m. the following day, (6th July) two or three minor attacks occurred. The drug was recommenced on the afternoon of the 6th in the same dosage, and continued until her discharge from hospital. On the 8th and 9th July, two short attacks were complained of about 8 a.m. on each occasion. By rearranging the times of dosage she remained entirely free from symptoms.

It seems reasonable to conclude that ephedrine (and also the single dose of adrenalin) played a/
a decided part in the prevention of attacks in this individual. Strangely enough, in the dose employed, there was no appreciable alteration in the heart rate during the time that the drug was being administered. The block remained for the most part as 2 to 1, though occasionally interrupted by periods of 3 to 1 rhythm.

SUMMARY.

A few isolated case reports collected from the literature suggest that ephedrine sulphate may enhance the ventricular and auricular rates in the presence of complete heart-block, and that this drug may be of value in the prevention of Stokes-Adams seizures.

The literature does not contain a report of an extended clinical trial of the drug in complete block, and no observations are available regarding its effect on the ventricles under experimental conditions (when block has been artificially induced). Resembling adrenalin in its pharmacology, it may be anticipated that ephedrine (in sufficient quantity) will accelerate the idio-ventricular rhythm. Its particular/
particular virtue lies in the fact that the drug, unlike adrenalin, is not destroyed in the alimentary tract. As a test of its capabilities, ephedrine was administered orally in tablet form to seven patients suffering from complete heart-block, and to one patient with partial block of the 2 to 1 type.

In three cases (using doses of \(\frac{1}{2}\) gr. twice a day, 1 gr. thrice daily, \(\frac{1}{2}\) gr. thrice daily, respectively), no acceleration of the ventricular rate was observed. The auricular rate was not decisively altered.

In three cases the doses used were \(\frac{1}{2}\) gr. thrice daily, \(\frac{1}{2}\) grain four times a day, and 1 grain four times a day respectively. Definite acceleration of the ventricular rate occurred in each of these instances. The dose required (to increase the ventricular rate) apparently varies in different cases.

Within twelve hours of stopping the drug, excessive slowing of the ventricles was observed in one case. The rate fell to 10 per minute, and, at a ventricular rate of 7.5 per minute, repeated Stokes-Adams attacks occurred. In this patient, ephedrine restored conduction, 3 to 1 block being induced. When the drug was stopped Stokes-Adams attacks occurred, the ventricles slowed and the block/
block became complete.

The effect of ephedrine in a case of intermittent complete block with repeated seizures was indecisive. The drug appeared to abolish the attacks and restore normal rhythm. With the data available, the exact influence of the drug on the course of the events recorded must remain problematical.

No positive conclusions can be drawn regarding the value of the drug in the prevention of Stokes-Adams seizures in the course of chronic complete block. When attacks occur sporadically for no known reason, the effectiveness of any therapeutic remedy is difficult to gauge. Ephedrine is worthy of an extended clinical trial, in the treatment and prevention of the Stokes-Adams syndrome.

In one case in which repeated periods of ventricular asystole occurred during the course of a 2 to 1 block, the drug did good. It lessened, and eventually abolished attacks. These recurred when the drug's administration was stopped.
THE RESPONSE TO DIGITALIS.
FIGURE 11.
(Case 2).

Before digitalis:

After digitalis:
An enormous amount of work has been done on the pharmacology of digitalis, but even to-day much remains to be discovered regarding the actions of this peculiar drug. It is generally agreed that, in so far as it affects the heart, digitalis tends to induce slowing largely as a result of central inhibition and partly also, in certain instances by increasing the refractory period of the A-V node, thereby delaying conduction to the ventricle. Besides the local action on the conducting tract, the drug has a powerful influence on the ventricular muscle increasing the force of contraction (46). Actually it would appear that after an adequate dose of digitalis the observed reaction, varying as it does in the healthy subject and in the cardiac patient, depends upon many interdependent readjustments.

The work of STEWART (206) and STEWART and COHN (207) who have recently reinvestigated the action of digitalis on the heart of man, demonstrates that in normal healthy individuals the administration of the drug produces a slight slowing of the heart rate, a decrease in the cardiac output and a diminution in the area of the X-ray shadow of the heart.
Before digitalis:

After digitalis:
heart. It is interesting to note that these relations to digitalis in healthy people may be accompanied by such subjective symptoms as a slight degree of dyspnoea and some precordial pain. With the exception of the pulse-slowing and the decrease in cardiac output the action of digitalis in health is the reverse of that observed when it is administered with benefit in the presence of congestive heart failure. COHN & STEWART observed that, in cardiac cases the clinical improvement, after the use of digitalis, ran parallel to the increase in the cardiac output.

The response to digitalis in a given instance is therefore largely determined by the pre-existing state of the heart and circulation. This principle, first enunciated by MACKENZIE (150), is of the utmost importance in any endeavour to study the effects of digitalis. Its significance cannot be over emphasised. In health this drug reduces the cardiac output; in certain diseased states the output is increased; ultimately in the most advanced stages of congestive failure, a state is reached in which the heart is incapable of responding satisfactorily to the drug. Toxic effects of serious significance are then liable to occur. Irregular bouts of ventricular tachycardia (84) with cardiac dilatation and a further diminution in the circulation rate/
FIGURE 13.
(Case 10).

Before digitalis:

After digitalis:
rate indicate that the limits of cardiac tolerance have been passed. The drug's action is therefore determined by a number of factors, so many of which are unknown that clinically it is not often possible to anticipate accurately the character or course of the response in a given patient.

The fact that in man the reaction to digitalis differs considerably in health and disease, and also in the various grades of heart failure is sufficient to indicate the complex problems involved in any attempt to reconcile the deductions made in the laboratory with those gleaned from prolonged clinical study. Nevertheless it is of considerable interest to note that in animals, after destruction of the conducting tract, effective doses of digitalis generally increase the idio-ventricular rate. First described by TABORA (217), this accelerating effect of digitalis on the ventricular rhythm in complete block had been confirmed by ERLANGER (63), CUSHNY (46) ROTHBERGER and WINTERBERG (188) and by VAN EGMOND (58). On the other hand when the action of the drug has been tested in man, the general conclusion has been that, though the auricles might be slowed, the ventricular rate remains unaltered (SEMERAU (197), DE GRAFT and WEISS (89), BACHMAN (6) HEMLETT and BARRINGER (108)). In actual practice a change in the rate of the usual ventricular rhythm/
FIGURE 14.
(Case 11).

Before digitalis.

After digitalis :

Lead 1

Lead 2

Lead 3
rhythm is not of great importance provided that the peripheral congestion is relieved. Benefit, following the administration of the drug to patients suffering from congestive failure and complete block, has frequently been reported (JOLLY and RITCHIE (118), MARTINET (153), MACKENZIE (150), JOSEPH (119), EDENS (57), VACQUEZ (214) and WENCKEBACH (222)). More recently SCHWARTZ (193) from a study of the response of eight patients to digitalis therapy came to the conclusion that the drug may be of great benefit in complete block complicated by congestive failure. The improvements in these patients is brought about by the direct action of digitalis on the heart muscle and not by an acceleration of the automatic ventricular rhythm. SCHWARTZ observed an increase in the ventricular rate in three of his patients but only after doses of digitalis larger than those commonly used in therapeutics. No mention of a slowing of idio-ventricular is made in this publication and presumably in the five patients, who made a satisfactory response to the drug the inherent rate of the ventricles remained unaltered. If it be accepted, that the ventricles in complete heart-block are no longer under the control of the vagus mechanism, then it is difficult to appreciate how digitalis could induce slowing of the automatic centre of impulse production/
FIGURE 15.
(Case 2).
production. So far as I am able to discover there are no clinical studies in the literature which demonstrate a significant degree of slowing of the ventricular rate after effective doses of digitalis. Many of the clinical reports are lacking in adequate control periods, and when the antecedent rate and range have not been recorded accurately for some days, the slighter changes in rate by the drug are apt to pass undetected. There seems to be no doubt that in the usual therapeutic doses digitalis may have no significant action on the ventricular rate. If it be continued until the full digitalising dose is exceeded, acceleration may occur as an expression of an abnormal ventricular rhythm. On the other hand, in the studies to be reported here, it has been possible to show that full therapeutic doses of digitalis slow the ventricular rate in certain cases.

THE ACTION OF DIGITALIS ON THE VENTRICULAR RATE.

The effect of digitalis in complete block has been tested in seven patients (Cases 1, 2, 4, 5, 6, 10, 11). During the entire period of observation all the patients were confined to bed; for at least ten days or a fortnight, the pulse rate was recorded at two hourly intervals before the drug's administration was/
FIGURE 16.
(Case 12).

FIGURE 17.
(Case 12).
was commenced. The temperature was also recorded, and if febrile, it was considered that the patient was unsuited for the test. None of the patients submitted to this investigation showed a rise of temperature above 98.0 during the experimental period. Digitalis was administered in different forms and by various methods. Several patients received doses calculated by the body-weight method of EGGLESTON. Some received a reliable tincture of uniform potency, others received a powdered preparation of digitalis leaves, with which we had previously had ample clinical experience to judge of its reliability (The powdered digitalis used was sample B, prepared by Magnus for the Hygiene Committee of the League of Nations and tested on man by GILCHRIST and LYON (88)). All patients received sufficient of the drug to produce full therapeutic digitalisation. Electro-cardiograms were recorded frequently in the control period, and at daily intervals thereafter. The pulse counts were checked by accurate measurements of the ventricular and auricular rates.

In four patients (Cases 2, 5, 10, 11) adequate doses of digitalis induced a slowing of the ventricular rate. Case 2 suffered from advanced congestive heart failure, and though a slight degree of slowing was/
was induced no benefit resulted from digitalisation. The remaining patients (No 5, 10, and 11) were in reasonably good health at the time of the drug’s administration. They were all free from peripheral congestion and presented no special indication for the use of the drug. The results obtained are best demonstrated diagrammatically as shown in the accompanying three charts in which are inserted the maximum and minimum pulse rates for each 24 hour period in relation to the digitalis dosage. The minimum ventricular rate recorded before the use of the drug and the minimum rate at the height of digitalisation are also recorded in the following table.

**TABLE XXIV.**

<table>
<thead>
<tr>
<th>CASE NO. &amp; INITS.</th>
<th>BEFORE DIGITALIS</th>
<th>AFTER DIGITALIS</th>
<th>METHOD OF ADMINISTRATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>2. W.W.</td>
<td>37</td>
<td>30</td>
<td>180 minims divided into 8 doses.</td>
</tr>
<tr>
<td>5. A.H.</td>
<td>30</td>
<td>26</td>
<td>Total of 800 minims in 12 days.</td>
</tr>
<tr>
<td>10. Mrs C.</td>
<td>32</td>
<td>26</td>
<td>Total of 560 minims in 7 days.</td>
</tr>
<tr>
<td>11. Mrs D.</td>
<td>40</td>
<td>32</td>
<td>Total of 24 ccs. in 4 days.</td>
</tr>
</tbody>
</table>
It may well be that actually a greater degree of slowing than that recorded from the counting of the pulse rate actually took place. For instance the slowest ventricular rate registered by the electrocardiograph in case 10 was 23.9, whereas the minimum daily count at the pulse on the same day was 26. Nevertheless the method employed in estimating the effect of digitalis is reliable in that a natural variation occurs in the 24 hour period and it is obviously not possible to record every fluctuation electrocardiographically or otherwise. A study of the diagrams indicates that the slowest rates were observed at the time when the concentration of the drug in the tissues was at a maximum.

To consider the course of the reaction in a little more detail it is necessary to refer to the charts. In Case 5 (Chart 24) repeated daily doses of the tincture of digitalis were employed until headache, nausea and finally vomiting were induced. Slowing of the ventricles resulted and on discontinuing the drug the rate rose gradually to the pre-existing level. Case 10 was a feeble old woman with signs of hypostatic congestion without generalised oedema. Digitalis slowed the ventricular rate and increased/
increased the force of the apex beat without benefit to her general condition. Three days after discontinuing the drug the temperature rose, her general condition deteriorated, the ventricular rate increased (the block remaining complete) and two days later she gradually sank and died peacefully in her sleep. Very definite effects followed the administration of massive doses of the tincture to Case 11 (Chart 25). No toxic symptoms were induced.

The electrocardiograms from these patients are of some little interest. In each individual a slight change in the form of the T waves was induced similar to that described by COHN, FRASER and JAMIESON (37) in patients treated with digitalis. The flattening and distortion of the T wave observed in these four cases of complete block affords additional evidence that the ventricular muscle was under the influence of the drug when the low rates were recorded. Further, the fact that the QRS waves continued to have the same form throughout the experimental period indicates that the slower rate was not the result of a dislocation of the centre of impulse production to a lower rhythmic level in the specialised tissues. Fig. 11 shows the electrocardiograms before and after digitalis in Case 2; Fig. 12 those in/
in Case 5; Fig 13 those in Case 10, and Fig 14 those in Case 11.

In contrast to the four cases described above, the administration of digitalis induced an acceleration of the ventricular rate in one patient (Case 1) without the appearance of extra systoles or the formation of an abnormal rhythm in the ventricles. Chart 26 shows the maximum and minimum rates in relation to a massive dose of 1.9 grams powdered digitalis ('B' preparation, referred to previously). As in the other patients, this woman was confined to bed throughout the whole period of observation and the increase in rate cannot be attributed to exertion, any other drug (for none was administered) nor to pyrexia. Vomiting occurred on two days but this must in itself be unrelated to the acceleration for after the gastric symptoms had subsided the ventricular rate continued faster than before the drug was given. Electrocardiograms demonstrated that the increase in rate was due to an increase of the natural frequency of the ventricular pace-maker. Extrasystoles were not observed and the QRS group of waves remained of constant form through the experimental period. The height of the T wave in leads 1 and 2 was reduced, and T3, formerly negative, became upright under/
under the influence of digitalis. The auricular rate was not significantly altered.

As well as increasing the rate of the ventricles, digitalis had a very marked effect on the myocardium in this woman. The clinical note made the day after receiving the large dose of digitalis reads: 'The apex beat is now easily felt as a prolonged localised thrust in the 5th interspace; the area of cardiac dulness appears to have decreased; the heart sounds are more easily heard; the systolic murmur at the apex is longer, harsher and more widely propagated. The most striking change is in the character of the apex beat, formerly difficult and almost impossible to detect, it is now obvious'.

It would therefore appear that, while digitalis may slow the ventricular rate in complete block, yet in certain cases, quickening may occur. The acceleration observed in this woman perhaps finds its counterpart in the experimental findings, though VAN EGMOND (58) was led to believe that the quickening which he observed in animals might be attributed to extra-systolic irregularities and later to an independent ventricular rhythm of toxic origin. In this patient an explanation for the acceleration may be found in the fact that, in contrast to those patients/
patients in whom the drug induced slowing, the pre-existing rate of the ventricles was already very slow, slower than that in the four patients referred to above. As shown in Chart 26 the minimum ventricular rate observed in the control period was 22 beats per minute. It has already been shown that at such an initial frequency atropine may produce little or no acceleration. This implies that the lowest rhythmic levels are virtually outside vagal control, and hence digitalis, if it act through central inhibition, is to all intents incapable of inducing a further degree of slowing. It would therefore appear that one factor in regulating the response of the independent ventricular rhythm to full doses of digitalis lies in the pre-existing rate of the ventricles. In this respect the reaction to digitalis closely resembles that already described in detail for atropine and adrenalin. GILCHRIST and LYON (88) have shown that in patients suffering from auricular fibrilation and heart failure, the rate of the ventricles at the time at which digitalis is administered largely determines the amount of slowing induced. It would appear that some similar relationship between initial rate and response also exists in complete heart-block. It is reasonable to suppose that ultimately such a slow rate of ventricular beating may be encountered/
encountered that the administration of a full dose of digitalis is incapable of producing further slowing. Actually at a minimum initial rate of 22, ventricular acceleration was induced by a full dose of digitalis in the patient under consideration. This suggests that a critical rate exists below which adequate therapeutic doses of digitalis are powerless to slow the ventricles. The figures at our disposal are insufficient to allow of the exact determination of this critical level, and indeed it may possibly vary in different hearts. In those cases (TABLE XXIV) who responded by a decrease in rate, the lowest frequency observed after the drug was 26 beats per minute. In Case 1, the minimum rate, before giving the drug, was 22. This suggests that at a ventricular rate about the neighbourhood of 24 beats per minute digitalis is powerless to produce further slowing, and probably by increasing the irritability of the ventricles, an actual acceleration of the idio-ventricular rhythm is induced. The facts at our disposal therefore suggest that in complete heart-block, the effect of digitalisation varies with the state of the ventricles at the time the drug is administered. A critical rate exists below which digitalis produces acceleration rather than slowing, and/
and above which retardation is induced, the amount of slowing being greater the faster the initial idio-ventricular rate. The fact that digitalis is capable of reducing the ventricular rate would support the contention that vagal influences may be active at least in certain cases of complete heart-block.

It is only necessary to mention briefly the two patients (Cases 4 and 6) in whom no definite change in the rate was observed. Case 4 received 60 minims of tincture of digitalis daily for four weeks. This man was of a nervous temperament, introspective and unduly anxious about his personal welfare. He insisted on getting up from bed in order to walk about the ward for the sake of exercise. As his ventricles responded promptly to physical exertion, the observations made on the rate of his pulse during the period of digitalisation are worthless. The probability is, however, that digitalis did tend to slow his pulse, for the rate increased after the drug was discontinued. Case 6, received small doses of the tincture, 10 minims t.i.d., for ten days without appreciable influence on the ventricular rate. In spite of being incompletely digitalised, auricular fibrillation replaced the regular S-A stimulus on the ninth day of treatment.

HEWLETT/
HEWLETT and BARRINGER (108) and NEUHOF (162) have noted the disappearance of the 'a' wave from polygraphic curves in two heart-block patients under treatment with digitalis, and more recently SCHWARTZ (193) has described the occurrence of auricular fibrillation in two cases of complete block during the administration of excessive doses of digitalis. The abrupt appearance of auricular fibrillation after full doses of digitalis to patients with normal sinus rhythm is an uncommon but well recognised effect of the drug on the auricular musculature. It has been investigated by RESNIK (177) and noted by the author in several cases (85). Whether or not digitalis was responsible for the inception of the abnormal auricular rhythm in the patient under consideration must remain problematical. The change in auricular rhythm took place before the man had received a full digitalising dose. It has not been possible to secure further electrocardiograms and whether the disorder persisted after the digitalis treatment was discontinued is not known.
THE TOXIC ACTION OF DIGITALIS IN COMPLETE HEART-BLOCK.

SCHWARTZ (193) has recently described the reaction of three patients, suffering from complete block, to excessive doses of digitalis. An increase in rate, he regards, as a sign of serious significance especially if this is brought about by an excessive production of multiple extra systolic irregularities. In certain instances the stimulation of multiple ventricular foci resulted in abnormal rhythms of short duration but at excessively high rates.

The example of digitalis intoxication to be reported here is unique in certain respects. The patient (Case 2), in a state of advanced congestive heart failure received a course of digitalis treatment amounting to 60 minims of the tincture daily for eighteen consecutive days without improvement in his clinical condition. Several weeks previously a single massive dose had slowed the ventricular rate. On this occasion, when a total of nine drachms had been administered, electrocardiograms demonstrated an increase in the rate of the ventricles and a change in the form of the ventricular complexes.
complexes. FIG. 15 shows an electrocardiogram recorded on the ninth day of digitalis therapy. It may be compared with that (FIG. 11) taken before treatment. The rate of the ventricles is 71.3. As the man's general condition had not appreciably deteriorated and as there was no complaint of headache or nausea and no vomiting it was decided to continue the drug. Further electrocardiograms demonstrated that the abnormal rhythm must have been of relatively brief duration for it was not again recorded. The drug was continued in the same dosage for a further period of nine days. At the end of this time the ventricular rate suddenly increased to 130 per minute. This tachycardia of the ventricles lasted for hours at a time and recurred at irregular intervals for five days after the administration of digitalis had been discontinued.

The man did not complain of palpitation and beyond an increase in the amount of congestion and some additional difficulty in breathing he had no particular symptoms to draw attention to the state of the heart. He did not suffer from syncopeal attacks or epileptiform seizures during the runs of tachycardia and was indeed quite unconscious/
292a

Plath 6
unconscious of its presence. The larger attacks of rapid heart action were associated with some mental confusion. He also suffered from double incontinence and had delusions regarding his neighbours in the ward during the days on which the tachycardia was observed.

Electrocardiograms taken during the attacks of rapid heart action are of interest in that they reveal a form of paroxysmal ventricular tachycardia. After many attempts, a record was secured which demonstrated the end of a paroxysm and the resumption of the slow idio-ventricular rhythm. This electrocardiogram, which is a unique example of the association of paroxysmal tachycardia with complete heart-block, is reproduced in PLATE 6. The paroxysm ends at a rate of 80.2 and after a pause of 5.25 seconds, the slow idio-ventricular rhythm is resumed at a rate of 25 beats per minute. The auricular waves are obscured by tremor and their rate can be determined only with difficulty. The white markings in the figure are placed over possible auricular contractions.

After the discontinuation of digitalis treatment, the attacks of abnormal ventricular action gradually subsided during the course of four or five days. The man's general condition progressively/
progressively deteriorated and eight weeks later he gradually sank and died in a state of advanced congestive failure with uraemic symptoms. In the hope of restoring compensation there is no doubt that an excessive amount of digitalis was administered to this patient. Strangely enough the toxic action of the drug was unaccompanied by the production of ventricular extra systoles, either singly or in groups and the abnormal rhythm revealed by electrocardiographic examination was the only evidence of aberrant ventricular action. Symptomatically increasing peripheral congestion and some mental confusion were the only indications of the additional burden imposed on the circulation by the abnormal cardiac action.

This is not the place to discuss fully the nature and mechanism of ventricular tachycardia but it is right to refer to the fact that over dosage with digitalis is the commonest precipitating cause for attacks. LEA (132) in 1905 noticed paroxysmal tachycardia in one case of complete block under treatment with digitalis, but it was not possible to determine the precise nature of the rapid rhythm. SCHWENSEN (196) in 1921 was the first to demonstrate that ventricular tachycardia could be a manifestation of digitalis intoxication and/
and in 1926, in the course of personal work on this subject (84), it was shown that other factors were also involved important amongst which was the nutrition of the heart muscle. All the individuals in whom this disorder of rhythm has been attributed to digitalis have suffered from congestive failure. The present case is no exception to this rule. Recently DAVIS (51) has attempted to show that in normal individuals the occurrence of ventricular tachycardia is prevented by a healthy conducting system which distributes impulses very rapidly to all parts of both ventricles and thereby induces a uniform state of refractoriness sufficient to prevent the formation of continuous runs of ectopic beats. From an analysis of the literature Davis believes that ventricular tachycardia is prone to occur when the conducting tissues are depressed by drugs or disease. In spite of this no instance is quoted where this arrhythmia was observed in the presence of complete heart-block. In the present state of knowledge, any discussion on the mechanism of the action of digitalis on the ventricular musculature, embarrased by such a complex process as advancing congestive heart failure, must needs be purely speculative.

SUMMARY/
A review of the literature indicates that while digitalis may induce some ventricular acceleration in complete heart-block under experimental conditions, yet as a general rule the idio-ventricular rate has been unaltered by therapeutic doses in man. The drug is not contra-indicated in complete heart-block for indeed many authors have used digitalis with great benefit.

Spontaneous fluctuations in the rate of the slowly beating ventricles make the effect of the drug on the heart rate difficult to assess without an adequate control period. For this reason it is impossible to determine the exact effect of the drug on the rate of the ventricles in many of the cases reported in the literature.

A study of the response of seven cases of complete heart-block to full therapeutic doses of digitalis has been made after an adequate control period. Accurate counts of the pulse and heart rate were made at two hourly intervals and the results checked by repeated electrocardiograms.

Employing these methods, it has been found that, when the ventricular rate is greater than 24 per minute, full digitalisation produces a degree of ventricular/
ventricular slowing depending upon the rate existing before the drug is administered.

At a ventricular rate of less than 24 per minute, full digitalisation is accompanied by a slight degree of acceleration.

The available facts suggest that the action of digitalis on the ventricular rate in complete heart-block is very much the reverse of that described for atropine. It has been previously shown that the greatest release after atropine occurs at the highest initial rates and vice versa. Down to a critical level (in the neighbourhood of 24 per minute) digitalis produces some degree of slowing of the ventricular action, the faster the initial rate the greater the degree of retardation.

This suggests that the parasympathetic system is stimulated in complete heart-block by digitalis, just as it is in other conditions in which the drug is used, and as a result of augmented vagal inhibition the ventricular rate in the blocked heart is effectively retarded.

Clinically the administration of full therapeutic doses of digitalis causes a considerable increase in the force of ventricular systole, and in all probability the output of blood per beat is increased in those individuals who derive benefit from digitalisation.
A unique case of ventricular tachycardia occurring in the presence of complete heart-block is recorded. This arrhythmia was precipitated by over dosage with digitalis. Congestive heart failure was accentuated by the presence of this disordered action of the ventricles. No similar case has been found in the literature.
THE RESPONSE TO BARIUM CHLORIDE.
THE RESPONSE TO BARIUM CHLORIDE.

As an outcome of experimental work on the action of barium on the heart muscle, this drug has recently been recommended for the prevention of the Stokes-Adams syndrome. Nearly fifty years ago BOEHM (15) observed an acceleration of the mammalian heart after small doses of the drug, and also some rise in blood pressure. After larger doses systolic arrest was readily produced. WERTHEIMER and BOULET (223) came to the conclusion that barium in some measure resembled digitalis in that it increased the force of contraction and heightened the excitability of cardiac muscle. ROTHBERGER and WINTERBERG (187) made careful experimental studies concerning the action of the chlorides of barium and calcium from which they concluded that both these drugs increase the irritability of the heart muscle. Extra-systoles in series were common after doses of 50 - 100 mgms. of barium chloride. Larger quantities provoked ventricular fibrillation. These workers came to the conclusion that the pacemakers of the heart (the S-A and A-V nodes) are but little affected by barium and/
and that the increase of rate is largely attributable to extra-systolic irregularities. The observations of ROTHBERGER and WINTERBERG were confirmed by van EGMOND (58) who demonstrated an increase of rate in heart-block preparations.

This experimental work suggested to LEVINE (36) that by increasing the excitability of the heart muscle, barium chloride might lessen the tendency to Stokes-Adams attacks by increasing the irritability of the ventricular muscle. Apparently WILSON and HERMANN (229) were the first to report on the clinical use of the drug in a case of complete heart-block complicated by repeated convulsive syncopal attacks. For 38 days, barium chloride was administered orally in doses of $\frac{1}{2}$ grain thrice daily without benefit. Stokes-Adams attacks continued despite its use. COHN and LEVINE (36) reported three severe cases of the Stokes-Adams syndrome. Barium chloride was administered to each apparently with benefit, but it is admittedly difficult to distinguish between a spontaneous alleviation of the symptoms and a definite effect produced by the drug. Their first case, for instance, had received 40 mgms. thrice daily for only two days when the seizures/
seizures ceased. Adrenalin, 0.3 c.c., was employed repeatedly along with the barium in the second case, and in the third example, while seizures recurred following the withdrawal of the drug yet repeated injections of adrenalin were necessary to bring them again under control. The impression formed by these authors was to the effect that barium, by increasing the ventricular excitability, probably tends to prevent the recurrence of seizures in those people in whom Stokes-Adams attacks are prone to occur. As a result of the favourable opinion expressed by COHN and LEVINE, further cases were soon reported. Reviewing the work of LEVINE (133), LEVINE and MATTON (135), HERMANN and ASHMAN (105), PARSONS-SMITH (170), STRAUSS and MEYER (210) and PRICE and NISSE (174) the conclusion is reached that in general barium chloride appears to have some effect in preventing the recurrence of seizures. On the other hand, when seizures are actually in progress less benefit would appear to result from its use. It is worth noting however that HEARD, MARSHALL and ADAMS (100) in a case of advancing partial block associated with numerous Stokes-Adams attacks, found that 5 grms. of barium chloride/
chloride over a period of three months failed to check a gradual increasing frequency of syncopal attacks. More recently PARSONETT and HYMAN (171) have had absolutely negative results in eight cases of complete block and DRAKE (55) has reported that barium chloride, administrated in adequate dosage, produced no effect on the idio-ventricular rate and failed to influence the frequency of syncopal attacks in one patient. The more recent clinical reports therefore cast a doubt on the efficacy of this drug and fail to substantiate the optimistic reports of some years ago.

It would appear that little is known concerning the action of barium chloride on the healthy human heart. The literature contains no studies on the drug's action in uncomplicated cases of heart-block in man. It would be natural to suppose that, if the drug in non-toxic doses is possessed of the power of so increasing the irritability of the myocardium as to prevent attacks of ventricular standstill, then it should be possible to demonstrate either an increase in the idioventricular rhythm during the use, or at least a production of extra-systolic irregularities, similar to/
to those described in the experimental studies of ROTHBERGER and WINTERBERG (187). Apart from isolated case reports of its use in the Stokes-Adams syndrome, no attempt has been made to study the effect of the drug on the independent ventricular rhythm of complete block in man.

**THE ACTION of BARIUM CHLORIDE in COMPLETE HEART-BLOCK.**

As a test of its capabilities barium chloride was administered to four of the patients in this series, in none of whom had Stokes-Adams seizures been recently experienced. Each individual was confined to bed for at least ten days before the administration of the drug was commenced. This served as a control period during which the pulse was counted at two hourly intervals, the blood-pressure recorded, and electrocardiograms made day by day. Similar methods were employed during the days on which the drug was taken and also for a corresponding period after its administration had been discontinued. Cases 1, 5, 10 and 14 were studied in this way. A short trial of the drug was/
was also made in one patient under Professor Ritchie's care during a series of repeated Stokes-Adams attacks of great severity (Case 7).

Case 1 received 40 mgms., at 6 hourly intervals for 2 days, the dose being then increased to 60 mgms. for the succeeding 9 days. This woman received in all 2.48 grms. of the drug. No change either in the pulse rate, or blood pressure was observed. The electrocardiograms remained unaltered. Extra-systoles were not observed.

Larger doses were employed in testing the response of Case 5. This man received 240 mgms. in the 24 hours for three days, followed by double this amount for a further period of three days. In spite of the administration of doses in excess of those recommended by COHN and LEVINE (36), again no effect on the rate of the auricles or ventricles was recorded. Extra-systoles were not observed.

Still larger doses were used in Case 10. For three days this woman received 60 mgms. thrice daily, followed by 90 mgms. thrice daily for a similar period and finally 120 mgms. thrice daily for 6 days. No appreciable alteration was observed in the rate of the pulse, though occasional extra-systoles were/
were induced. In 12 days she received a total of 4.11 grms.

Negative results were also obtained in Case 14. For one week this woman received 120 mgms. daily and for a second similar period double this dose. The rate of the ventricles was unaltered and extra-systoles were not observed.

It is only necessary to state that all treatment directed to the prevention of the Stokes-Adams attacks in Case 7 proved unavailing. In spite of repeated doses of barium chloride, adrenalin, and ephedrine seizures recurred repeatedly. In the last 48 hours of life this man had approximately 170 attacks of syncope with convulsions, his condition towards the end resembling the status epilepticus. Attack succeeded attack despite every endeavour to control the ventricular rate.

SUMMARY.
SUMMARY.

The effect of barium chloride on the ventricular rate has been tested in five cases of complete heart-block. Four patients, were entirely free of Stokes-Adams attacks when the drug was in use.

The doses employed were greater than those which have been recommended for the prevention of Stokes-Adams attacks. In spite of this no alteration in the rate of the idio-ventricular rhythm was observed. Extra-systoles were produced in but one patient.

Previous studies have shown that the ventricles in these four patients responded to adrenalin, and to atropine and, with the exception of one patient, all also showed an acceleration after ephedrine. Barium chloride in full doses therefore failed to induce an increase of the idio-ventricular rate in patients in whom it was known that the ventricles were capable of responding to appropriate stimulation. The irritability of the myocardium - as judged by the appearance of extra-systoles - was apparently increased in one case.
No change was observed in the four of the Q R S T group. If barium acts directly on the ventricular muscle its effect is probably entirely different from that of digitalis. Barium produced no change in the T waves such as has been attributed to the action of digitalis on the myocardium.

In one exceptionally severe example of the Stokes-Adams syndrome, barium chloride was without influence on the frequency or duration of the seizures. Ephedrine and adrenalin were equally unsuccessful in this patient.

It would seem justifiable to conclude that barium chloride can have but little if any influence in increasing the rhythmicity of the idio-ventricular centre even in doses greater than those originally recommended. In the absence of positive findings it is difficult to credit the drug with the power of preventing Stokes-Adams attacks. If barium chloride be employed in sufficient quantity to produce multiple and irregular extra-systoles, the liability to Stokes-Adams attacks will probably be increased (as result of exhaustion of the muscle and interference from a number of irritable centres) rather than decreased.

The studies reported here fail to support the suggestion that the physician can place any reliance in barium chloride as a means of preventing Stokes-Adams attacks.
RESPONSE TO EXERCISE.
RESPONSE TO EXERCISE.

In earlier pages reference has been made to the effects of exercise on the heart of the healthy subject and it has already been shown that in the majority of the heart-block patients, included in the present study, auricular and ventricular acceleration was readily induced by a short period of physical exertion. It is proposed to discuss certain aspects of this subject in a little more detail in the present section.

It is universally agreed that, under the influence of physical effort, the increased demands of the muscles for oxygen become satisfied in a number of different ways. The production of heat in the working muscles, the local increase in the hydrogen ion concentration, and the redistribution of blood flow throughout the body have the effect of facilitating the increased utilisation of oxygen by the active muscles. Moreover, by the opening up of new capillaries and by the delivery of oxygen at an increased tension, full advantage is made of a larger diffusion surface and the demands of the tissues for oxygen are more readily satisfied.

Fundamentally/
Fundamentally the supply of an adequate amount of oxygen to the tissues depends on the volume of blood flow and the efficiency of the heart as a pump. Local circulatory readjustments in the active tissues are subsidiary to an increase in the minute volume output of the heart. In the readjustment of the circulation rate to the demands for oxygen there are two main factors concerned namely, the output of blood per beat, and the rate of the heart. Both vary considerably in health and also in disease. GROLLMANN (92a), using the acetylene method, has recently demonstrated that the cardiac output of normal individuals per square meter of body-surface is $2.2 \pm 0.3$ liters per minute (and its average duration from the mean only 6.4 per cent) under basal resting conditions. Under the influence of muscular exercise, such as climbing steps or riding a bicycle ergometer the cardiac output may increase enormously. A rise from 4.2 to 37.3 liters per minute, with an associated increase in the pulse rate from 70 to 179 has been observed (CHRISTENSEN (28)). In other words the augmented blood flow of physical exercise is the result not merely of an increase in the pumping rate of the heart, but in an enhanced delivery at each stroke.
While the increased circulation rate is largely an affair of the heart itself (141) and is ultimately dependent on the venous return, yet the autonomic nervous system plays an important part in regulating the heart rate. The sino-auricular node has a rich supply of vagal and sympathetic fibres. Hering's researches (103) led him to the conclusion that the acceleration of the normal heart in response to exertion was due partly to release from vagal inhibition and partly to increased sympathetic stimulation. From the experiments of Favil & White (67) on an individual who was able to produce a voluntary acceleration of the heart it would seem quite definitely established that in man, as in most animals, the normal fluctuations in pulse rate are produced by the combined action of the parasympathetic and sympathetic nerves.

It is therefore not without interest to consider the response of the blocked-heart to exercise. In 1909 Erlanger & Blackman (64) demonstrated quite conclusively that in dogs, surviving by several months the operative destructing of the bundle of His, exercise induced a decided increase in the ventricular rate. After the animals had run about, the rates in three observation were
55.5, 57.0, and 62.2, whereas under resting conditions before exercise they were respectively 49.5, 54.0, and 46.5 per minute. The clinician, however, is inclined to be more guarded in attributing to exercise the power of increasing the ventricular rate in his heart-block patient. (Cowan & Ritchie (41), Mackenzie (150), Vacquez (214) and Wenckebach (222)). Similarly de Graff & Weiss (90) in a study of two cases of complete block were unable to satisfy themselves that the ventricles increased in rate after exercise.

On the other hand an increased pulse rate has been noted as a result of the addition of extra-systolic beats Wenckebach (222) Lian (140) founded a decided acceleration of the ventricular rate in one patient. The usual ventricular rate in this subject (aged 20 years) during a state of bodily rest, was in the neighbourhood of 40 beats per minute. After walking quickly for a few minutes, Lian observed that in succeeding quarter minutes there were 25, 19, 15 and 13 beats, representing a rate of 72 beats in the first minute after the completion of the exercise. Tracings demonstrated complete heart-block.

FREDERICQ/
FRÉDÉRICQ (74) has reported a similar observation. In his patient, an over-weight man, 56 years of age the usual A/V ratio was 75/30. Before an exercise test there were 15 beats in 30 seconds. After physical exertion had been accomplished the ratios in succeeding half minutes were 23, 18 and 16. The two cases just quoted suggest that the ventricular acceleration of complete heart-block may be of very short duration. The corresponding auricular rates are not quoted. ZANDER (141) has published a careful investigation on the effect of exercise on three patients suffering from complete heart-block. In all three a considerable increase in the pulse rate resulted. Electrocardiograms demonstrated that the acceleration was brought about by the appearance of extrasystoles in one instance, but in the two other cases the idio-ventricular rhythm was definitely increased. Similarly LUNDSGAARD (144) found that during muscular work the pulse rates in two patients increased by 15 and 33 per cent respectively. LILJESTRAND & ZANDER (141) after varying amounts of exercise found a rise in ventricular rate from 52 to 109, from 52 to 76, and from 41 to 69 in one patient/
patient in whom the observations were controlled by electrocardiograms. These authors point out that not only is the percentage increase in the rate greater for the ventricles than for the auricles but that the response to the demands of exertion is made as readily and as abruptly as in the normal heart. ALT, WALKER & SMITH (3) have also reported an increase of the idio-ventricular rate after exercise in two patients with complete block.

Apart from the more formal exercise test, it is of interest to discover the influence of the additional muscular exertions imposed by pregnancy and delivery on the heart-block patient. It is well known that pregnancy, by placing an increasing burden on the heart, may unmask unsuspected evidence of cardiac disease. Delivery in itself is a great muscular feat fraught with danger for the cardiac patient. Not many instances of complete heart-block occur in women during the child bearing period, and yet, the fact that several cases are reported where women have gone successfully to term, is sufficient to indicate that the reserve power of the heart in the presence of complete dissociation must vary very considerably in different individuals. HERMANN & KING (106) report the case of a woman who had six deliveries without/
without incident despite the presence of complete block. DRESSLER (56) in reporting two cases of pregnancy and spontaneous delivery in women suffering from complete heart-block, mentions that one patient was in labour for twenty-three hours during which time the ventricular rate rose from 36 to 52 beats per minute. After a spontaneous delivery the rate fell rapidly to 40. TITUS & STEVENS (213) have given a full account of the successful pregnancy, delivery and puerperium in a woman aged 25, who had had a slow pulse from the age of 12. Electrocardiograms demonstrated complete heart-block. During pregnancy there was a slight rise in the auricular and ventricular rates under uniform conditions of observation. Shortly before labour the auricular and ventricular rates were 68 and 45. During labour they varied from 88 to 72 and 58 to 50 respectively. After the birth of the child the auricular rate was 57 and the ventricular 38. It is true that factors other than the muscular straining of the hours of the confinement, may well influence the rate of the heart, but the fluctuations in rate coinciding with the spasmodic physical efforts, indicate that muscular effort must be of prime importance in determining the changes in the rate of the heart.

It/
It would therefore appear that an inability to accelerate the ventricle is not an essential feature of the heart in the presence of complete block. Doubtless the power varies very considerably in different cases. It can hardly be expected that the response of the young person (such as ZANDER's patient aged 9 years) in good health apart from the block, will be identical with that of the elderly arterio-sclerotic subject (such as Case 2 in the present series) handicapped by a gradually increasing amount of peripheral congestion. In the latter instance the myocardium is exhausted, the output per beat is minimal, and the muscular effort of breathing provides in itself more exercise than is desirable. Under such circumstances it is reasonable to suppose that the heart is already responding (in an unsatisfactory way) to the effects of exercise, the best available balance being struck between output per beat and ventricular rate.
EXPERIMENTAL METHODS.

Observations on the effect of exercise on the rate of the heart have been made in twelve of the fourteen patients who comprise the present study. Cases No. 7 and 9 (both examples of intermittent complete-block) were not tested owing to the frequency of Stokes-Adams seizures and the very unsatisfactory state of their general health.

In three patients (Cases No. 2, 10 and 11) a modified test was used. Two (Cases 2 and 11) were confined to bed owing to the presence of peripheral congestion, and for these reasons, the results obtained are not comparable to those in the rest of the series. Case 10 was a feeble old woman, who was unable to perform the routine test. One further case (No. 14) has been subjected to an exercise test on many occasions, the rhythm of the heart being then of the nature of a partial block of the 2 to 1 or 3 to 1 type. Although, at the date of writing, electrocardiograms reveal the gradual development of complete block in this woman, sufficient time has not elapsed to conclude with certainty that the block is likely to remain of the permanent type. Indeed
it was possible to induce a 3 to 1 rhythm by the administration of ephedrine during complete block in this patient. The changing nature of the defect in this case makes it undesirable to include her response to exercise with the rest of the series. The results obtained in eight individuals (Cases 1, 3, 4, 5, 6, 8, 12 and 13) may therefore be considered suitable for comparison, as each was sufficiently fit to undertake the amount of exertion imposed by the test.

It has been shown by GROLLMAN (92a) that the nature of the heart's response to exercise varies considerably according to the nature of the work performed, and in the present study it seemed desirable that the type of muscular exercise should be one with which all the patients were well accustomed. Many methods of gauging the heart's response to effort are described in the literature but it seemed quite out of the question to expect elderly patients either to swing dumb-bells, work an ergometer or hurry up and down stairs. In recording the effects of exercise in the heart-block patient it was necessary to devise a simple test, which could be done under continuous observation and which for convenience could be/
be performed as close as possible to the recording apparatus. The method adopted was a modification of that described by MASTER & OPPENHEIMER (154).

After the patient had rested on a couch for about 20 minutes in the examination room until the blood pressure and pulse rate had assumed fairly steady levels, a strip of electrocardiographic film was exposed, generally by Lead I. The electrodes, which consisted of jars of salines into which the hands are immersed, were found to be particularly convenient, as on the completion of exercise the patient had simply to return to the couch, lie down and place the hands in the saline solution. In this way the time elapsing between the end of exercise and the first electrocardiogram was reduced to a minimum. By having the film already running, and the deflection of the fibre previously standardised, it was possible to secure accurate records of the heart's rate within 5 seconds of completing the test. The alternative method of having the leads attached to the arms throughout the test was found cumbersome in that the wires leading to the recording apparatus introduced an element of confusion, handicapped the patient in the performance of the/
the exercise, and aroused an unnatural state of apprehension or excitement. It was found essential to secure electrocardiograms with the minimum amount of delay and, as the technique outlined above permitted records to be obtained within 5 seconds of performing the test, satisfactory results were obtained. The film camera was employed and with the aid of a stop-watch exposures were made at regular intervals. As a general rule the film was allowed to run until 30 seconds had elapsed from the time of completing the test. Thereafter further records were made at alternate 15 second intervals for 1½ minutes and for such additional periods as seemed desirable.

The test of physical activity consisted in ascending and descending two steps a variable number of times in a given period. The apparatus, which was made for the purpose consists of a two-step contrivance, the patient climbing up two steps on one side and down two steps on the other. Each step is exactly nine inches high (and about 22 inches wide). The patients were instructed to climb over the steps and back again, as quickly as possible, in the space of 1½ minutes. By making the total height of the two steps 1½ feet, and by limiting the exercise/
exercise period to 1½ minutes, the calculation of the amount of work done per minute is greatly simplified. Since, during the descent of the steps, there is no actual lift of the body-weight against gravity and any work done will be proportionate to work done on the ascent, the descent is omitted in the calculation of the foot-pounds of work performed. A man of 140 lbs. weight climbing 1½ feet 20 times will do \((140 \times 20 \times 1\frac{1}{2})\) 4200 foot-pounds in 1½ minutes or \((140 \times 20)\) 2800 foot-pounds per minute.

This plan, whereby the amount of work done can be compared with the pulse rates changes, forms the basis of the test of circulatory efficiency devised by MASTER & OPPENHEIMER (154). It has the great advantage that the method is both simple and convenient in its execution, and provides a ready means of regulating the amount of work done in a given time.

Emphasis has been placed on the technique of recording the response of the blocked-heart to exercise, for the reason that the acceleration of the ventricles are often of such short duration that unless accurate and systematic methods are employed, the quickening induced may easily escape detection.
\[ y = 0.41x - 3.13 \]

\[ \sum xy = +0.930 \]
The method outlined above has the further advantage that, from the electrocardiograms recorded in the recovery period, the auricular and ventricular rates can be calculated over similar periods of time. Further, the electrocardiogram reveals any change in the site of impulse formation or conduction.

In performing the test, the patient was instructed to climb the steps as quickly as possible. All co-operated to the best of their ability and, in each instance, sufficient physical exertion was undertaken to induce dyspnoea, and even considerable orthopnoea. Two patients suffered from praecordial pain immediately after the test (Cases No. 3 and 8). Their co-operation was such that all were glad to return to the couch on completing the 1½ minutes of exercise.
**TABLE XXV.**

<table>
<thead>
<tr>
<th>CASE NO. &amp; INITS.</th>
<th>AURICULAR RATE</th>
<th>VENTRICULAR RATE</th>
<th>REMARKS, AND WORK DONE.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BEFORE EXERCISE</td>
<td>AFTER EXERCISE</td>
<td>GAIN IN RATE</td>
</tr>
<tr>
<td>1. Mrs T. 2a(a)</td>
<td>72.1</td>
<td>129.6</td>
<td>57.5</td>
</tr>
<tr>
<td></td>
<td>89.6</td>
<td>144.0</td>
<td>54.4</td>
</tr>
<tr>
<td>2a(b)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. D.C. 2a</td>
<td>102.0</td>
<td>128.7</td>
<td>26.7</td>
</tr>
<tr>
<td></td>
<td>50.6</td>
<td>97.4</td>
<td>46.8</td>
</tr>
<tr>
<td>3. Mrs H. 1.</td>
<td>71.1</td>
<td>110.0</td>
<td>38.9</td>
</tr>
<tr>
<td></td>
<td>73.1</td>
<td>98.3</td>
<td>25.2</td>
</tr>
<tr>
<td>4. W.T. 2a</td>
<td>67.3</td>
<td>116.3</td>
<td>49.0</td>
</tr>
<tr>
<td>5. A.H. 2b(a)</td>
<td>66.3</td>
<td>111.3</td>
<td>45.0</td>
</tr>
<tr>
<td>6. J.W. 2b</td>
<td>55.6</td>
<td>131.0</td>
<td>75.4</td>
</tr>
<tr>
<td>7. J.B. 2b</td>
<td>79.2</td>
<td>123.8</td>
<td>44.6</td>
</tr>
<tr>
<td></td>
<td>55.2</td>
<td>68.6</td>
<td>13.4</td>
</tr>
<tr>
<td>8. J.D. 2b</td>
<td>73.1</td>
<td>88.3</td>
<td>15.2</td>
</tr>
<tr>
<td></td>
<td>55.2</td>
<td>68.6</td>
<td>13.4</td>
</tr>
<tr>
<td></td>
<td>48.7</td>
<td>82.9</td>
<td>34.2</td>
</tr>
<tr>
<td></td>
<td>39.8</td>
<td>90.0</td>
<td>50.2</td>
</tr>
</tbody>
</table>

**TABLE XXV.** To show the effect of exercise on the auricular and ventricular rates. In each instance a quickening of the ventricular rate was induced.
THE RESPONSE OF THE VENTRICLES TO EXERCISE.

From the long strips of film recorded during the period of recovery from the immediate effects of exercise, the auricular and ventricular rates have been calculated and compared with the time elapsing from the end of exercise. In a previous section the rate of the ventricles after exercise has been considered in relation to the minimum resting rate - the difference between these two extremes being defined as the maximum natural range in rate. It was further demonstrated that an inverse relation existed between the ventricular and auricular ranges. When the ventricle made but a small response to the demands of exercise the increase in the auricular rate was great and vice versa. Further those individuals who exhibited a large ventricular range were for the most part those in whom the efficiency of the circulatory system as a whole was maximum. In the present section it is proposed to present the rates of auricles and ventricles immediately before exercise in contrast with those existing during the 20 seconds period after the test. The results of eleven exercise tests in eight patients are presented in the adjoining TABLE (No. XXV)
<table>
<thead>
<tr>
<th>GROUP</th>
<th>CASE NO. &amp; INITS.</th>
<th>FOOT POUNDS PER MIN.</th>
<th>PERCENT VENTRICULAR GAIN</th>
<th>AVERAGE FOR GROUP FOOT POUNDS % VENTRICULAR GAIN</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>13. Mrs H.</td>
<td>2975</td>
<td>126.1</td>
<td>2975</td>
</tr>
<tr>
<td>2a</td>
<td>1. Mrs T.</td>
<td>2500</td>
<td>50.0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3. D.C.</td>
<td>3519</td>
<td>31.7</td>
<td></td>
</tr>
<tr>
<td></td>
<td>4. W.T.</td>
<td>3249</td>
<td>6.9</td>
<td></td>
</tr>
<tr>
<td>12.</td>
<td>J.D.</td>
<td>2790</td>
<td>70.1</td>
<td>3015</td>
</tr>
<tr>
<td>2b</td>
<td>5 A.H.</td>
<td>2950</td>
<td>3.4</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>J.W.</td>
<td>3021</td>
<td>29.5</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>J.B.</td>
<td>2346</td>
<td>22.4</td>
<td>2772</td>
</tr>
</tbody>
</table>

TABLE XXVI. To show the average percentage ventricular acceleration induced by exertion in relation to the various grades, (Group 1, 2a, and 2b) of functional efficiency.
That an adequate amount of exertion was undertake on each experiment is evident from the calculation of the work done, which varied from 2052 foot-pounds to 3519 foot-pounds per minute. If the patients be graded according to their functional efficiency, as described on page , it is found that on the average the amount of work performed varied but slightly in the different groups. While each individual performed as much work as possible in a given time the degree of distress (dyspnoea and discomfort) was in general greater in group 2b than in group 1. Further the amount of acceleration induced was maximum in Group 1 and minimum in Group 2b. It is hardly necessary to point out that those patients classified as Group 3 were incapable of performing this test. It is of interest to compare the amount of work done, the percentage ventricular acceleration and the functional efficiency, as shown in the following TABLE (No.XXVI). In spite of almost equal amounts of work in the different groups the degree of ventricular acceleration induced shows on the average a progressive decline as functional efficiency diminishes.

Of the eight patients studied in this way,
TABLE XVI shows that in two individuals (Cases 4 and 5) the degree of ventricular acceleration was considerably less than in the others. Both these men have been previously classified as belonging to the degenerative group in which the available clinical evidence suggested that the lesion causing the block was primarily of vascular origin. They were each in their sixty-fifth year. The available facts are scanty from which to draw conclusions but it may be that the response of the blocked heart to exertion is influenced in some measure by the age of the patient, the presence or absence of coronary artery disease, and the nature of the lesion responsible for the production of the dissociation.

In earlier studies some reference has been made to blood-pressure. It was hoped that during the course of the recovery of the heart from exercise that comparable blood-pressure readings might be obtained. In actual practice it was not found possible to secure accurate figures. The acceleration of the ventricles is of such a fleeting nature that by the time pressure readings were secured the acceleration had frequently subsided. In general the systolic pressure was increased by
Reference has already been made to a possible relationship between the maximum ventricular range and the pulse pressure at rest. It is of interest to find that a high degree of correlation exists between the systolic pressure under prolonged resting conditions and the maximum ventricular rate attained after exercise. In arriving at this conclusion it is right to point out that the results obtained in two individuals (Cases 12 and 13) have been omitted, quite an exceptional amount of acceleration (70 and 126 per cent of the pre-existing rate, respectively) being recorded. On the other hand three subjects, (Cases 2, 10 and 11) omitted from TABLES 25 and 26 have been included. They were of such feeble constitution that they were unable to perform the standard test. Case 2 suffered from advanced congestive heart failure and being confined to bed in the orthopnoeic position the effect of bending the trunk backwards and forwards at the hips about half-a-dozen times was as much effort as could be accomplished. Case 10 was a frail old woman 84 years of age. She was quite unable to carry out the usual test and walking slowly across/
<table>
<thead>
<tr>
<th>CASE NO. &amp; INIT'S.</th>
<th>AURICULAR RATE</th>
<th>VENTRICULAR RATE</th>
<th>REMARKS</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BEFORE EXERCISE</td>
<td>AFTER EXERCISE</td>
<td>IN RATE</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. W.W.(a)</td>
<td>69.9</td>
<td>88.4</td>
<td>18.5</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>71.5</td>
<td>83.5</td>
<td>12.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10 Mrs. C.</td>
<td>Auricular fibrillation</td>
<td>26.8</td>
<td>27.3</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11. Mrs. D.</td>
<td>84.5</td>
<td>111.1</td>
<td>26.6</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

TABLE XXVII. To show the effect of a modified exercise test in three individuals unable to perform the standard test. In functional efficiency all belonged to Group 3.
across the examination room with some assistance from her nurse, was as much exertion as was possible for her to undertake. Case 11, likewise suffered from a slight degree of congestive heart failure. This woman was able to walk once across the examination room. The effect on the auricles and ventricles of these three patients is recorded in the accompanying TABLE XXVII.

In each of the four experiments shown in TABLE XXVII definite auricular quickening was induced. The ventricular response was slight - being minimal in Case 11. All were arterio-sclerotic subjects over 70 years of age and all have since died.

In comparing the systolic blood pressure at rest with the maximum ventricular rate, the relative data have been taken from the records of nine patients - (Cases 1, 2, 3, 4, 5, 6, 8, 10 and 11). The actual figures are shown in TABLE XXVII and have been plotted in GRAPH 27.

Applying mathematical methods it is found that in these individuals a remarkably high degree of positive correlation exists between the resting systolic pressure and the maximum rate after exercise, the coefficient being +0.930. The regression line/
<table>
<thead>
<tr>
<th>CASE NO. &amp; INITS.</th>
<th>SYSTOLIC BLOOD-PRESSURE AFTER A PROLONGED REST (X)</th>
<th>MAXIMUM VENTRICULAR RATE RECORDED AFTER AN EXERCISE TEST. (Y)</th>
<th>REMARKS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Mrs. T.</td>
<td>165</td>
<td>33.0</td>
<td></td>
</tr>
<tr>
<td>2. W.W.</td>
<td>180</td>
<td>35.0</td>
<td>Died some weeks later.</td>
</tr>
<tr>
<td>3. W.C.</td>
<td>240</td>
<td>57.4</td>
<td>Aortic regurgitation.</td>
</tr>
<tr>
<td>4. W.T.</td>
<td>220</td>
<td>46.2</td>
<td></td>
</tr>
<tr>
<td>5. A.H.</td>
<td>162</td>
<td>36.0</td>
<td></td>
</tr>
<tr>
<td>6. J.W.</td>
<td>158</td>
<td>30.6</td>
<td></td>
</tr>
<tr>
<td>8. J.B.</td>
<td>208</td>
<td>43.2</td>
<td>Aortic regurgitation and auricular fibrillation.</td>
</tr>
<tr>
<td>10. Mrs. C.</td>
<td>170</td>
<td>27.3</td>
<td>Died some weeks later.</td>
</tr>
<tr>
<td>11. Mrs. D.</td>
<td>200</td>
<td>40.4</td>
<td>Died some months later.</td>
</tr>
</tbody>
</table>

**TABLE XXVIII.** To show the minimum systolic blood-pressure at rest and the maximum ventricular rate (exclusive of any extra-systoles) in nine cases of permanent complete heart-block.
line, entered in the chart and drawn to the formula
\[ y = 0.14x - 3.13, \]
represents the optimum ventricular rate after exercise for a given minimum systolic blood-pressure. Similar methods of analysis demonstrated that a less perfect significant relationship (Coefficient of Correlation +0.781) existed between the diastolic pressure and the minimum ventricular rate. This implies that the pulse pressure at rest (i.e. the systolic pressure less the diastolic pressure) reflects fairly closely the maximum natural range in ventricular rate.

THE COURSE OF THE REACTION TO EXERTION.

The effect of exercise on the ventricular rate is best demonstrated by means of diagrams. Four representative experiments may be selected as representative of the course of the recovery period. Chart 28 (Case 1) shows resting auricular and ventricular rates of 89.6 and 30.3. After 1½ minute's exertion the rates are 144 and 38.1. One minute after the test the ventricular rate has returned to the pre-existing level. The auricular recovery is slower and more gradual.

In Case 6 (Chart 29) exercise induced a great increase in the speed of the auricular beat, the/
the rate rising from a resting level of 55.6 to 131.0 seven seconds after completing the test. The ventricular rate rose from 25.2 to 32.6. Forty-five seconds after the test coupled rhythm commenced and continued throughout the period of observation. The rate of the heart was thereby increased. Nevertheless the inherent ventricular rate showed at successive observations a progressive decline. In this patient, as is demonstrated in PLATE 7, exercise not only increased the inherent idio-ventricular rate, by regular extra systoles transmitted to the radial pulse, also augmented the blood flow.

An example of the effect of exercise on a case of complete heart-block complicated by auricular fibrillation is shown in Chart 30 (Case 8). In this instance the ventricular rate rose from 35.3 to 43.2, 15 seconds after the test. Recovery is more gradual, nearly 3½ minutes elapsing before the ventricular rate returns to the pre-existing level. Sections of the electrocardiograms from which the rates have been calculated are shown in PLATE 8.

Case 13 showed an exceptional reaction to exercise. Seven seconds after completing the test...
30.

AURICULAR FIBRILLATION

VENTRICLES.

J.B.

26. VI. 02.

MINUTES AFTER EXERCISE.
the ventricular rate (as shown in Chart 31) reached 90 per minute. The corresponding auricular rate is difficult to calculate from the electrocardiogram owing to the presence of tremor, but is in the neighbourhood of 110 per minute. Recovery is rapid. Within 1½ minutes auricles and ventricles approximate to their resting rates. Section from the electrocardiograms, obtained after exertion in this case are presented in PLATE 9.

SUMMARY.

With a suitable technique it has been possible to show that physical exertion induces a quickening of the ventricular rate (despite the presence of complete block). In eight individuals, each able to perform a standard step-climbing test, the amount of ventricular acceleration induced varied from 3·4 to 126 per cent of the pre-existing rate. In three individuals, unable to perform the standard test owing to a gross circulatory deficiency, the effect of exercise on the ventricular rate was almost negligible.

In general those individuals, in whom the reserve power of the heart was maximum, reacted by a pronounced/
VENTRICLES,

**RATE PER MINUTE**

**EXERCISE**

**MINUTES AFTER EXERCISE**

**AURICLES**

**VENTRICLES**

_Mrs. H._
pronounced ventricular acceleration when the functional efficiency of the circulatory system as a whole was grossly defective, exercise had considerably less effect on the rate of the ventricles.

In health, it is known, that in order to satisfy the demands of the working muscles for an adequate blood supply, the heart increases not only its rate but also its output of blood at each beat. It is natural to suppose that in complete heart-block the amount of blood ejected at each stroke of the ventricles represents almost, if not quite, their full capacity. The power of the blocked-heart to increase its systolic discharge is therefore limited. In those circumstances in which a great augmentation of the circulation rate is required, successful adaptation must largely depend upon the ability of the ventricles to increase their rate without a reduction in their stroke volume. So well did certain patients perform the exercise test, in spite of but a small increase in ventricular rate, that it seems reasonable to suppose that, the ventricles had still retained in some measure the power of increasing the output per beat.

The
Hamilton
Plate 9
The rate of the recovery process (after a short burst of strenous exertion) varies very considerably in heart-block patients. In some the ventricles have returned to their pre-existing rate within one minute, in others three four or five minutes may elapse before the auricular and ventricular rates return to normal.

Without exception every patient responded to exercise with a greater or less degree of quickening of the ventricular rate.
REMARKS on (1) the EFFECT of TEMPERATURE on the IDIO-VENTRICULAR RATE, on (2) FUNCTIONAL AURICULO-VENTRICULAR DISSOCIATION and on (3) the NATURE of the STOKES-ADAMS SEIZURE.
REMARKS on (1) the EFFECT of TEMPERATURE on the IDIO-VENTRICULAR RATE, on (2) FUNCTIONAL AURICULO-VENTRICULAR DISSOCIATION and on (3) the NATURE of the STOKES-ADAMS SEIZURE.

During the course of the work described in the preceding studies observations have been made on many other aspects of heart-block as opportunities arose. Though incomplete in themselves some of these observations are perhaps of sufficient interest to warrant mention. In certain respects they shed some light on the mechanism of the heart beat in what are relatively uncommon circumstances. Much remains to be discovered regarding the nature of partial block, (and indeed also regarding the control of the ventricles in complete block), and though a large number of experiments have been made in the different form of defective conduction it is not proposed to discuss these at the present time. In the first instance it is intended to refer briefly to the effect of pyrexia in chronic complete heart-block.

I./
I. THE EFFECT OF PYREXIA ON THE IDIO-VENTRICULAR RATE.
I. THE EFFECT OF PYREXIA ON THE IDIO-VENTRICULAR RATE.

The literature contains little or no information regarding the precise effect of a rise of temperature on the idio-ventricular rate. SCHWARTZ (193) for instance states, "while it is known that fever increases the heart rate of patients with normal sinus rhythm, in complete heart-block observations on this point are lacking. It would seem probable that in complete heart-block due to organic involvement of the bundle of His, the factor of pyrexia may be discounted as responsible for increasing the ventricular rate". As will be shown immediately the facts at my disposal are completely at variance with these conclusions.

It was observed that on those occasions on which certain patients had a trifling spontaneous rise of temperature to 98.6 or thereabouts that the ventricular rate appeared to quicken by one or two beats. The opportunities for making more precise observations being scanty, it was decided to test the effect of an artificially induced pyrexia on the ventricular rate.

After an ample control period of 10
<table>
<thead>
<tr>
<th>CASE NO. &amp; INITIALS</th>
<th>BEFORE INOCULATION WITH B. TYPHOSUS</th>
<th>AT HEIGHT OF PYREXIAL PERIOD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>AURICULAR RATE</td>
<td>VENTRICULAR RATE</td>
</tr>
<tr>
<td>1. Mrs T.</td>
<td>69.0</td>
<td>26.0</td>
</tr>
<tr>
<td>5. A.H.</td>
<td>56.5</td>
<td>29.2</td>
</tr>
<tr>
<td>13. Mrs H.</td>
<td>75.7</td>
<td>43.5</td>
</tr>
</tbody>
</table>

TABLE XXIX. To show the effect of a rise in temperature ("protein-shock" reaction) on the ventricular and auricular rates.
or 14 days, during which hourly or two hourly pulse rates and mouth temperatures were recorded, and electrocardiograms registered daily, a "protein-shock" reaction was induced in three patients (Cases 1, 5 and 13). Each patient received a 30 million dose of a stock B. Typhosus vaccine intravenously. Thereafter pulse rate and temperature were accurately recorded at hourly intervals throughout the pyrexial period. It is a common experience that the "incubation" period elapsing between the administration of the vaccine and the onset of pyrexia varies considerably in different individuals. In some, after an hour a rigor occurs and the temperature then rises, in others six or eight hours may elapse before a decided reaction is observed. For this reason it was not always possible to record electrocardiograms during the height of the reaction, but nevertheless by registering the heart beat in association with the temperature reaction observed during the day hours, it was possible to check the pulse counts and derive information regarding the auricular rate. The essential details of three experiments are recorded in TABLE XXIX.
<table>
<thead>
<tr>
<th>CASE NO. &amp; INITIALS</th>
<th>INCREASE IN TEMPERATURE</th>
<th>GAIN IN VENTRICULAR RATE</th>
<th>GAIN IN VENTRICULAR RATE PER °F.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Mrs T.</td>
<td>6.8°</td>
<td>19.0</td>
<td>3.58</td>
</tr>
<tr>
<td>5. A.H.</td>
<td>5.2°</td>
<td>18.8</td>
<td>3.63</td>
</tr>
<tr>
<td>13. Mrs H.</td>
<td>4.8°</td>
<td>20.5</td>
<td>4.27</td>
</tr>
</tbody>
</table>

Average rise in ventricular rate for an increase of 1°F. 3.82 beats per minute.
These figures demonstrate that the blocked heart responds in rate to a rise of temperature. MURRAY LYON (149) has shown that, in the presence of normal sinus rhythm, an increase of 1°F. causes a rise in the pulse rate of 9.13 beats per minute. The figures at our disposal are too few to submit to precise methods of mathematical analysis but they are sufficient to show that a rise of temperature is accompanied by an increased ventricular rate. TABLE XXIX.

From the limited material available, the results suggest that in contrast to the healthy heart, a rise of 1°F is associated with an increase in the ventricular rate of about 4 beats per minute. If it be assumed that under resting conditions the S-A node has a natural frequency of 70 per minute and that a rise of 1°F produces an increase of 9.13 beats, then as a matter of simple proportion the ventricles, with a spontaneous frequency of, say 30, ought to augment their rate by 3.91 beats per minute for a similar elevation of temperature. This is in close agreement with the average value of 3.82 as shown in TABLE XXX. It would however be desirable to submit a large number of corresponding rates and temperatures to exact methods of analysis.
The course of the protein shock reaction in one individual is depicted in Chart (Case 5).

It is well known that changes in temperature have a profound effect on the functional activity of certain tissues. GASKELL (80) showed that by applying heat to the frog's sinus a marked increase in the rate of the heart resulted. Similar observations were made on the influence of temperature on the activity of the sino-auricular node in the mammalian heart by McWILLIAM (152), ADAM (1) and FLACK (69). Moreover, ZAHN (236) has shown that rhythms arising in the auriculo-ventricular node are accelerated by heating and retarded by cooling the region of the node itself. LAUDER BRUNTON (21) and CLARK (32) have concluded that vagal activity is diminished by heat.

From the facts derived from the "protein shock" reaction in three patients suffering from complete heart-block, and from the results of temperature experiments on the isolated heart, it seems reasonable to conclude that the idio-ventricular rhythm is susceptible to changes in body temperature. A rise of 1°F may account for an increase in the ventricular rate of about 4 beats per minute.
II. FUNCTIONAL AURICULO-VENTRICULAR DISSOCIATION.

THE EFFECT OF ADRENALIN.
II. FUNCTIONAL AURICULO-VENTRICULAR DISSOCIATION.

THE EFFECT OF ADRENALIN.

It is not proposed to discuss the full significance of functional dissociation. The condition has now been recognised for several years but it is only recently that much attention has been devoted to it.

The term A-V dissociation denotes the condition in which the auricles and ventricles are independent in their rhythms, the auricles beating in response to their own pacemaker and the ventricles likewise. If the ventricles and auricles are entirely independent in their rhythms then there is complete dissociation but not necessarily complete block. On the other hand, if in the presence of dissociation an occasional ventricular beat is in response to an auricular stimulus the condition is termed dissociation with interference. Dissociation with interference may be called incomplete dissociation. It is analogous to partial block but not identical with it.

Complete A-V block is the most perfect example of A-V dissociation but the terms are by no means/
means synonymous. The distinction lies in the nature of the defect in conduction. In primary or essential block, such as occurs as a result of an organic lesion of the bundle of His, or as a result of digitalis action on the conducting pathway dissociation arises either as a result of destruction of the essential tissues in the first instance, or, in the case of digitalis, as a result of prolongation of the refractory period of the A-V node. In functional dissociation, on the other hand, auricles and ventricles beat independently, as a result not of abnormal prolongation of the refractory period of the A-V node, but on account of the fact that the impulse from the SA node reaches the AV node during the natural refractory period of the latter structure. For this reason a momentary dissociation commonly occurs immediately after a ventricular extrasystole. The first auricular impulse after the extrasystole fails to reach the ventricle, hence the postextrasystolic pause. The ventricle remains quiescent until the second auricular impulse is delivered. The failure of the first auricular impulse is due to the fact that it falls at a time when the bundle tissues exhibit a normal amount of refractoriness/
refractoriness after the premature beat. This is an example not of heart-block but of functional dissociation. Similarly during a paroxysm of ventricular tachycardia the auricles commonly continue to beat at their own inherent rhythm. The A/V ratio may then be, say 70/200. On the subsidence of the paroxysm normal rhythm may be resumed. The independent action of auricles and ventricles during the abnormal rhythm constitutes a functional dissociation, but not a primary or essential block.

Suppose that two independent rhythms exist in the heart, one controlled by the S-A pacemaker and the other by the A-V pacemaker and that primary block does not exist. If the auricular rate is less than or equal to the ventricular, functional dissociation must be present, for the reason that if the auricular rate were greater than the ventricular then, in the absence of block, the ventricles would respond to the auricular impulse. On the other hand it is possible that functional dissociation may be associated with a primary partial block.

Consider Case 12. FIGS. 16 and 17 are typical of many and might be taken to represent complete block. FIG. 18 was recorded in/
Figure 18.
(Case 12.)

Auricles:

P wave 17.4 2 16.8 3 16.7 4 16.4 5 18.0 6 18.0 7 18.7 8 18.3 9 17.0 10

Ventricles:

R1 20.0 20.1 19.9 19.7 19.8 19.9 19.9 19.9 26.9 9

Time measurements: 1 = 0.04 sec. in both diagrams.

Figure 19.
(Case 12.)

Auricles:

P wave 21.3 2 19.3 3 17.7 4 17.4 5 16.5 6 16.3 7 15.5 8 16.9 9

Ventricles:

R1 2 3 4 5 6 7 8 9
in the course of an adrenalin test 13 minutes after a dose of 0.5 c.c. The fact that the auricular rate (85.8) is greater than the ventricular (72.2) indicates that primary block must be present and this is indeed obvious for P9 is dropped after a succession of lengthening PR intervals commencing at P4. P1, P2 and P3 are not conducted. The record might therefore be interpreted as a change from complete dissociation to partial block.

Consider next FIG. 19 from the same patient (Case 12) recorded 17 minutes after an injection of 1.0 c.c. adrenalin. Up to the eighth auricular cycle (P8) the sinus rate quickens, the inter-auricular intervals decreasing. In spite of the progressive quickening of the auricles, the ventricles remain remarkably constant in rate until after R6 and beat at a faster rate than the auricles in the early stages of the record. For instance the time elapsing between R1 and R5 is 3.512 sec. equal to a rate of 68.3 beats per minute, whereas the time elapsing between P1 and P5 is 3.688 seconds representing an auricular rate of 65.1. The auricles and ventricles are therefore momentarily dissociated, the ventricles beating faster than the auricles.
auricles. After P5 and R5 the auricles beat more quickly than the ventricles, (indicating that primary block is present) and the record assumes the form of that demonstrated in the previous FIGURE (No. 11284a). The PR intervals lengthen from 0.208 to .520. In partial heart-block of the form associated with simple prolongation of the PR interval there is no dissociation.

The electrocardiogram just considered may therefore be taken to represent a functional dissociation for the first five (or possibly six) auricular and ventricular beats. Thereafter the auricular impulses regain control of the ventricle. The condition may therefore be described as "dissociation with interference". The auricle "interferes" with the ventricular rhythm, and in doing so partial block is produced. In other words a functional dissociation consorts from time to time with a primary block. It may be remarked that "interference" seems a cumbersome term to use, and if a more apt expression for this phenomenon seems justifiable then I suggest that the phrase "auricular capture" more nearly describes the mechanism and better fits the facts.

Dissociation with "interference" was observed/
observed to occur only after adrenalin. The effect was constantly reproduced by doses of 0.5 c.c., 0.75 c.c. and 1.0 c.c. No other procedure had the same effect. This in itself suggests that adrenalin may be a valuable means of distinguishing between certain forms of complete dissociation. The case is recorded as a very exceptional instance of dissociation.

III. 'THE STOKES-ADAMS SYNDROME.'

In this section it is proposed to record electrocardiograms depicting the mechanism of the heart during the Stokes-Adams seizures. Before considering the facts to be presented it is right to point out that in the literature there is no adequate or comprehensive definition of what is implied by either the term "Stokes-Adams syndrome" or "Stokes-Adams seizure". Neither Adams nor Stokes defined a syndrome nor did they offer an explanation for their clinical observations. The original attempt to marshal the relevant facts and define a clinical entity was made by HUCHARD (117). In describing a group of patients thought to be suffering from bulbar arterio-sclerosis Huchard (fifty years after the/
the appearance of the work of Stokes and Adams,

wrote:

"Dans cette forme, les battements du cœur sont très lents, les pulsations radiales peuvent s'abaisser jusqu'à trente, vingt ou même cinquante par minute et s'accompagner souvent d'attaques syncopales et épileptiformes triade syndromique signalée par Adams dès 1827 ensuite par Stokes d'où le nom de maladie d'Adams ou de Stokes-Adams que je propose de lui donner".

The earlier clinical observations of Morgagni and of Spens were presumably unknown to Huchard, and the peculiar sequence of events were thus for the first time described as the Stokes-Adams syndrome. With the lapse of years an unfortunate tendency to include other clinical events under the same title has gradually become manifest, until today certain authors (54) go so far as to state that any sudden failure in the maintenance of an adequate cerebral circulation of such degree as to produce apoplecticform or epileptiform seizures, (as a result of a lessened cardiac output) warrants the title of a Stokes-Adams attack. In my opinion this is altogether wrong for with such a wide definition any simple/
simple syncopal attack, or the epileptiform seizures associated with certain forms of paroxysmal tachycardia might well be included under the title proposed by Huchard. It would seem better to confine the term "Stokes-Adams seizure" (or syndrome) to the syncopal attacks associated with a defect in the conducting mechanism of the heart.

In the Case Reports (page 350) there are included several descriptions of the clinical phenomena witnessed during undoubted Stokes-Adams attacks and in certain instances continuous film electrocardiograms were recorded during the attacks. Stokes-Adams attacks were witnessed in Cases 7, 9, 10, and 14. Electrocardiograms throughout the period of the attack were secured in Case 7 (PLATE 10), Case 10 (PLATE 11), and Case 14 (PLATE 5). One other patient who did not suffer from complete heart-block in the intervals between attacks, has also been under observation and an electrocardiogram of a typical seizure is also reproduced in PLATE.

WENCKEBACH & WINTERBERG (222) in reviewing this subject, point out that for the elucidation of the nature of the Stokes-Adams attack in the presence of complete block, sufficient observations/
observations have not been accumulated to allow of the discovery of a common cause for the attacks. On the contrary, (as the electrocardiograms here reproduced clearly demonstrate) the mechanism of the heart beat varies before, during and after the attack in different subjects, and it is natural to suppose that the immediate provoking cause may also differ. For the same reason rational treatment of the seizure must depend on a more perfect knowledge of the cardiac mechanism before and during the period of asystole, and also on a better understanding of those natural influences which tend to produce a spontaneous re-awakening of the dormant rhythm during the phase of ventricular arrest.

It is not proposed to discuss this subject in any detail at the present time, and it may be of sufficient interest to describe briefly the electrocardiograms which have been secured.

1. IN THE PRESENCE OF PARTIAL BLOCK.

PLATE 12 shows a continuous film record of a Stokes-Adams attack occurring in the course of partial heart-block. In this patient digitalis administration was responsible for several seizures.
Strip 1 shows the mechanism of the heart beat immediately before the onset of ventricular asystole. Normal rhythm is depicted with a PR interval of 0.545 seconds gradually increasing to (?) 0.835 before conduction fails. In strip 2 (continuous with strip 1) there is a period of complete ventricular arrest of 9.4 seconds, during which time the auricles rapidly increase in rate. In strip 3, a spontaneous "bundle-branch" rhythm at a rate of 20.8 provides the means of recovery. In strip 4 the change in the form of the ventricular complex indicates that a higher rhythmic centre has displaced the slow beat and ultimately in strip 6 normal rhythm with prolonged conduction time is restored.

A further example of a mild Stokes-Adams attack in the course of partial block has already been described (Case 14) in connection with the therapeutic use of ephedrine. PLATE 5. shows 2 to 1 block before the attack and 3 to 1 during the recovery period.
2. IN THE PRESENCE OF COMPLETE HEART-BLOCK.

Case 7 was an example of intermittent complete heart-block. One of many Stokes-Adams seizures is recorded in PLATE 10. In strip 1 four ventricular beats at a rate of 28.65 are recorded immediately preceding the standstill. The auricular rate over the same period is 129 per minute. Ventricular arrest commences in Strip 1, continues throughout Strip 2 and the first ventricular beat, (the first of the returning beats) is recorded towards the end of Strip 3. The ventricular beat is absent for 50.4 seconds, and the irregular movements of the fibre in the middle of strip 2 and beginning of strip 3 are due to the epileptiform movements. Strip 4 was taken 1 minute after the end of strip 3 and a minute also elapses between it and the next record. These show that after a succession of various abnormal complexes the original rhythmic focus ultimately regains control at a rate of 25.5 beats per minute (strip 5).

The Stokes-Adams attack recorded in Case 10, of which a full account is given in the Case-Report (page 421) forms a remarkable record of ventricular/
ventricular fibrillation with recovery. The mechanism of the heart before during and after the seizure is shown in PLATE 11. The uppermost strip was photographed a few minutes before an intravenous injection of atropine. Attention is directed to the coupled rhythm and the group of six ventricular extra-systoles in rapid succession towards the end of the record. These extra-systoles constitute a short run of paroxysmal ventricular tachycardia at a rate of 157.9 per minute. The paroxysm ends with bidirectional deflections - the rarest form associated with this arrhythmia. Immediately after the injection, a further bout of paroxysmal tachycardia was observed but not recorded. The patient then fainted. The continuous record (strip 1, 2, 3, 4 and 5) demonstrates that the seizure was associated with ventricular fibrillation. At the extreme end of strip 5 complete ventricular arrest is recorded. The records demonstrate that the period of ventricular fibrillation lasted for 101.6 seconds. By this time the patient appeared to be dead and the electrocardiograph string remained absolutely motionless. The lowermost record was photographed less than a minute after an intra-cardiac injection of 0.5 c.c. adrenalin solution. After one or two
extra-systoles the rhythm becomes regular at a rate of 67.9.

Although adrenalin (intravenously) is credited with the power of inducing ventricular tachycardia and fibrillation, yet in this patient, in spite of an attack of ventricular fibrillation immediately previous to the injection of the drug, its intra-cardiac administration apparently did good. Davis & Sprague (50) have recently reviewed the literature concerning ventricular fibrillation in man. Less than a dozen cases are known in which the patient survived the attack. That it is occasionally the dominant cardiac mechanism during the Stokes-Adams seizure seems established from the clinical reports of Kerr & Bender (125) de Boer (16) Gallavardin & Berard (79) von Hoesslin (112) Levine & Matton (135) and Schwartz (194 & 195). The latter author emphasises the remarkable frequency of seizures in those in whom ventricular fibrillation is the underlying mechanism of the attack. The patient reported here (Case 10) suffered from as many as 114 seizures in a period of 24 hours, but whether ventricular fibrillation was associated with each and all of these attacks it is impossible to say. 

SUMMARY/
SUMMARY.

Electrocardiograms have revealed four distinct disorders of cardiac mechanism over the various phases of the Stokes-Adams syndrome. Two examples of ventricular arrest occurring during partial heart-block are recorded, and two during complete heart-block. In one of these paroxysmal ventricular tachycardia followed by ventricular fibrillation preceded the onset of asystole. This attack was treated by the intra-cardiac injection of 0.5 c.c. adrenalin. The patient recovered.
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CASE RECORDS.
CASE 1.

Mrs. Th. Age 55 years, (1928).

Occupation, washer and cleaner.

Admitted to Royal Infirmary September 14th. 1928, and discharged on 30th. November, 1928. (This patient was originally under the care of Dr. Alexander Goodall. She was transferred to Prof. Murray Lyon's ward where I had the opportunity of carrying out certain investigations).

COMPLAINT.

Attacks of loss of consciousness intermittently for five years.

Flatulence, and breathlessness on exertion, both of approximately five years duration.

HISTORY of ILLNESS.

Until five years ago the patient was in good health and accustomed to go to her work, which was of a strenuous nature, regularly each day. About this time she began to suffer from various vague symptoms, consisting of occasional frontal headaches, slight shortness of breath on exertion and a sense of fatigue. She attributed these symptoms to the onset of the menopause and as they did not cause her much distress, she was able to continue her work as a cleaner.

After remaining in somewhat indifferent health for about two months, she had in 1923 a fainting attack without warning. She has forgotten the date of this occurrence but remembers quite clearly certain events connected with the attack. She recollects that, one morning, after breakfast, when sitting comfortably by the fire she suddenly and quite unexpectedly lost consciousness. She recovered in a few minutes to find herself lying on the floor in front of the fire. She can remember the events immediately before the attack quite distinctly. No one saw her faint, but some neighbours, who called at the house after she/
CASE 1.

She had regained consciousness, noticed that she was very pale, her lips blue and her hands very cold. After the attack she was in bed for a few days and during that time felt very weak and helpless. While she was confined to bed she had no further attacks of loss of consciousness but on several occasions she complained of feeling lightheaded accompanied by a sinking sensation. She also was giddy and sometimes suffered from temporary dimness of vision. She says that these feelings suggested to her that another fainting attack was imminent. These attacks were quite transient and did not last longer than half-a-minute at a time but were often frequently repeated at short intervals. Her husband noticed that, when she was suffering from these sensations, her face became pale and her lips blue. Twitching of the hands, arms or legs was not observed. In a few days she was able to be about the house again and then gradually resumed her work.

Since the first fainting attack her health has not been so good as formerly. Breathlessness has been more easily provoked, and her work rather too much for her. She noticed that fatigue readily set in after attempting household duties which she had been able to accomplish in comparative ease only a few months previously.

During the past few years she has had many attacks similar to the first one coming on unexpectedly and without warning. The precise number is not known to her - but they have occurred at irregular intervals, a month or two elapsing between successive attacks. She has fallen unconscious in the street several times and has been brought to the hospital in the police ambulance. She fainted once when she had a kettle of boiling water in her hand and now (1926) has the scar of a healed scald over the left side of her chest. Some months ago she had an attack in the King's Park and sustained a severe bruise on the left temple.

During the past two years she has attended the New Town Dispensary on account of flatulence.
CASE 1.

flatulence. She believes that when "wind gathers in the stomach" and when her abdomen is swollen, the likelihood of an attack is increased. Abdominal distension causes her considerable discomfort.

Within the past year she has lost weight - a stone or more. In spite of breathlessness she is able to do a fair amount of work in the house though she has been forced to give up her outdoor employment. The headaches are now less troublesome than formerly and at the time of her admission to hospital her main complaints were the dread of further fainting attacks and the discomfort due to flatulence.

PREVIOUS HISTORY.

Pneumonia and pleurisy at the age of 28. She was confined to bed at this time for nearly three months.

A severe attack of tonsillitis at the age of 49. This illness preceded by a few months the onset of her present complaint.

"Colds" have troubled her from time to time all her life.

She has not suffered from rheumatic fever, influenza, scarlet fever, diphtheria, typhoid or any of the manifestations of rheumatism. She has no recollection of any illnesses beyond those stated above.

FAMILY HISTORY.

She has had six children and no miscarriages. Her only daughter died of rheumatic heart disease with acute pericarditis in 1926 at the age of 22. She has five sons alive and well.

STATE on EXAMINATION.

The patient lies comfortably in bed. Her face has a healthy colour. There is no pallor and no cyanosis of the lips, ears or cheeks. Her face is rather expressionless. Her intelligence is average but she rambles in her conversation/
conversation and it is exceptional to receive a coherent answer to the usual questions. She lies very still in bed and rarely moves or looks about her. She is in no distress and complains of no pain.

CIRCULATORY SYSTEM.

The Pulse is slow in rate being 21 beats per minute. It is regular in time and force. The up-stroke is strong and is followed by a gradual decline. The systolic and pulse pressure are both raised. The radial artery is a little thickened but hardly more than might be expected in one of her age. It can just be palpated and rolled by the finger between successive pulse beats. There is no tortuosity and there are no calcareous plaques to be felt. The blood pressure is 165 systolic and 56 diastolic. There is no capillary pulsation in the bed of the finger nail nor in the vessels of the lips. There is no clubbing of the fingers. The hands are cold but there is no cyanosis. The superficial veins of the arms are prominent and a little distended.

THE CHEST AND NECK.

The chest wall is well covered. There is no bony deformity and no indrawing of the intercostal spaces. The superficial veins in the neck are full. Regular pulsations in the neck over the jugular vein, particularly in the right side, are easily detected. These are at a rate of approximately 74 per minute and are interrupted from time to time by a more violent and prolonged pulsation at a rate of 22 per minute. The thyroid gland is not enlarged.

There are no abnormal pulsations seen over the praecordium and this part of the chest has the usual contour. Pulsation slow in rate is seen in the epigastrium. There is no pulsation in the interspaces to the right of the sternum. The apex beat is not visible.

THE HEART.

The apex beat is difficult to locate. It can just be felt in the 5th interspace. It is/
CASE 1.

is diffuse and the area of pulsation appears to be about the size of half-a-crown but cannot be determined with accuracy. No thrill is felt at the apex or at the base. There is no diastolic shock.

On percussion the area of cardiac dullness is found to be -

<table>
<thead>
<tr>
<th>Cm (right)</th>
<th>Interspace</th>
<th>Cm (1 ft)</th>
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<tbody>
<tr>
<td>2.5</td>
<td>2</td>
<td>5</td>
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<tr>
<td>3.5</td>
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<td>9.5</td>
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<tr>
<td>5</td>
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</tbody>
</table>

The left mid-clavicular line is 9.5 cm. from the mid-sternal line. The heart is therefore enlarged.

On auscultation, the first mitral sound is completely replaced by a harsh systolic murmur which is maximum in intensity immediately inside and above the apex beat. It can be heard less clearly all over the praecordium, and also in the axilla. From time to time the systolic murmur is almost entirely replaced by a thudding and accentuated first sound. The second mitral and aortic sounds are pure in character. The second pulmonary sound is not accentuated. Careful and repeated auscultation failed to detect any evidence of auricular contractions.

ALIMENTARY SYSTEM.

The patient complains of flatulence and swelling of the upper part of the abdomen; she has occasionally some discomfort, amounting to pain at times spreading over the epigastrium. This generally occurs an hour or more after food has been taken. She is accustomed to take aperients once or twice a week. The bowels are kept regular in action by this means. She has had no vomiting and does not suffer from diarrhoea. She eats all kinds of food. The abdominal discomfort/
CASE 1.

Discomfort is not related to any particular article of diet.

The abdomen is well covered. It moves freely with each respiration. There is no tenderness detected. There is no distension. The liver and the spleen are not enlarged. The kidneys are not felt on deep palpation. There is no ascites.

RESPIRATORY SYSTEM.

She has suffered from breathlessness on exertion. She has no cough and has no expectoration.

Expansion is good and equal on both sides. The chest is of normal shape and there is no flattening of the interspaces or wasting of the muscles. A healed scar is present on the left side involving the left anterior axillary fold.

The percussion note is resonant throughout. Auscultation reveals vesicular breathing. There are no accompaniments.

NERVES and LOCOMOTORY SYSTEMS.

She has suffered from headaches in the past and fainting attacks more recently. She sleeps well. Her memory is fairly good. Intelligence is average, but she has apparently some difficulty in concentrating.

CRANIAL NERVES.

No weakness or paralysis detected. The eye movements are perfect in all directions. The pupils are equal, of medium size and each reacts to light and accommodation. The fundus of each eye was healthy. The tendon reflexes are easily elicited. The biceps are present and equal in each side. The knee jerks are equal and brisk. Ankle jerks are present. Babinski's test produces a flexor response on each side. There is no disorder of sensation. The muscular power of the extremities is normal. There is no wasting of the muscles. Romberg's sign is negative/
CASE 1.

There is no oedema of the extremities.

THE URINE.

Repeated examinations revealed no abnormal constituents. The daily output averaged 900-1000 c.c.s.

BACTERIOLOGICAL REPORT.

The blood Wassermann reaction before and after a provocative dose of "914" is negative on each occasion.

COURSE in HOSPITAL.

She complained from time to time of neuralgic headaches. This was accompanied by a visual sensation, described as a "mist over the eyes". She also suffered occasionally from flatulence and feeling of faintness. She had no Stokes-Adams attacks while under observation and had none since during the four years which she has been under my observation.

CLINICAL DIAGNOSIS.

- Etiological: Unknown "toxic".
- Structural: Cardiac hypertrophy, Mitral insufficiency.
- Rhythm: Complete heart-block.
- Functional Efficiency: Grade 2a.

SUMMARY of CASE 1. (Mrs. T.)

CASE 2.


Occupation, retired station master.

Admitted to Royal Infirmary April 10th, 1929. Died October 20th, 1929. Post-Mortem Examination 21st. October 1929. (This patient was under the care of Dr. Edwin Matthew to whom I am indebted for the opportunity of investigating the case).

COMPLAINT.

Loss of speech of 2 day's duration.

General weakness 1 year.

HISTORY of ILLNESS.

For the past year or more the patient has noticed that his health has been gradually failing. In particular he has been more easily made tired than formerly, has become slower in his actions and has not had the same desire to go about as in the past. He remained comparatively well until the Summer of 1928 when, on dismounting from a tram car, he suddenly became giddy, "turned faint". He did not fall to the ground but felt unsteady, lurched and thinks that he lost consciousness for only an instant. He described his sensations as being "as though something had flashed across my brain". He did not vomit and did not have a fit. With the help of his wife he was able to walk slowly home, a distance of three hundred yards or more. The same night he suffered from headache and much general weakness but felt fully recovered the next morning. Following this experience he was less able to go about and became gradually more and more confined to his house.

In January 1929 the sense of fatigue and general weakness began to increase. In the evening his feet and ankles were swollen and he was breathless on slight exertion. The swelling of his legs gradually increased and his doctor prescribed treatment with satisfactory results. The swelling gradually subsided and the dyspnoea became/
CASE 2.

became progressively less so that he was again able to move about out of doors.

Within the last month he has become rather weaker. His home is a flat reached by the ascent of three flights of stairs. This is a greater climb than he can manage with comfort. Dyspnoea checks his activity and he is forced to rest many times on each flight.

Two days before his admission to hospital, he was walking slowly across the Meadows, when he was suddenly seized with a violent headache chiefly in the frontal region. His wife noticed that his face and lips were blue in colour. The headache was so severe that for a few minutes he stood still in great agony. He was not giddy and did not stagger or fall. In a moment the pain began to abate and he was able to make his way home but in considerable distress. By the time he reached his house it was noticed that, in endeavouring to speak, he merely mumbled meaningless syllables. He did not lose the power in his arms or legs, could recognise his friends and swallow his food. He understood what was said to him, could write down his requests, read his newspaper but was unable to express himself in words. The following day his condition had deteriorated and by the time he was admitted to the Royal Infirmary (on the third day of his present illness) he was very confused and seemed not to understand all that was going on around him.

PREVIOUS ILLNESSES.

No diphtheria. No rheumatic fever. He only admits to have suffered from the usual illnesses of childhood.

STATE on EXAMINATION.

He lies very quietly in bed, is drowsy and confused. He is only able to utter the one word "Yes" which is the response to every question put to him. He does not appreciate fully where he is and has apparently no idea of time. Common objects, such as keys, money, pencils and cigarettes, are not recognised.
There is no evidence of any paresis, he moves his head, face, eyes, tongue, hands and arms and legs slowly and deliberately.

CIRCULATORY SYSTEM.

Pulse is regular in time and force. The rate is 36 per minute. The upstroke is strong, slow, well sustained and the wave of good volume. The wall of the radial artery is thickened and the vessel tortuous. The Blood Pressure is 180 systolic and 98 diastolic.

The thyroid gland is not enlarged. The veins in the neck are a little distended. Over the jugular bulb rapid regular pulsations are seen three to four times as fast as the carotid beat.

Heart. The praecardium presents no abnormalities. The apex beat is in the 5th interspace, 6.5 cm. to the left and within the midclavicular line. The apical thrust is powerful and localised to a small area. There is no thrill and no diastolic shock at apex or base. Percussion confirms that the area of cardiac dullness is not increased to right or left of the midsternal line. Auscultation reveals that the first mitral sound is completely replaced by a loud blowing systolic murmur maximum at the apex but heard all over the chest. From time to time the murmur is replaced by a dull thudding sound. The second mitral sound is pure. In the aortic area the first sound is accompanied by a soft systolic murmur. The second sound in the aortic area is accentuated and occasionally reduplicated. The pulmonary sounds present no striking change. Both are heard clearly. There is a faint systolic murmur with the first sound. In the tricuspid area a soft systolic murmur is heard. Sounds which might be attributed to auricular contractions, are not heard over the praecordium.

RESPIRATORY SYSTEM.

No subjective phenomena. Breathing is regular and not laboured. No cyanosis detected.

Thorax/
THORAX.

The chest is poorly covered. The muscles are poorly developed. The thorax is symmetrical in form and expansion is equal on the two sides. Vocal fremitus is unaltered. The percussion note is resonant over both lungs. The breath sounds are of the usual intensity over the whole of both lungs. In the mid-clavicular region on the right side the breath sounds have a broncho-vesicular quality. Rhonchi can be heard over the chest, a few in number and scattered here and there. No moist sounds are detected.

ALIMENTARY SYSTEM.

He has suffered from constipation from time to time. On examination nothing abnormal was detected. There was no tenderness detected. The liver was of normal size, the lower pole of the spleen was not felt and there was no ascites.

GENITO-URINARY SYSTEM.

He was incontinent for two nights following the onset of the severe attack of headache and aphasia. Otherwise he has not complained of any urinary symptoms.

The output averages 900 c.cm. per day. The specific gravity is 1015 and the reaction is acid. It contains a faint trace of albumin and microscopic examination revealed the presence of a few granular casts.

NERVOUS SYSTEM.

Subjective phenomena have been intense headaches and aphasia. He is right handed. His memory has always been good until within the last few months.

The Cranial Nerves. No evidence of weakness or paralysis is detected. The eye movements are perfect in all directions. The pupils are equal and reacted to light and accommodation. The fundus of each eye is healthy except for the presence of some sclerosis of the retinal arteries. The discs are of normal appearance.
CASE 2.

The tendon reflexes are brisk and equal on each side of the body. Babinski's test gives a negative reaction in each foot. There is no loss of muscle power. There is no disorder of sensation.

LOCOMOTOR SYSTEM.

The extremities present no oedema. The superficial veins of the arms are a little distended and more prominent than usual. There is free and unrestricted movement at the joints of the extremities.

X-RAY REPORT. (24-4-29)

The heart did not appear to be enlarged but the lateral border of the left auricle was a little more prominent than in the usual antero-posterior view.

BACTERIOLOGICAL REPORT. (20-4-29)

The blood Wassermann reaction is negative.

BIOCHEMICAL REPORT. (10-7-29).

The non-protein nitrogen of the blood is 41 mgms%, the urea N2 is 23 mgms%, and the creatinine 3 mgms%.

COURSE in HOSPITAL.

By the 14th April his condition had improved considerably. He was clearer in his mind, could recognise objects and was able to articulate well. By 20th April he was able to get up from bed. His improvement was only temporary for, after sitting up daily in a chair for an hour or two, oedema of the feet and ankles was noted and he became a little dyspnœic on the slightest exertion. He was accordingly kept in bed where he remained until his death on 20th October, 1929. The oedema gradually increased though he had little respiratory difficulty until near the fatal termination. On the 31st May his speech again became indistinct, headache and hallucination/
hallucination of sight. The following day his temperature rose to 101.4°F. and his pulse increased in rate from 36 to 48 per minute. After the febrile attack, which was probably related to further cerebral infarction, he had an increase in the quantity of albumen in the urine and was more confused mentally than before. By September his general condition had improved somewhat though oedema persisted in spite of active treatment with digitalis and diuretics. Towards the end of September he developed occasional periods of Cheyne-Stokes breathing and occasionally had slight twitching movements of the facial muscles. He was frequently very confused at night. During his final weeks the pulse ran faster than formerly often in the neighbourhood of 40 or 50 per minute and at various times he had attacks of paroxysmal ventricular tachycardia. Some of these were related to the administration of digitalis. The congestion of the tissues gradually increased and he died peacefully on 20th October, 1929 in a state of advanced heart failure.

THE CLINICAL DIAGNOSIS WAS

<table>
<thead>
<tr>
<th>Etiological</th>
<th>Vascular Degeneration</th>
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<tbody>
<tr>
<td></td>
<td>Chronic nephritis.</td>
</tr>
<tr>
<td>Structural</td>
<td>Cerebral Thrombosis</td>
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<td>Generalised Arteriosclerosis.</td>
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<td>Chronic Interstitial myocarditis.</td>
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<td>Mitral insufficiency.</td>
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<tr>
<td>Rhythm</td>
<td>Complete heart-block.</td>
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<tr>
<td></td>
<td>Paroxysmal ventricular tachycardia.</td>
</tr>
<tr>
<td>Functional</td>
<td>Grade 3 (with uraemia).</td>
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</tbody>
</table>

SUMMARY of CASE 2.

A male aged 71. History of increasing fatigue and gradually failing health for at least a year. He had a mild attack of congestive/
congestive heart failure in January 1929 from which he recovered. A cerebral thrombosis followed a few months later which rendered him aphasic for some days. From the immediate effects of this he again recovered but developed a further attack of congestive heart failure which gradually became progressively worse. The heart-block was only known to have been present for six months. The heart rate was in the neighbourhood of 36 per minute. The blood pressure was 180/98. Later the rate varied considerably being often about 40 per minute. It rose during a period of pyrexia and fell with the subsidence of the temperature. He had several attacks of paroxysmal tachycardia. Towards the end he exhibited signs suggestive of uraemia and ultimately died in advanced congestive heart failure six and a half months after the first attack of aphasia. The Wassermann reaction was negative. There were no Stokes Adams seizures in this case. Post-mortem revealed generalised vascular disease. There was a calcified area in the outer-ventricular septum.

POST-MORTEM EXAMINATION. (21-10-29)

(Only the essential pathological features are described here).

All the tissues were congested, free fluid being present in all the serous sacs.

THE HEART.

The Heart was slightly enlarged. The pericardial surface was smooth and glistening and there was a reduction in the average amount of subepicardial fat. That which remained was oedematous in character.

The ventricles and auricles were slightly dilated. There was no evidence of any ante-mortem clot in any of the cavities. The myocardium of the left ventricle was atrophic in character due to the dilatation of the chamber. It was moderately firm in consistency and pale red in colour. No naked-eye evidence of any thrombosis could be seen. In the interventricular septum, immediately lateral to the posterior aortic cusp, there was a small patch of calcification.
CASE 2.

it extended right through the septum. Otherwise there was nothing to note in the heart. The pulmonary and tricuspid valves were healthy. The aortic cusps showed definite evidence of patchy fibrotic thickening. The mitral valve admitted two fingers. Both cusps were thickened.

THE VESSELS.

The Coronary vessels were markedly atheromatous. In the aorta there was fairly extensive atheroma. The cerebral vessels showed very advanced atheromatous change throughout. The renal vessels had also undergone atheromatous thickening.

THE LUNGS AND ABDOMINAL ORGANS were congested.

THE KIDNEYS.

These organs were definitely reduced in size. The capsules were thickened and, on stripping, left a very pale, almost smooth surface but on closer examination a commencing fine granularity could be seen. The cortex in both was much reduced in size, exceptionally pale in colour and somewhat structureless in appearance, although here and there could be seen little areas of congested vessels. The appearances were of the type associated with the so-called "small white kidney".

CRANIUM AND CONTENTS.

The brain convolutions were somewhat flattened. On section an area of softening about 2 sq. cm. in size was present in the right hemisphere immediately anterior to the right occipital lobe. It involved the cortex and a small part of the white matter. This area had been present for some time, as most of the cavity was filled with fluid-like material which gradually merged into disintegrating white matter. In the left ventricular nucleus there was a small area which was somewhat pigmented and suggested either a previous softening or a small haemorrhage. There was nothing abnormal to note in the cerebellum.

SUMMARY/
CASE 2.

SUMMARY OF POST-MORTEM FINDINGS.

Chronic parenchymatous nephritis.

Myocardial degeneration.

A calcified area in the interventricular septum.

Generalised arterio-sclerosis, affecting particularly the cerebral arteries and the coronary arteries.

Cerebral softening. Chronic venous congestion.
CASE 3.

David C: Age 64 (1926) Male. Married.

Occupation, a painter.

Was first admitted to the Royal Infirmary on 7th. December, 1926 and discharged on 2nd. February, 1927. He was again a patient from 16th. April, 1929 until 6th. May, 1929. A third period of hospital treatment extended from 12th. April until 29th. April, 1932. (This patient was under the care of Professor W.T. Ritchie who has kindly placed his clinical notes of the cases at my disposal and who has given me the opportunity of making certain investigations).

COMPLAINT. (At date of first admission to Hospital)

Weakness and pains in the knees)
Breathlessness ) 6 weeks.

The patient states that he has for many years been subject to rheumatic pains, particularly in the knees. Six weeks previous to admission to hospital, he was seized with pains in the legs and knees. Fearing that an attack of rheumatism was imminent, he consulted his doctor, who discovered that the heart's action was slow - in the neighbourhood of 36 or 40 beats per minute.

For six weeks he has not felt fit for his work on account of increasing fatigue and failing health. During this time he has noticed that exertion was accompanied by breathlessness. He has occasionally had to give up his work in past years for a short time on account of similar symptoms. Discomfort in the epigastrium and tightness in the chest also troubled him. This has been more noticeable during the past few weeks.

PREVIOUS ILLNESSES.

39 years ago at the age of 25 he had a lump in his right leg. This was lanced by his doctor, who told him that he had syphilis and advised him to seek hospital treatment. He did not accept this advice and has in consequence had no treatment./
CASE 3.

34 years ago he had an attack of subacute rheumatism. He was confined to bed for several weeks. His doctor warned him of the dangers and recommended him to have a long rest. He was unable to take this advice and shortly afterwards returned to his work.

12 years ago he met with an accident. He was engaged in carrying a long ladder in a high wind. It fell and struck his chest a heavy blow. Since this time he believes that he has always been subject to attacks of palpitation and occasional tightness in the chest.

3-4 years ago, when hurrying to his work he had a slight syncopal attack. He felt dizzy, fainted and recovered immediately.

For a number of years he has been subject to attacks of rheumatism - of a subacute nature - in which his knees have been swollen and painful.

STATE ON EXAMINATION.

He is sturdily built and of average development. He lies comfortably in bed in no distress. His colour is good though the face exhibits a slight degree of pallor. There is no cyanosis and no respiratory embarrassment.

CIRCULATORY SYSTEM.

The Pulse. The radial artery is thickened and easily palpated, but not tortuous. The pulse is slow - 40 per minute - and perfectly regular in time and force. The pulse is of the type described by Corrigan - a rapid and abrupt upstroke, only maintained for an instant, and falling away rapidly. It is easily recognised even with his hand lying on the bed clothes and is greatly exaggerated by holding the arm in a vertical position. Capillary pulsation is seen beneath the nail and also in the vessels of the mucous membrane of the lips. There is no clubbing of the/
CASE 3.

the fingers. The Blood Pressure is 240 systolic and 60 diastolic.

CHEST AND NECK.

The chest is well covered and symmetrical in shape. In the neck the pulsation in the carotid and subclavicular arteries is strikingly exhibited. Pulsation is also visible in the suprasternal notch, particularly to the right side and immediately medial to the sternal head of the right sterno-mastoid muscle. Over the jugular bulb rapid regular pulsation at a rate of approximately 80 per minute can be detected. These waves are weak in force and cannot be felt by the finger. The carotid pulsations are slow regular and powerful at a rate of 40 per minute. The veins in the neck are a trifle more prominent than usual. The thyroid gland is not enlarged. The neck is deeply set, short and relatively thick. The praecordium is not deformed.

THE HEART.

The apical pulsation is visible in the 6th. interspace, 5½ inches from the mid-sternal line. Pulsation is visible, in the region of the apex, over an area of approximately 2 inches in diameter. On palpation the beat is found to be a strong, prolonged and forcible thrust occurring regularly. The outermost and lowest point is 5½ inches from the midline. Percussion confirms this point. The right border of the heart could not be outlined beyond the right border of the sternum and no enlargement of the area of supracardiac dullness is detected.

In the mitral area the heart sounds are completely replaced by systolic and diastolic murmurs. The systolic element is conducted into the axilla. The diastolic murmur can be faintly heard at the apex of the axilla. In the Aortic area there is a to-and-fro murmur. The systolic murmur is conducted along the arteries in the neck and as far as the brachial artery. The diastolic murmur, which is harsh and grating in character is well heard over the sternum. It is maximum/
CASE 3.

maximum along the left sternal border and is heard with diminishing intensity at the apex but becomes a little louder in the armpit. The second pulmonary sound is accentuated and accompanied by the diastolic murmur of aortic origin already described. The sounds are of constant character.

RESPIRATORY SYSTEM.

Beyond breathlessness there are no subjective symptoms. The chest is well covered and rather barrel-shaped. Expansion is equal on the two sides but limited, there being less than 1 inch of difference between the size of the circumference in full inspiration and expiration. Vocal fremitus is weak. The percussion note is hyper-resonant. The breath sounds are vesicular with no accompaniments and the vocal resonance is of average quality throughout.

ALIMENTARY SYSTEM.

There are no subjective symptoms. The mouth and tongue are clean and healthy. The tonsils are not enlarged. The abdomen is well covered and moves freely with respiration. Regular pulsations are seen in the epigastrium. There is no tenderness detected on deep palpation. The lower edge of the liver can be palpated one finger's breadth between the costal margin. The lower pole of the spleen is not felt. There is no ascites.

UROGENITAL SYSTEM.

There are no subjective phenomena. The urine output averages 1000 to 1200 c.c.s. It contains a small quantity of albumen but no other abnormal constituents. The specific gravity averages 1018 and the reaction is acid.

NERVOUS AND LOCOMOTOR SYSTEMS.

He has had some pains in the legs. There is no oedema of the legs, feet or sacorun. The joint movements are free but the muscles of the calves/
CASE 3.

calves are a little tender on firm pressure.  

The pupils are small and fixed and do not react to light and accommodation. They are irregular in outline. The eye movements are free and equal in all directions. The fundus of each eye is healthy. The discs have the usual normal appearance. The retinal arteries do not pulsate.

The tendon reflexes are equal and present on both sides of the body. There is no evidence of an organic nervous lesion beyond the pupillary changes.

BACTERIOLOGICAL REPORT. (12:12:36.)

The blood Wassermann reaction is negative. This finding has been confirmed on several occasions.

X-RAY EXAMINATION.

Reveals a greatly enlarged "boot-shaped" heart. There is no aneurysm formation. The left ventricle is greatly hypertrophied.

COURSE.

Since his first admission to hospital this man has been at fairly frequent intervals. He has remained well except for occasional rheumatic pains. He has had no Stokes-Adams attacks while under observation and the presence of complete heart-block has been confirmed by numerous electrocardiograms since his first admission to hospital.

During his stay in hospital in the month of April 1932 the opportunity was taken to make observations on the response of the heart to various procedures including the administration of certain drugs. He was discharged from Ward 23 on the 29th April in fairly good health and able to go about and exert himself without much discomfort.

CLINICAL DIAGNOSIS./
CASE 3.

CLINICAL DIAGNOSIS.


Structural.  Cardiac hypertrophy  Aortic regurgitation  ? Aortitis

Rhythm  Complete heart-block  Coupled rhythm (intermittently)

Functional  Grade 2a.

SUMMARY OF CASE 3.

A male aged 64 (1926), admitted syphilitic infection in youth. Began to have praecordial pain and palpitation in 1914. Off work on account of general weakness, and fatigue and "rheumatism" on many occasions since then. Increasing dyspnoea noticed in 1926 and then found to have complete heart-block with ventricular rate of 40, aortic regurgitation and cardiac hypertrophy. Blood pressure 240/60. Has remained in fair health, able to go about with some slight distress on exertion, but has been forced to give up his work. One fainting attack in 1923, but no definite Stokes-Adams admitted or observed while under observation.
CASE 4.

William T. Age 65, Male.

Occupation, bootmaker.


(This patient was referred to me as an out-patient by Dr. George Brewster. Through the kindness of Professor Murray Lyon I was able to have the patient under observation in his Ward in the Royal Infirmary).

COMPLAINT.

Attacks of loss of power of the whole body for three years.

HISTORY of ILLNESS.

At the age of 45 (twenty years ago) the patient had a bicycle accident striking his head on the road. He was rendered unconscious for some hours, suffered from concussion and sustained a deep wound on the right frontal and temporal regions of the skull. Within a month after the accident he began to suffer from what he calls epileptic attacks. These attacks occurred at intervals of about one month and with great regularity. By experience he learned to anticipate an attack towards the end of the third week of every calendar month. The attacks occurred singly and generally at night or in the late afternoon. Usually, a peculiar feeling in the epigastrium preceded the seizure by a few minutes and served to warn him that he was about to lose consciousness. His friends have told him that during an attack his arms and legs move violently and then become rigid. During the seizure he foamed at the mouth and on more than one occasion he has bitten his tongue. The violent stage of the fit generally lasted for about fifteen minutes or even longer. Following the attack he sleeps heavily but is generally well enough the next morning to go to work as usual.

For one year he had fits of the type described/
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described above quite regularly. They then became much less frequent and in the succeeding seven years he had in all only three fits. Each of these, according to his statement, was of similar type to those which he experienced after the bicycle accident. They occurred at long intervals, the actual dates being now forgotten.

From the year 1920 until 1928 he had no fits of any kind, felt in perfect health, and was able to carry out his usual employment with regularity.

Early in 1929 (at the age of 62) he began to have attacks of a different nature. During these he feels dazed and has to lie down immediately in order to prevent himself falling. He has himself volunteered the statement that these "turns" are of a distinct type and differ from those which formerly affected him. He has never had any warning of their onset and, so far as he knows, he has not been seized with any convulsive movements during the attack. These seizures, which have generally been of the nature of a sudden momentary faintness or giddiness accompanied by loss of muscular and mental power, have never produced loss of consciousness and he has always been able to save himself from falling. Each attack lasts as a general rule for less than a minute, and occurs at the present time at irregular intervals of three, four or six weeks. Sometimes more than one may occur within the course of an hour or so. His friends have told him that his face turns pale during the attack. After the actual faintness has passed off, which it does quite suddenly, he is generally tired, and drowsy, and may sleep heavily for some hours.

During the week previous to admission to hospital he had two of these "fainting turns". For the same period he has not felt so well in his general health. He has had a little breathlessness on exertion for about a year, though when this commenced/
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commenced, he is not able to state with accuracy. During the past week it has certainly been more troublesome for he has found that dyspnoea makes sleep at night unusually difficult. Propped up in bed his distress is alleviated to some extent, but even in spite of this, insomnia has troubled him greatly during the past few days. He has noticed a little puffiness of the ankles at night during the last week. A slight cough with mucor-purulent sputum has troubled him, more particularly in the mornings, for a similar period of time.

PREVIOUS ILLNESSES.

He has never suffered from rheumatic fever, scarlet fever, or diphtheria. With the exception of the bicycle accident he has had no other injuries of any consequence.

In 1930 a prostatectomy was performed. Since then he has had no urinary trouble of any kind, and he has been greatly relieved by the operation. He has never suffered from any venereal disease.

FAMILY HISTORY.

His father and mother died past middle age of a cause unknown to the patient. There were seven in the family, all being, with the exception of the patient, in excellent health at the present time. He is married and has had one son and one daughter. Both are alive and well. There is no history of epilepsy in his family or amongst his relations. His wife died some years ago, cause of death unknown.

SOCIAL HISTORY.

Since his youth he has always been a keen/
keen athlete, being a long distance runner of some distinction. Until 1929 he was a cycling enthusiast, riding his bicycle every day and often for long distances. The prostatic trouble, which first began to develop about three years ago, forced him to give up this form of exercise, and since the operation he has never felt strong enough to resume this activity. Even up to ten days before his admission to hospital he has made a practice of having as much exercise out of doors as his work permitted. He has always been a non-smoker and only drinks a small amount of whisky or brandy - generally as a restorative after a fainting attack.

STATE on EXAMINATION.

The patient is a well developed muscular man, 5 ft. 11 inches in height and 12 stone 10 lbs. in weight. He looks his age. He lies in bed, propped up by three pillows and is in this position a trifle breathless. There is no cyanosis, and the face has a good healthy colour. He is of rather nervous temperament, has a slight stammer of his speech, and appears somewhat worried about his health.

CIRCULATORY SYSTEM.

The Pulse: The radial pulse beats are regular in time and force. The rate is 25 per minute. The upstroke is slow and powerful, well sustained, and falls away gradually. To the palpating finger the tension is distinctly raised. The vessel wall is thickened but the artery is not tortuous. No calcification of the vessel is detected. The Blood Pressure is 220 systolic and 74 diastolic. Both radial pulses are similar in character. There is no capillary pulsation under the finger nails or in the vessels of the lips. There is no clubbing of the fingers. The hands are/
are cold, but there is no cyanosis. The superficial veins of the arms are prominent and a little distended with the arm resting on the bed in a horizontal position.

The Chest and Neck: The chest is well covered. The musculature is of good development and tone. The chest is not deformed. The neck is full and thick-set. The thyroid gland is not enlarged. The veins in the neck are full and over the jugular bulb small regular waves of uniform excursion are distinctly seen at a rate of approximately 60 per minute. The more powerful carotid beat occurs regularly at a rate of 32 per minute.

The Praecordium: There are no abnormal pulsations seen in the region of the praecordium and this region of the chest has the usual shape and contour found in a well developed muscular man. A slow regular pulsation is seen in the uppermost part of the epigastrium and also to a much less extent in the suprasternal notch.

The Heart: The apex beat can be distinctly seen as a small localised area in the fifth interspace five inches to the left of the mid-sternal line.

On palpation the position of the apical thrust is confirmed and the outermost and lowest point of contact with chest wall is found to be 5\(\frac{1}{4}\) inches from the middle of the sternum. The apical beat is forcible, localised, slow in action, and well sustained. There is no thrill felt either at apex or base. There is no diastolic shock felt.

Percussion reveals that the left border of the heart in the fifth interspace is 5\(\frac{1}{4}\) inches from the mid-sternal line. The right border cannot be percussed. The area of supracardiac dullness/
dullness in the second interspace is not increased in extent.

The mid clavicular line is 4\(\frac{3}{4}\) inches from the midsternal line. The heart is therefore enlarged.

On auscultation the first mitral sound is completely replaced by a loud harsh systolic murmur, maximum just within the apex beat, conducted into the axilla and heard with diminishing intensity as the base of the heart is approached. It can be heard all over the praecordium and accompanies the first aortic and pulmonary sounds. The second mitral sound is pure in character. The pulmonary second is a trifle louder than usual. The second aortic sound is definitely accentuated and has a hollow ringing quality.

Quite frequently the systolic murmur heard at the apex is replaced for a single cardiac cycle by a booming first sound. On other occasions, when the murmur is temporarily absent, the first mitral sound is reduplicated. This phenomenon may be attributed to the coincident contraction of auricles and ventricles.

Listening over the middle sternum or at its left lateral border about the region of the third interspace a short dull distant sound may be heard to occur regularly at a rate of approximately 54 per minute. These sounds are faint but sufficiently clear to count with accuracy and are synchronous with the small waves seen in the veins in the neck. They have been heard on many occasions by different observers. The best position for hearing these sounds is obtained by having the patient sit up in bed and lean forwards from the hips. They are also heard less distinctly when he lies flat on his back in bed but not with such regularity as when he adopts the former position.

ALIMENTARY SYSTEM/
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ALIMENTARY SYSTEM.

On enquiry it is found that within the last few months he has had some discomfort after food. This amounts to a sense of fullness after meals, occasional eructations and some slight swelling of the upper part of the abdomen. He says that he suffers from occasional griping pains in the lower part of the abdomen. He is accustomed to take aperients once or twice a week, and doing so the bowels act regularly each day. His appetite is good and he enjoys his food.

The mouth is healthy. The tongue is clean and moist. Many teeth are absent. One or two of the molars are carious. There is no pyr-rhoea but some slight retraction of the gum margin. The tonsils are small and healthy.

The abdomen is well clad and a little pendulous. It moves freely with each respiration and there is no tenderness or rigidity detected. The lower edge of the liver can be palpated half-an-inch below the costal margin in the right nipple line. The spleen is not enlarged. The lower pole of either kidney is not felt on deep palpation. There is no evidence of ascites.

RESPIRATORY SYSTEM.

He has a slight morning cough with mucopurulent sputum. This has only been present for a week or thereabouts and causes him little inconvenience. He has had a slight degree of dyspnœa on exertion for about a week. This has also been noticed at night, unless he uses an extra pillow, and has hindered him in dropping off to sleep for a few days. He has had nothing in the nature of an asthmatic attack and is not subject to colds and coughs as a general rule.

The chest wall is well formed and covered. It moves freely with respiration. Expansion is/
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is equal and symmetrical on each side of the thorax. Palpation revealed the usual amount of femities found in a man of his build.

On percussion the note is resonant throughout the lungs.

The breath sounds are vesicular but weak in intensity all over the chest. No accompaniments are heard either at the bases of the lungs or elsewhere.

NERVOUS SYSTEM.

He has had fits of an epileptiform nature since a head injury and within the last few years "fainting turns". Within the week previous to admission to hospital his sleep had been poor. His memory for recent events is a little defective, and he tends to ramble in conversation from one topic to another. He repeats himself and this, with the presence of a certain hesitancy in speech amounting almost to a stammer, makes his conversation rather confused. He is nervous, anxious about his personal welfare, and inclined to be introspective, magnifying the least ache or pain.

No abnormalities were detected in any of the cranial nerves with the exception of the olfactory. Since the bicycle accident he has had no sense of smell. The pupils are equal in size, rather small, perfectly round and contract briskly to light and accommodation.

There is no loss of sensory or motor power. The leg muscles are well developed. The tendon jerks are easily elicited and equal on each side. Babinski's test gives a flexor response on each side.

There is no disorder of gait, no ataxia and no Rombergism.

GENITO URINARY SYSTEM/
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GENITO URINARY SYSTEM.

A prostatectomy was performed one-and-a-half years ago. Since then his urinary symptoms, which were frequency or micturition and polyuria, have been completely relieved.

The urine output averages 1000 c.c. daily. The specific gravity is 1017 and the reaction acid. It occasionally contains a faint trace of albumen, but no sugar or other abnormal constituents. A microscopic examination revealed a few cells but no casts.

LOCOMOTOR SYSTEM.

He has always been an active man until quite recently.

The muscles are well developed. The joint movements are perfectly free. There is a trace of oedema on the posterior aspect of each ankle. Pitting on pressure is elicited.

BACTERIOLOGICAL REPORT. (2.5.'32)

The Blood Wassermann is negative.

BIOCHEMICAL REPORT. (7.5.'32)

The blood Urea \( N_2 \) is 20 mgms%

The blood Creatinine is 2.7 mgms%

COURSE in HOSPITAL.

During the first ten days of observation he continued to have mild attacks of breathlessness at night which tended to prevent sleep and caused him often to waken with a start. These symptoms soon subsided. He was troubled with flatulence and some difficulty was experienced in providing/
providing a daily action of the bowels. When this result was achieved the flatulence and resulting pain ceased to trouble him. From time to time, during his first fortnight in hospital, a mild degree of periodic breathing - waxing and waning respirations - but with no apnoeic intervals, was observed during sleep, particularly when the head was low on the pillows. He did not complain of this and it was not continually present. The oedema, which was noted on his admission to hospital, soon cleared spontaneously with the provision of complete rest in bed. Treatment with digitalis greatly improved all his symptoms, and, after gradually increasing activities, he was discharged feeling very well and able to go about briskly without distress. No Stokes-Adams attacks were witnessed while he was in the hospital, nor had he any symptoms to suggest their presence while he was under observation. Alive October 1932.

THE CLINICAL DIAGNOSIS:-

Etiological: Arterio-Sclerosis
Structure: Cardiac hypertrophy
Mitral insufficiency
Rhythm: Complete heart-block
Functional: Grade 2a.

SUMMARY of CASE 4. (W.T.)

A male aged 65 (1932). In his youth was an athlete and in later life indulged in more strenuous exercise than usual for one of his years. After a bicycle accident, he developed epileptic attacks which ultimately ceased. In 1929 he began to have fainting turns, probably mild Stokes-Adams seizures. These occurred at intervals of a few weeks until his admission to hospital. Nothing to suggest an attack was witnessed in hospital. He had/
had had slight dyspnoea, and a trifling amount of oedema for a few days before admission. Flatulence was troublesome in hospital. No etiological cause was demonstrable beyond arteriosclerosis. The heart rate was 28-32 per minute. The B.P. averaged 220 systolic, 74 diastolic. The Wassermann reaction (blood) was negative.
CASE 5.

Alex. H. Age 64 (1931) Male.

Occupation, Street Scavenger.

Admitted to Royal Infirmary 10th August 1931, and discharged 19th September 1931. Readmitted 22nd February 1932 and discharged 23rd April 1932. (Dr Alexander Goodall kindly placed this case at my disposal. The patient was transferred to Professor Murray Lyon's ward, where I had the opportunity of investigating the cardiac condition.

COMPLAINT. Shortness of breath on exertion for 3 to 4 years.
Cough for one month.

For approximately three and a half years the patient has suffered from a gradually increasing degree of breathlessness on exertion. When precisely this symptom commenced he is unable to state. At first he paid little attention to it and only within the last year has he found it a handicap to his activities. He continued to go about and exert himself and has been able to carry heavy loads for a short distance without undue fatigue. In December 1930 he was engaged in his usual occupation when he was suddenly seized with a feeling of dizziness and faintness. He fell in the street and was unconscious for a few minutes. He was carried to his home a short distance away, and remained in bed for a few days. Since that time the breathlessness on exertion has been more easily provoked than formerly and he has been forced to limit his activities on this account. For the past four months his health has been deteriorating. He is more easily fatigued than formerly, his breathing has become increasingly difficult and in the past month he has developed an irritating cough accompanied by a muco-purulent sputum. The dyspnoea has not occurred when asleep at night but bouts of coughing interrupt his rest and for this reason he has suffered from some degree of insomnia. He has had no swelling of the ankles, no praecordial pain and no palpitation.

PREVIOUS ILLNESSES.

Repeated attacks of bronchitis, which he attributes/
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attributes to exposure and damp at his work. He had an attack of pneumonia in 1920 from which he made a protracted recovery. He had an accident to the right eye in 1923. He has never suffered from rheumatic fever, rheumatism, nor diphtheria. He has had no other illness to his knowledge. He has not suffered from venereal disease.

FAMILY HISTORY.

His father and mother have been dead for many years. The cause of death in each instance is unknown to him. One brother died at age 70 and one sister at age 68, each of unknown cause. His wife is alive and well. He has a family of 9 children of whom three daughters died in childhood. The remainder are in good health at the present time.

SOCIAL HISTORY.

His work as a scavenger in the streets exposes him to all kinds of weather. He is a heavy smoker and drinks alcohol, mostly in the form of beer, with regularity but in small amounts and not to excess.

STATE ON EXAMINATION.

The patient is a poorly developed man, thin and under nourished. He is 5 ft. 4½ inches in height and 8 stones 6 ¾ lbs. in weight, being approximately 2 stone under weight for his age and height. In appearance he looks decidedly older than his years. He is able to lie in bed in no distress with one pillow to support his head. He is not breathless at rest and presents no cyanosis. The face is weather-beaten. The eyes water and the conjunctiva of each, particularly the right, is injected. He is of a cheerful disposition and is interested in all the ward activities.

CIRCULATORY SYSTEM.

Pulse. The radial pulse is regular in time and force. The rate is 34 per minute. The upstroke is abrupt and powerful, well sustained and falls away gradually. The vessel wall is hardy and wiry. The artery is thickened and a little tortuous. The blood pressure is 162 systolic/
systolic, 84 diastolic. The radial pulses are of similar character. Capillary pulsation is absent both under the nail and in the mucous membrane of the lips. There is no clubbing of the fingers. The hands are warm and there is no cyanosis. The superficial veins of the arms are not distended.

**Chest and Neck.** The chest is poorly covered. The ribs are prominent and the interspaces hollowed. The chest is somewhat barrel-shaped and the muscles of poor development. The neck is thin. The thyroid gland is not enlarged. The veins are not unduly prominent and there is no obvious pulsation over the jugular bulb. Waves attributable to auricular systole cannot be detected in the veins of the neck, even by altering the position of the head. The carotid pulse is just visible as a slow regular beat.

The praecordium is of normal form. The chest wall is thin and the interspaces hollowed. There is no abnormal pulsation to be seen. The apex beat is detected in the 5th left interspace in the mid-clarivular line. On palpation the apical thrust is slow in rate, regular in time and of a prolonged forcible nature. It is localised to a small area. There is no thrill at the apex or at the base. No diastolic is felt. The lowest and outermost point of pulsation is 4½ inches from the mid-sternal line. On percussion the position of the left border of the heart is confirmed. The right is one inch from the mid-sternal line in the 4th interspace. On auscultation the heart sounds are pure. No murmurs are heard at any of the valvular areas. The first mitral sound is loud. The second sound at the base is accentuated at both the aortic and the pulmonary areas. No sounds attributable to auricular systoles are heard and the first mitral sounds remain of the same character from cycle to cycle.

**Respiratory System.**

The patient complains of a cough, usually worst in the morning and accompanied by the expectoration of muco-purulent sputum. The repeated coughing, particularly at night, interferes with his sleep.

The chest is somewhat barrel-shaped and
the costal angle is wide. Expansion is deficient on both sides but equal. Vocal fremitus is of the usual intensity. On percussion a hyper-resonant note is detected over the lungs. The area of superficial cardiac dullness is decreased. Auscultation reveals that the breath sounds are harsh in character. Expiration is considerably prolonged. Rhonchi and rales are heard over both lungs.

**ALIMENTARY SYSTEM.**

No subjective phenomena. His appetite is fairly good. The bowels act regularly each day. He does not suffer from flatulence.

Only a few teeth remain. These are incisors. The gums are unhealthy and pus is discharged from the teeth sockets. The tongue is clean and moist. The tonsils are not enlarged.

The abdomen is poorly covered. The anterior wall moves freely with respiration. No tenderness is detected. The liver and spleen are not enlarged.

**GENITO URINARY SYSTEM.**

He has at times some frequency of micturition. There is no incontinence. Examination of the urine: the average daily output is between 1500 - 2000 c.c.s. The specific gravity is 1018. There are no abnormal constituents.

**NERVOUS SYSTEM.**

Examination of the nervous system reveals no abnormalities. The reflexes are equal on the two sides. There is no loss of muscular power. Sensation is perfect throughout.

**BACTERIOLOGICAL REPORT. 13-8-31.**

Blood Wasserman is negative.

**X-RAY REPORT. 12-8-31.**

The heart's diameter is enlarged in the antero-posterior view. The outline suggests some slight hypertrophy of the left ventricle.
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The great vessels show no evidence of dilatation.

COURSE IN HOSPITAL.

During the five weeks in which this patient was in hospital his general condition improved considerably. His cough became much less frequent and his hours of sleep became sounder and longer. After a few weeks in bed he was soon able to be up and about in the ward. He was discharged much improved in health, the pulse rate remaining in the neighbourhood of 36.

SECOND ADMISSION. (22nd February until 23rd April 1932)

He was readmitted to Ward 21 for further observation. Since his discharge from hospital he had remained in fairly good health and has found casual employment. Ten days ago, while engaged in unloading a lorry, a heavy box slipped in his hands and the corner struck him a glancing blow on the right temple. He sustained a lacerated wound about an inch in length which required stitching. The breathlessness has not troubled him so much since he was last in the hospital but within the past month there has been a return of the cough and expectoration. He has had no further fainting attacks, no palpitation and no oedema of the feet or ankles.

Physical examination was the same as the previous admission. He had lost some weight since his discharge from hospital. The pulse was regular at 36 per minute. During the period he was in hospital the pulse rate was recorded hourly and observations were made regarding the effect of certain drugs and other procedures on the heart rate. He was discharged from hospital after a stay of two months in fairly good health and has reported back at hospital several times since. His health remains quite satisfactory, but has been forced to give up attempting to work on account of dyspnoea easily induced.

THE CLINICAL DIAGNOSIS IN THIS CASE WAS

Etiological. Arterio-sclerosis
          Chronic bronchitis and emphysema.

Structure/
CASE 5.

Structure. Cardiac hypertrophy.
Rhythm. Complete heart-block.
Functional. Grade 2b.

SUMMARY OF CASE 5. (A. H.)

Male aged 64. (1931). Suffered from slight degree of breathlessness for three or four years, gradually handicapping him at his work. Had a sudden and unexpected fainting attack on the street 8 months previous to admission to hospital, was unconscious for a few minutes. Increasing weakness since then. Admitted to hospital on account of acute exacerbation of chronic bronchitis. Pulse rate 34 per minute then discovered for the first time. Bronchitis soon subsided and he was able to return to work. He was readmitted for further investigation. The Wassermann reaction was negative. The blood pressure was 162 systolic and 84 diastolic. The heart was a trifle enlarged without any valvular lesion. He had no signs of congestive heart failure and was able to go about and in spite of dyspnoea, was fit enough to gain casual employment for a time as a labourer, but has since been forced to give up active duties.
CASE 6.


Occupation, a forester.

Admitted to the Royal Infirmary on 5th April, 1931 and discharged 2nd. June, 1931.
(This patient was under the care of Dr. John D. Comrie, who has kindly placed the clinical notes at my disposal and has given me the opportunity of making observations on the response of the heart to various procedures.)

COMPLAINT.

Shortness of breath on exertion, and increasing fatigue of 1½ years duration.

HISTORY OF ILLNESS.

In October 1929 the patient was engaged in his usual occupation which necessitates a fair amount of physical exertion. One afternoon, while engaged in cutting a hedge, he experienced a feeling of weakness in his chest and became momentarily light-headed and giddy. He did not faint, stagger or lose consciousness and felt well enough to continue his work in a few minutes. At the end of the day, he cycled home as usual, a distance of four or five miles. On reaching his house he felt unusually tired, short of breath and quite exhausted. He retired to bed but felt well enough to continue his work the next day. During the succeeding month he continued with his occupation but noticed that he had little of his former vigour and that tasks which were previously accomplished with ease made him unduly tired and breathless. After suffering from these symptoms for almost a month he consulted his doctor who told him he had developed heart-block. On his doctor's advice he stayed in bed for nearly two weeks and was absent from work for a month. The rest was only of temporary benefit for on resuming work he found that by the end of two or three weeks he was again becoming incapacitated as the result of similar symptoms.

By/
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By conserving his strength and doing less work of a strenuous nature he was able to continue his occupation for nine months. In June 1930 the breathlessness and fatigue again began to handicap his activities and about this time he noticed a puffiness of the feet and ankles towards evening. He again consulted his doctor, who finding his feet and ankles swollen, advised him to have a further period of rest. He remained in bed for nearly a month and by the end of this time was so much improved in health that he felt able to return to his work. In spite of fatigue and breathlessness on exertion he was able to continue his employment until January 1931.

During the succeeding month all his symptoms gradually became worse than formerly. The breathlessness was more easily induced than ever, the sense of exhaustion so great that he had to spend long hours in bed, and the swelling of the ankles more extensive than ever before. In February 1931 a jaundiced tint appeared in the eyes and gradually spread over his face and chest. He remained in bed for a fortnight by which time the jaundice had disappeared and he felt well enough to go about again. He recommenced his work but found the exertion too much for him and since then has spent the greater part of the past month in bed or sitting about the house. A sense of weakness in the chest has troubled him greatly during the past few weeks but he has had no pain, nor palpitation. If he remains quiet he feels reasonably well but the slightest exertion induces breathlessness and a sense of exhaustion. He has had no recurrence of the giddiness which marked the onset of the present illness one and a half years ago, but during the past fortnight the swelling of the ankles has returned.

PREVIOUS ILLNESSES.

In childhood he had repeated sore throats culminating in a severe attack of diphtheria which confined him to bed for three months. This illness was responsible for a paralysis of both/
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both legs, which prevented him getting up and which lasted for several weeks.

He has not suffered from rheumatic fever scarlet fever, chorea or influenza. He has had no illnesses of any severity with the exception of diphtheria and occasional colic. Venereal infection is denied.

FAMILY HISTORY.

His father died, cause unknown, before the patient was born. His mother died at the age of 81 of heart failure. He has two brothers, both are alive and well. One sister is alive and well and one sister died at the age of 42 of cancer of the throat.

SOCIAL HISTORY.

He has worked as a forester all his life and has been accustomed to ride his bicycle to and from his work for many years. He has been in the habit of cycling eight to twelve miles a day. In 1914 he enlisted and served through the whole war in an infantry battalion. He fought in France, kept perfectly fit, and was discharged in good health at the close of hostilities. He then resumed his occupation and has been in regular employment until his present illness commenced one and a half years ago. He smokes only a small quantity of tobacco in the day. Though not a teetotaller his consumption of alcohol is small and never to excess.

STATE ON EXAMINATION.

The patient is a keen intelligent man of good muscular development. He lies in bed in no distress. He is not short of breath but there is a faint cyanotic tint of the lips and lobes of the ears. The face is sallow in colour, and has lost all the usual appearances associated with an active out-door occupation. His expression is a little anxious. The features are drawn. The face is thin and the cheeks hollowed. The sallowness of/
of the skin is such as to suggest the presence of a slight degree of jaundice, but the conjunctivae are a clear white colour and there is no staining of the palate. There are two small sebaceous cysts on the right side of the neck behind the ear at the edge of the hair. He is 10 stone 11 pounds in weight.

CIRCULATORY SYSTEM.

The Pulse. The pulse is slow in rate being 30 beats per minute. It is generally regular in time and force but on occasions a single premature weak beat is felt shortly after the full pulse wave. This coupling of a strong and a weak beat continues in series for several cycles. The weak beat is an extrasystole occurring regularly in association with the normal beat and gives rise to the phenomenon of pulsus bigeminus. The coupling of beats at the wrist lasts for varying periods of time. It may be present for a few seconds or several minutes. In the intervals the pulse is perfectly regular of good volume and well sustained. The upstroke is abrupt and powerful. The wave declines gradually. The tension of the pulse is raised. The wall of the radial artery is not palpable. There is no thickening or tortuosity of the vessel wall. The Blood Pressure is 158 systolic and 64 diastolic.

Capillary pulsation is not observed under the finger nails or in the lips. The fingers are not clubbed. There is a faint degree of cyanosis of the finger tips and the hands are cold. The superficial veins of the arms are distinctly distended and more prominent than in health, even with the arm lying flat in the horizontal position.

Chest and Neck. The chest wall is thin, but the muscles are well developed. There is no bony deformity and the chest is of normal shape and size. The superficial veins of the neck are swollen and turgid. Regular pulsation is present over the jugular bulb at a rate of approximately 90 per minute. The carotid artery beats/
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beats regularly and slowly at 30 per minute. The thyroid gland is not enlarged.

There are no abnormal pulsations seen over the praecordium and this portion of the chest has the usual contour. Pulsation, slow in rate, is seen in the epigastrium. There is no pulsation visible in the supra-sternal notch.

The Heart. In the region of the apex beat there is a wide area of diffuse pulsation, about the size of half-a-crown and maximum in the fifth space 4½ inches to the left of the mid-sternal line. There is some slight indrawing of the tissues in the 5th. interspace immediately below the maximum pulsation.

On palpation the apex beat is diffuse and is felt over a wider area than normal. The furthest down and outermost point lies behind the 6th. rib 5 inches to the left of the mid-sternal line. No thrill is felt at the apex or over the base. There is no diastolic shock on palpation.

On percussion the left border of the heart is found to be 5½ inches to the left of the mid line in the 5th. interspace. In the fourth right interspace the area of dullness extends at least 1½ inches laterally. The mid-clavicular line is 4½ inches from the mid-sternal line. The heart is therefore enlarged.

On auscultation a soft whiffing systolic murmur accompanying the first sound is heard all over the praecordium. As the mitral area is approached, it increases somewhat in intensity and is loudest at a point just above and within the apex beat. It is also heard less distinctly in the axilla. The second heart sound is pure in all areas. There is no diastolic murmur. The second aortic and second pulmonary sounds are accentuated. At intervals and for a single cardiac cycle the murmur becomes obscured at the apex. It is replaced by a dull thudding sound which has a reduplicated quality. No auricular contractions can be heard. From time to time the rhythm of the/
the heart alters. Premature contractions giving rise to a single heart sound, or two short sharp sounds, are heard occurring regularly after each normal beat. They give rise to a feeble pulsation in the carotid arteries at the wrist.

RESPIRATORY SYSTEM.

He has had no cough or expectoration. There is no breathlessness at rest in bed, but there is a faint tinge of cyanosis in the lips, lobes of the ears and in the nail beds.

The thorax is of normal form. Expansion is equal and free on both sides of the chest. Vocal fremitus is unaltered. The percussion note is resonant though with the exception of a slight degree of impairment at the right apex in the region above the clavicle. In this situation the local fremitus is rather stronger than usual. On the auscultation the breath sounds are everywhere of normal vesicular quality. There are a few scanty moist sounds to be heard at the bases of the lungs particularly on the right. These rales are of medium quality and are heard at the height of inspiration.

ALIMENTARY SYSTEM.

His appetite has been poor recently. He has been bothered with a troublesome hiccough after food from time to time. This usually occurred at his work after dinner and lasted for ten or twenty minutes. He has had no flatulence, no distention of the abdomen, and no pain after food. The bowels are regular.

The mouth is healthy. The lips are faintly cyanosed. The teeth are all artificial. The gums are healthy. The tongue is coated with a thin gray fur. It is moist. The palate, tonsils and pharyngeal wall are a little congested. The tonsils are not enlarged. The lymphatic glands in the neck are small.

The abdomen moves freely. The muscles are flaccid and the subcutaneous tissue is less thick/
thick than usual. The abdomen bulges slightly in the flanks. There is no rigidity and no tenderness detected. The liver is enlarged, the lower edge being felt 1\(\frac{1}{2}\) to 2 inches below the costal margin in the mid-clavicular line. Pulsation of the liver cannot be detected. The liver edge is smooth, rounded in form. It is not tender. The spleen is not enlarged. No free fluid is detected within the peritoneal cavity.

GENITO URINARY SYSTEM.

He has had no urinary symptoms. Examination of the urine revealed a trace of albumen. There were no other abnormal constituents. The specific gravity was 1030, the reaction acid, and the twenty-four hour sample amounted to 1500-2000 c.cs.

NERVOUS AND LOCOMOTOR SYSTEMS.

His memory is good and his intelligence is above the average. He has had no pain. The legs were paralysed in childhood. The cranial nerves. No disorders of function observed. The pupils are of average size, round and equal. They react to light and accommodation.

The muscles are well developed and powerful. There is no muscular weakness or wasting in the legs. The joint movements are free and unrestricted.

The biceps, knee and ankle jerks are present and equal on each side. Babinski's test gives a flexor response on each side. The umbilical jerk is present in each quadrant of the abdomen.

There is some oedema present around the ankles, on the dorsum of the feet and extending up a short way on the posterior aspect of the leg. There is no sacral oedema.

PATHOLOGICAL REPORT.

The Blood Wassermann reaction is nega-
CASE 6.

negative. 27:4:31.

COURSE IN HOSPITAL.

When admitted to hospital this man had signs of congestive heart failure. The heart-block was complete with auricular rate in the region of 60 and a ventricular 30 per minute. At the end of a week's rest in bed the oedema had disappeared, the liver had shrunk under the costal margin, and the urine cleared of albumen. He improved very considerably with rest and was discharged to a convalescent home. On the day before leaving the Royal Infirmary it was observed that the regular auricular waves in the neck were no longer present. An electrocardiogram demonstrated that the regular auricular contraction had been replaced by auricular fibrillation, the heart-block remaining complete. Four weeks later, on his reporting at the Infirmary, auricular fibrillation still persisted. He was then in fairly good health, able to go about with a greatly diminished response to effort.

CLINICAL DIAGNOSIS.

Etiological "Toxic"? diphtheria.

Structural Cardiac hypertrophy.
Mitral insufficiency.

Rhythm Complete Heart-Block.
Auricular fibrillation.

Functional Grade 2b.

SUMMARY OF CASE 6. (J. W.)

A male aged 48, suffered from severe attack of diphtheria at the age of 12. He joined the army at the age of 26 and served throughout the war. Works as a forester and in October 1929 had a giddy attack at his work. Since then increasing dyspnoea and fatigue on exertion culminating in an attack of congestive heart failure for which he was admitted to hospital. The heart rate was/
CASE 6.

was 30. The blood pressure was 158 systolic, 64 diastolic. The blood Wassermann was negative. The heart was enlarged. He was treated with digitalis and on his discharge from hospital it was found that the regular sequence of auricular contractions had been replaced by auricular fibrillation. The ventricular rate remained about 30 per minute. Alive October 1932.
CASE 7.

Occupation, Miner.

(Professor W. T. Ritchie invited me to make certain investigations and has kindly placed his case-records and clinical notes at my disposal.)

COMPLAINT:

Repeated fainting attacks. Five weeks duration.

Until five weeks ago the patient felt in his usual health. One afternoon, while he was sitting in his chair by the fireside, he suddenly lost consciousness and fell to the floor. He had never previously experienced a fainting attack. After an indefinite period, probably a few minutes, he recovered consciousness and commenced to take his boots and clothes off preparatory to going to bed. Whilst doing so, he again had another turn, again fell unconscious on the floor and after a few minutes recovered. He was able to undress, but had no sooner got into bed when a third fainting attack occurred similar to the others. He was in bed for three days, remained free of further attacks and was soon able to go about again. Exertion, however, produced breathlessness and he did not feel able to do much. On the day before admission identical attacks recurred in rapid succession. He fainted repeatedly at intervals of about 15 minutes. On the previous evening he had drunk a little brandy, and the following morning ate a hearty and rather heavy breakfast. The seizures commenced between 1 and 2 o'clock that afternoon and continued at short intervals until he was admitted next morning to Ward 23. The attacks varied in severity, some lasting longer than others. Consciousness was lost suddenly and in a few seconds regained just as abruptly.
CASE 7.

PREVIOUS ILLNESSES:

He had gonorrhoea at the age of 31. He had also been infected with syphilis in youth and was admitted to Ward 5a the Royal Infirmary in September 1925 on account of acute retention of urine. On examination at this time he was found to have tabes dorsalis. Since that time he has attended regularly for anti-specific treatment.

FAMILY AND SOCIAL HISTORY:

Nothing of significance to note.

STATE ON EXAMINATION:

The patient is a man of rather less than average height and weighs 11 stone 11½ lbs. He is of stocky build and muscALLY well developed. He lies back comfortably in bed. The face is drawn and anxious, and both cheeks have a spotted eczematous appearance. His colour is good and the face is not otherwise abnormal. There is no cyanosis. The respirations are a little quickened, but breathing is not embarrassed. Between 2-50 p.m. and 3 p.m. he had three Stokes-Adams seizures in rapid succession.

DESCRIPTION OF A SEIZURE:

The patient sits up in bed breathing quietly and slowly his face bearing an anxious expression. The pulse is felt to be slow, but regular, usually in the neighbourhood of 40 per minute of moderate volume and tension. The observer feeling the pulse notes its sudden disappearance and on auscultation, the absence of the heart sounds. For a few seconds no change is observed in the patient's demeanour or respiratory rate. After about ten seconds from the time of the disappearance of the pulse wave, the patient becomes aware of something amiss and more than once said "It's coming, Doctor" before lapsing into unconsciousness. This usually occurs about twelve seconds from the commencement of asystole. Then the breathing becomes more and more stertorous, the respirations shallow and infrequent. The patient by this time is deathly pale. At an interval varying between 20 and 30 seconds from the beginning of ventricular asystole, convulsive movements appear. The/
The head is moved slowly from side to side, the chin being elevated as if he were seeking relief from a tight band round the neck. The upper extremities stiffen. The right forearm is usually pronated and brought across the front of the chest. Slight jerking movements of a to and fro character then occur. Clonic movements of the upper extremities are observed in some of the attacks, the shoulders being alternately raised and depressed, and the outstretched arms appearing to shiver.

By this time it is noticed that the patient's eyes are open and staring, the eyeballs being usually turned upward and to the right but sometimes directed straight ahead. The pupils dilate and do not react to light. The corneal reflex is lost. After taking about 30 stertorous respirations, the last three or four breaths being at longer intervals than the others, the breathing ceases. This apnoeic period varies greatly in length and may be as short as five seconds or as long as fifty. The pulse returns at the wrist usually about 15 seconds after the apnoeic period begins. The pulse may be absent as long as 70 seconds.

After three or four beats have been felt at the wrist, the patient suddenly regains consciousness and the pallor of his face is replaced by a pink flush. He recovers immediately and in a moment or two is able to resume conversation. During the unconsciousness period he states that he has no dreams or other subjective sensations.

CIRCULATORY SYSTEM:

Between attacks the pulse is regular in time and force. It is of moderate volume, and the tension is slightly raised. The pulse wave is quite normal, full, and falls away rapidly. The vessel wall is palpable and uniformly thickened. The Blood Pressure is 158 systolic and 94 diastolic.

The chest is of normal form and well covered. The neck is thick set. The thyroid gland is not enlarged. On the right side, over the jugular bulb and extending up into the veins of the neck there is a rapid but slight venous pulsation at a rate of approximately 120 per minute. Occurring independently but regularly there/
there is a slower and more powerful beat of larger volume in the carotid artery. Three or four little venous waves intervene between successive carotid beats.

No abnormal pulsations are seen over the praecordium, which is of normal form. The apical on pulse is invisible and unpalpable. No thrills are felt either in the region of the apex beat nor at the base of the heart.

The size of the heart is difficult to determine. The left border appears to be $\frac{3}{2}$ to 1 inch outside the mid-clavicular line in the 5th. interspace. The right border could not be percussed.

The heart sounds are pure in all areas. Over the entire praecordium but heard with greater clearness in the mitral area sounds attributable to auricular systole are detected. They vary in distinctness from time to time. On certain occasions they cannot be detected. (While the sounds were generally present, yet on certain days they could not be recognised). The auricular sound, when present, is heard most clearly in the middle of the long ventricular diastolic. It is not unlike the ordinary first sound in the mitral area, but is of shorter duration, more distant, and of less intensity. It is of the nature of a dull short thud and may be heard occurring regularly sometimes louder in early diastole, sometimes late, and occasionally coinciding with the first ventricular sound which is then louder and more intense.

RESPIRATORY SYSTEM:

There is some congestion of the bases of both lungs. No consolidation is detected.

ALIMENTARY SYSTEM:

The abdomen moves freely with respiration and the abdominal wall is well covered. There is no tenderness detected. The liver and spleen are not enlarged. There is no ascites.

GENITO URINARY SYSTEM:

In September 1925 the patient had an acute/
acute retention of the urine. No urinary trouble since then.

Analysis of the urine. The daily output amounts to 1000 c.c.s. The reaction is acid and the specific gravity 1021. It contains albumen and blood. Microscopic examination reveals the presence of red and white blood corpuscles and epithelial cells.

NERVOUS SYSTEM:

In August 1925 he complained of shooting pains in the legs, lost voluntary control of urination, and was unsteady in gait. In September 1925 examination of the nervous system showed evidence of syphilitic involvement. The pupils were equal in size, the left being slightly irregular in shape. Both reacted to light and accommodation but the shade reflex was sluggish. There was a left lateral nystagmus in both eyes (probably occupational). The knee jerks were both brisk. The ankle jerks were not elicited. The plantar reflex on the left side was flexor, the right indefinite. There was some loss of sensation on the outer part of the legs and soles of the feet. Slight Rombergism was present and the gait was ataxic. Though the Wassermann reaction was negative, the cerebro-spinal fluid showed globulin ++++, and a colloidal gold curve of 335551100. The cell content was increased.

Under treatment his general condition improved materially. No new physical signs appeared in the nervous system and the spinal fluid showed a steady improvement. In October 1930 examination of the spinal fluid revealed the presence of 2 cells per c.mm., no increase in the globulin content, the Wassermann reaction negative and a colloidal gold curve of 0111000000.

RADIOLOGICAL REPORT: (24-7-31).

On screening the chest in the antero-posterior view of the heart was seen to be enlarged in its transverse diameter. The disturbed rhythm of the heart was recognised, the right auricle being seen to beat independently of the ventricles and about three times as fast.

COURSE IN HOSPITAL:

Numerous Stokes-Adams seizures were observed on the day of admission to hospital. Between 2.50 p.m. and 11.40 p.m. he had thirty-six attacks/
attacks, the pulse being absent at the wrist for varying periods of time up to 25 seconds. The following day only one fit was observed at 2.30 p.m. Thereafter no fit occurred until July 24th - a month later. They continued practically incessantly from that time until his death took place about 4.30 p.m. on 26th. July. From the time of admission until the 26th. June the block was complete. On this date the block had decreased to a regular ratio of 2 to 1 with P-R interval of 0.2010 seconds. Two minutes after the injection of 1.0cc. adrenaline on the 26th. June the block again became temporarily complete but reverted to 2 to 1 some 20 minutes later. A few days later normal rhythm became established, the rate averaging 67 with a P-R interval 0.220 seconds. Left vagus pressure at this time produced 2 to 1 block with a P-R interval of 0.180 seconds. Otherwise the same rhythm remained steady. By this time the patient's general condition was much improved. On the 14th. July this rhythm reverted spontaneously to complete heart-block without the occurrence of fits. He looked and felt better and on the 18th. July he was allowed to get up from bed and sit by the fire. On the 23rd. he was up for 2 hours. On the 24th. he was X-rayed, complete auricular-ventricular dissociation being recognised by screen examination. On returning to the ward a series of fits commenced. These continued incessantly until his death on the 26th.

THE CLINICAL DIAGNOSIS:

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<th>Etiology</th>
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<th>?Syphilis.</th>
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<td>Cardiac dilatation,</td>
<td>?Acute myocarditis.</td>
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<td>Rhythm</td>
<td>Intermittent complete heart-block.</td>
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<td>Stokes-Adam syndrome.</td>
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<td>Function</td>
<td>Grade III. (Death).</td>
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<td></td>
<td>Stokes-Adams Syndrome.</td>
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POST/
POST MORTEM EXAMINATION:

An examination of the body was made on the day after death. Only the essential findings are described here.

The pericardial sac contained a large quantity of clear serous fluid. The pleural and peritoneal cavities appeared to be healthy. The heart was enlarged due to dilation of both the right and left sides. The muscle was soft and flabby. The heart was preserved intact for further detailed examination. Professor Ritchie has since informed me that in none of a large number of sections of the bundle of His and A-V node has a structural lesion been found. The first part of the ascending aorta was not seen. The remainder showed slight atheromatous changes, but nothing suggestive of syphilitic aortitis. The respiratory organs (larynx trachea and bronchi and lungs) were congested. In the lungs the congestion was very intense particularly towards the bases. There was no consolidation or infarction. The stomach, duodenum and intestines showed considerable congestion of the mucosa. There was no ulceration. The liver was of average size, soft in consistency and pale in colour. The appearances were those of a "nutmeg" liver with fatty change. The pancreas showed considerable post-mortem change. The spleen was of normal size, very firm in consistency, and dark red in colour, having the appearance of chronic venous congestion. The kidneys were of average size, of india-rubber like consistency and deeply congested. The cortex and medulla were of average proportions and quite well differentiated. The appearances were these of chronic venous congestion. There were no obvious naked eye changes in the brain, medulla, cerebellum or spinal cord.

THE PATHOLOGICAL DIAGNOSIS:

Arterio-sclerosis.
Cardiac dilatation.
Myocarditis syphilitic.
Chronic venous congestion.

SUMMARY/
SUMMARY OF CASE 7. (R.R.).

A male aged 57. (1931). Syphilitic infection in youth, developed symptoms locomotor ataxia in 1925. Received thorough treatment from this time with improvement in his general condition. In May 1925 had three Stokes-Adams seizures. Remained free of attacks for a month and then had repeated seizures at intervals of about 15 minutes on the day before admission to hospital. These continued during his first day under observation in the Ward (22nd. June '31) when he was found to have complete heart-block with an A/V ratio of 96 to 36. The attacks ceased spontaneously, the block remaining complete until the 26th. June, when 2 to 1 rhythm appeared. On the 2nd. July full conduction became established and the heart continued to beat in normal rhythm until the 14th. July when the block again relapsed to complete dissociation without seizures occurring to mark inception of the new ventricular rhythm. Repeated seizures recommenced on the 24th. July, and in spite of all treatment, continued until his death on the afternoon of the 26th. The heart was found dilated. There was some degree of arterio sclerosis. The blood pressure was 158 systolic 94 diastolic during the independent ventricular rhythm, and 110 systolic 78 diastolic during normal rhythm. The blood Wassermann was negative.
CASE 8.


Occupation, a forester.

Admitted to Royal Infirmary on 14th. June 1932 and discharged on 2nd. July, 1932. (The opportunity to study this patient was kindly granted by Professor Edwin Bramwell, and I am also indebted to him for offering to place the clinical notes at my disposal).

COMPLAINT.

Shortness of breath for 10 months.

HISTORY OF ILLNESS.

The patient was in perfectly good health until August 1931, when he began to notice himself a little short of breath on exertion and more easily tired than previously. He can remember distinctly the first occasion on which the breathlessness affected him. He was engaged in cutting the lawn with a motor mower and had done nothing unusually strenuous. The discomfort was so great that he was forced to stop for a moment or two until it passed off. Since then it has tended to grow worse being more easily induced and rendering him unfit for work. Exertion invariably produces breathlessness but occasionally he is troubled by his breathing when lying still in bed at night and at other times when no exertion is being undertaken. He has had to give up attempting to walk at all briskly and to walk, even slowly up a hill, always gives rise to difficulty in breathing. The severer attacks of shortness of breath are accompanied by a certain amount of discomfort almost amounting to pain across the upper part of the chest. This sensation only lasts a moment or two and generally passes off, as the palpitation and breathlessness diminish.

He was able to continue his work, under difficulties, until February 1932 when he suffered from/
CASE 8.

from "influenza complicated by rheumatism". At this time he had a curious feeling in his stomach, which he attributed to wind. The abdomen felt swollen after his meals and he was bothered by repeated eructations. This discomfort lasted for fully a week and passed off gradually without treatment. It still however troubles him for a few days at a time. He was in bed for five weeks complaining chiefly of joints and aching limbs. For two days at the beginning of his illness his feet and ankles were swollen and, the difficulty in breathing was so great that he was forced to sit up in bed at night in an endeavour to get relief. The shortness of breathing prevented him from sleeping and attacks of dyspnoea at night appeared to come and go without obvious cause. He made a gradual recovery, the pains slowly subsiding and in five weeks time he was able to be up and about again. Since then he has not been able to do very much and the ease with which breathlessness is induced has prevented him from resuming his occupation. He has never fainted or suddenly felt giddy.

PREVIOUS ILLNESSES.

At the age of 5 he had a severe attack of scarlet fever and measles. He has never suffered from diphtheria and has had no venereal diseases.

In February 1916, while on service with an infantry battalion in France, he was wounded in the left leg. His slow pulse was first noticed when he was in the base hospital recovering from his wound. He remembers that the doctors in attendance frequently referred to it and asked how long he had had it. He was not able to tell them and, as it apparently caused him no discomfort, he paid no further attention to it.

In November 1916 he was again wounded. He was struck in the right shoulder and both thighs. At a subsequent operation, the head of the right humerus was removed and a new joint formed. He was discharged from the army on this account. His slow pulse was again recognised when/
when he was in hospital at this time.

In September 1925 he suffered from rheumatic fever and was confined to bed for six weeks. His doctor told him that a valve of his heart had been damaged and that it would be necessary for him to avoid strenuous work.

In August 1931 he had "influenza rheumatics" and was in bed for five weeks. The joints were stiff but not swollen. He has had no other illnesses and is at a loss to account for his slow pulse.

FAMILY HISTORY.

His father is alive and well. His mother died at the age of 45 of an unknown cause. He has three sisters and two brothers alive and well. One brother died in infancy. His wife is alive and in good health. She has had no miscarriages. He has two children. Both are alive and well.

SOCIAL HISTORY.

He was 29 years of age when the war commenced. He enlisted and served overseas until his discharge in 1917 as a result of wounds. He then returned to his former occupation. He has always led an active, outdoor life, and has been accustomed to hard physical work. He smokes about 1 oz. of tobacco in a week and is very temperate in regard to alcohol.

STATE ON EXAMINATION.

The patient is a good witness, a little nervous and of a slightly highly strung temperament. His height is 5 ft. 5 inches and his weight is 9 stone 11½ lbs. He sits up in bed and appears quite comfortable. His breathing is free and unembarrassed. There is no cyanosis. He has a very good colour and a healthy appearance. The complexion is ruddy and the face weatherbeaten.

CIRCULATORY SYSTEM.
CASE 8.

CIRCULATORY SYSTEM.

**The Pulse.** The pulse rate is 32 per minute and regular in time and force. The upstroke is abrupt, the wave poorly maintained and falls away with rapidity. The pulse is of the Corrigan type, the peculiar wave being exaggerated by holding the arm in the vertical position. The vessel wall is palpable and a trifle thickened. It can be rolled by the finger during diastole. The vessel wall is hard but not tortuous. The blood pressure is 208 systolic and 116 diastolic.

There is capillary pulsation under the nails and in the mucous membrane of the mouth. The tips of the fingers are not clubbed. The veins of the forearm are swollen, and distended with the arm resting in the horizontal position.

**The Chest and Neck.** The neck is thin. The veins are a trifle more prominent than usual. No regular venous waves in the neck can be detected. The carotid beat is slow and powerful. The thyroid gland is not enlarged.

The chest is well developed and amply covered with subcutaneous fat. The praecordium is of normal form. The apex beat is visible in the 6th, left intercostal space 5 inches from the mid line. The apex is displaced outwards and downwards. On palpation the apical thrust is localised to an area about the size of a shilling. It makes a powerful and prolonged thrust against the chest wall. A faint thrill, systolic in time can be felt, confined to the region of the apex beat. No thrill is felt over the base and there is no diastolic shock.

On percussion the left border of the heart is found to be 5 inches from the mid-sternal line. The right border cannot be percussed. The heart is considerably enlarged. The supra-cardiac dullness in the second intercostal spaces is not increased.

On auscultation a harsh systolic murmur completely/
CASE 8.

completely replaces the first sound at the apex. It is heard far into the axilla and also over the whole of the praecordium, but it is maximum in intensity at the apex. Accompanying the murmur a peculiar high-pitched short squeaking sound is heard. This is confined to apical region and is of maximum loudness about 1 inch within the left border of the heart. It varies somewhat with respiration being louder during the expiratory phase. The second mitral sound is pure but a trifle louder than usual.

At the base a distant systolic murmur accompanies the first aortic sound. The second sound at the 2nd. right costal-ster nal junction is greatly accentuated being loud and booming in character. At the left border of the sternum over the 3rd. and 4th. interspaces the second sound has a ringing quality. It is greatly accentuated and is followed by a short diastolic murmur. This murmur is heard with difficulty but is quite distinct when the breath is held.

No sounds due to auricular systole can be detected.

RESPIRATORY SYSTEM.

No subjective phenomenon, apart from breathlessness chiefly on exertion.

Thorax. The chest is well developed and of normal form. Both sides move equally. Expansion is good, and equal on the two sides, though the patient has some difficulty in taking a full deep breath. There is no change in vocal fremitus. The breath sounds are vesicular. There are no accompaniments and there is no change in vocal resonance.

ALIMENTARY SYSTEM.

He had occasional attacks of flatulence with some discomfort after meals. The appetite is fair. The bowels are regular. He has had no vomiting. The mouth is healthy. The tongue is moist/
moist and clean. The teeth are all artificial.

The abdomen is of normal contour and the abdominal wall moves freely with respiration. No tenderness or rigidity is detected. No abnormal swellings are palpated. The liver and spleen are not enlarged. There is no abnormal dullness. There is no fluid within the peritoneal sac.

NERVOUS SYSTEM.

His intelligence is good. He has no hallucination delusions, and has never suffered from fits or fainting attacks.

The cranial nerves are healthy. There is no ptosis, squint or diplopia. The ocular movements are full and free. The pupils are round and regular in shape and of equal size. They react to light and accommodation.

REFLEXES.

The abdominal reflex is present. The arm jerks are present and equal. The knee and ankle jerks are present and equal. The plantar reflex yields a flexor response on both sides.

There are no disturbances of sensation.

MOTOR FUNCTION.

The right arm is shorter than the left. The head of the right humerus was shattered as a result of a war wound. The comminuted remains of the bone were removed at a subsequent operation and an artificial joint formed. This gives a free range of movement in all directions, but there is some loss of strength in the arm. It is shorter than the right and all the muscular movements less powerful than those on the opposite side. Both legs are thin. There is no muscular weakness or other abnormality in the arms or legs.

URINARY SYSTEM.

For years he has had frequency of micturition. Since the breathlessness commenced this symptom/
symptom has disappeared and he does not now require to rise at night to pass water. He says that at home he passes a large quantity of water during the day.

ANALYSIS OF THE URINE.

The daily output varies considerably, but the average runs from 1200 to 1700 c.cs. The reaction is acid. The specific gravity 1016. It contains albumen.

BACTERIOLOGICAL REPORT. (16:6:32.)

The Blood Wassermann reaction is negative.

BIOCHEMICAL REPORT. (18:6:32,)

The blood urea N 2 is 15.0 mgms%. The blood creatinine is 2.7 mgms%.

ELECTROCARDIOGRAMS.

Confirmed the presence of complete heart-block and demonstrated that the auricles were fibrillating.

COURSE IN HOSPITAL.

The patient was kept at rest in bed and made good progress. He was allowed to get up after a fortnight in bed. He felt better and was not so breathless as formerly. The musical character of the apical murmur disappeared and as the patient was anxious to return home he was discharged from hospital and advised to be less active and to have longer hours of rest than he had been having.

THE CLINICAL DIAGNOSIS WAS.

Etiology

Rheumatic heart disease.
Chronic interstitial nephritis.

Structural/
CASE 8.

Structural Cardiac Hypertrophy.
Chronic mitral and aortic endocarditis (aortic and mitral regurgitation).

Rhythm Complete heart-block.
Auricular fibrillation.

Functional Grade 2b.

SUMMARY OF CASE 8. (J.B.)

A male aged 47 (1932). Had suffered from scarlet fever at the age of 5. No diphtheria.
At the age of 31, was wounded, while serving in France, and his attention was then drawn to the slow rate of his pulse. Was able to continue active service until discharged on account of further incapacitating wounds. Returned to his former occupation. Remained well until he suffered from rheumatic fever in 1925. In August 1931 dyspnoea on exertion commenced. Had an attack of "influenza-rheumatics" in February 1932. He was then breathless at night and the feet and ankles were swollen. Recovered from this attack to be more breathless on exertion than before. Admitted to hospital 14th. June, 1932, when examination revealed complete heart-block and auricular fibrillation. The pulse rate varied from 32-36. He had mitral and aortic regurgitation, but no evidence of congestive heart failure whilst in hospital. The heart was greatly enlarged. There was nothing in his history to suggest the occurrence of Stokes-Adams attacks and he had none while under observation. The blood pressure was 208 systolic and 116 diastolic. The urine contained albumen. The Wassermann reaction was negative.
Mr. J.B.G. Aged 77 (1932). Retired.

(This patient was seen in consultation at Eskbank with Dr. Somerville, Bonnyrigg and Dr. Ballantyne, Eskbank on the 20th. and 22nd. June 1932)

COMPLAINT.

Repeated fainting attacks 1 week.

In December 1931 the patient was in his usual state of health, when one morning he unexpectedly fainted at the bottom of the stairs. He fell on the floor and was unconscious for a few moments. He was living in Corstorphine at that time and the doctor who saw him told him that he should spend a few days in bed and take things more quietly. He soon felt all right and was up and about again within a week. He felt no ill effects until 13th. June, 1932 when he had quite unexpectedly another attack. On the 18th. and 19th. June he had several attacks - perhaps eight or ten in all coming on as a rule about meal times. Dr. Somerville told me that his impression was that the act of swallowing induced certain attack though a number occurred without recognizable cause. He was confined to bed from the 18th. but only slept at intervals. On the morning of the 20th. he had a severe attack at 7-50 a.m. immediately after one mouthful of toast and a little tea. The pulse was absent at the wrist for almost three minutes (this was carefully timed by the nurse in attendance who was aware of the possible association of swallowing with the production of attacks and was prepared to note the effect). He became very pale, then deeply cyanosed with twitching of the mouth and was completely unconscious. The breathing became stertorous, slow and shallow and then ceased for nearly half a minute. Suddenly the pulse started again and the lividity of the face gave way to a bright pink flush. He had no further attacks this day until 6-35 p.m. - less than five minutes after swallowing a glass of milk. This was not so severe as the seizure of the morning but he was/
was unconscious for nearly 2 minutes. At 7.30 p.m., he had water to drink, an attack of equal severity followed immediately. Up to that time of the day he had had nine fluid feeds, and three of these were immediately followed by seizures. Dr Somerville and I saw him together at 8.55 p.m. He was then resting comfortably in bed and said that he felt better. He was in no distress and the breathing was comfortable. The pulse rate throughout the day had varied from 34 to 32.

DESCRIPTION OF STOKES-ADAMS ATTACK.

I had just commenced my examination and was engaged in counting his pulse, when a seizure occurred, without obvious cause. The rate was 30, and as I finished counting, a progressive slowing in rate occurred. In the next half minute there were only 10 beats. The pulse then stopped. His face became pale and he turned to me and said, "it's coming over me again doctor" —— The rest of his words were incoherent and he became deeply comatose in less than half a minute. The eyes turned up, the pupils dilated widely and the breathing became slow and deep. Not a sound was to be heard at the heart. He lay immobile. A tinge of cyanosis appeared over the face and rapidly deepened until he was a dark livid colour. The arms, legs and face then twitched and the extremities passed into clonic convulsions. The movements lasted for only a few seconds until the muscles became flaccid, and the limbs fell back motionless on the bed. The breathing, which up to that point had been slow and stertorous, became gasping, irregular, and gradually ceased. The heart remained still, a faint sweat appeared upon the livid brow, the purple tongue protruded from the angle of the mouth, saliva dribbled over his cheek, and the eyes had a glassy fixed stare. He remained to all intents in this condition well over a minute, during which time we did artificial respiration and nurse filled a hypodermic syringe. He then gave a faint gasp for breath. I had my hand on his pulse and felt the first of the returning beats. They came in rapid succession. During the first minute the rate was 120 and the tension/
tension low. The face flushed pink and the breathing grew in strength. It was at first periodic in type but in a minute Cheyne Stokes respiration developed and a few minutes later normal breathing became established. The pulse dropped to 60 as the breathing became established and a few minutes later the rate was 34 per minute. He did not fully recover consciousness for nearly 20 minutes after the attack and when he did so was quite confused. Half an hour after the attack, while an electrocardiogram was being recorded, he was sufficiently clear mentally to engage in conversation and even apologised for his strange behaviour. He could then remember telling us that he was going to faint.

PREVIOUS ILLNESSES.

He has always been a healthy man. He has had no illnesses of any importance, until three years ago, when a prostatectomy was performed. Subacute cystitis with occasional exacerbations since the operation.

FAMILY HISTORY.

His parents are dead. His wife alive and well. He has two married daughters both alive and well.

STATE ON EXAMINATION. (20 minutes after the seizure)

After the attack described above, he looked tired and exhausted. He was quite a good colour and lay comfortably in bed using one pillow. He was of average height and not over weight. He looked younger - perhaps by ten years, than his age.

CIRCULATORY SYSTEM.

The Pulse. The pulse rate was 34 per minute, regular in time and force. The wave was of full volume with an abrupt and sustained up-stroke. The wave fell away rapidly. The tension was a little raised. The vessel wall thickened and somewhat tortuous. The blood pressure was in the neighbourhood of 160, the diastolic pressure was/
CASE 9.

was low, approximately 60. There was no capillary pulsation and no clubbing of the fingers.

The chest and neck were well covered and of normal form. The thyroid gland was not enlarged. The veins in the neck were a trifle distended, and by turning the head slightly to the left, regular small waves could be seen and counted in the right jugular vein. Their rate was 75 per minute. At regular intervals they were obscured by the powerful carotid beat.

The apex was not visible. There were no abnormal pulsations over the praecordium. The apex beat, on palpation, was found to lie in the fifth left interspace within the mid-clavicular line. It was slow in action and the beat was confined to a small area. The thrust was not unduly forcible. There was no thrill felt at apex or base.

On percussion the left border of the heart in the fifth space was within the mid-clavicular line. The right border could not be percussed. The heart was not enlarged.

On auscultation the heart sounds were pure. There were no murmurs heard. The second aortic sound was a trifle loud but not unduly accentuated. No sounds attributable to auricular systole were heard.

RESPIRATORY SYSTEM.

The chest was well covered and expansion was equal on the two sides. The lungs were resonant. There were a few scattered moist sounds to be heard at the bases of the lungs. Vocal fremitus and resonance were unaltered.

ALIMENTARY SYSTEM.

He had had some flatulence and abdominal discomfort for the past three or four days, and has found that charcoal biscuits which he has been eating from time to time, give him some relief.

The mouth is healthy. The abdomen moves freely/
freely with respiration. There is no tenderness detected. There is a firmly healed mid-line scar below the umbilicus. The liver and spleen are not enlarged. There is no ascitis.

GENITO URINARY SYSTEM.

In recent months he has had some frequency of micturition rising twice at night. Within the last ten days micturition has been accompanied by a burning sensation and the urine has been of a foul odour.

ANALYSIS OF THE URINE.

The daily output is increased, the night sample amounting to 8 or 10 ozs. The urine contains albumen in fair quantity and also pus cells. It has a "fishy" odour. It is strongly acid in reaction.

NERVOUS SYSTEM.

The patient is an intelligent man. He has no hallucinations or delusions. His memory is good. He has been taking "fainting turns" for a week. The cranial nerves are healthy. The pupils react to light and accomodation. There is a slight arcus sensilis. There is no diplopia and the eye movements are free in all directions. The muscular power is good. The limbs are well developed. There is no muscular wasting. There is no oedema.

THE TENDON REFLEXES.

The arm jerks are present and equal. The knee jerks and ankle jerks are present and equal. The plantar reflex gives a flexor response on both sides.

An electrocardiographic examination was made within 30 minutes after the heart beat returned. This demonstrated the presence of complete heart-block with a regular auricular sequence. The rates were, auricular 72.6 and ventricular 34.0 per minute.

COURSE OF ILLNESS.

On the 21st June he had an attack in his sleep at 2.20 a.m. The pulse was absent at the wrist for nearly 4 minutes. There were no twitchings or clonic movements of the muscles in this seizure, though he became deeply cyanosed. This was/
was the last attack he experienced. He commenced taking ephedrine tablets at 10.30 p.m. on the 20th and continued it at 4 hourly intervals thereafter. With the exception of the attack at 2.30 a.m. on the 21st he had a comfortable day. In the evening the pulse and heart rate rose to 74 per minute, without any subjective symptoms. It was 40 in the morning.

On 22nd June 1932, Dr Somerville and I saw him together again at 5.30 p.m. He had had a very comfortable day, had slept well and had no further attacks. He felt much better. The pulse rate was 74. The blood pressure was 156 systolic and 106 diastolic. The lungs were clear of moist sounds. The heart sounds were pure and by percussion the area of cardiac dullness appeared to be of the same size as at the first examination. A second electrocardiogram was recorded at 5.45 p.m. This showed the presence of normal sinus rhythm at a rate of 74.4 per minute with a P-R interval of 0.205 seconds. We decided to continue the ephedrine thrice daily and to have adrenalin ready for injection if attacks recurred.

Dr Ballantyne has been in attendance since then. He has told me that Mr G. continues to make good progress, though the bladder infection is still troublesome. To date (August 1932) he has had no further seizures and has been able to be up and about in the house.

THE CLINICAL DIAGNOSIS WAS:

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Arteriosclerosis</th>
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<td>&quot;Toxic&quot; Subacute cystitis</td>
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<table>
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<th>Focal myocarditis</th>
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<tr>
<th>Rhythm</th>
<th>Intermittent complete heart-block.</th>
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| Functional               | Grade 2b.                      |
|--------------------------| Stokes-Adams Syndrome.         |

SUMMARY/
SUMMARY OF CASE 9. (Mr J. B. G.)

A male aged 77 (1932) Arterio-sclerosis. Subacute cystitis following a prostatectomy. He had a severe fainting attack without warning in December 1931 and remained in good health until 13th June, when he had another attack. On the 18th and 19th June he had in all about ten seizures, some of which were apparently precipitated by swallowing. On the 20th June he had three attacks, in one of which the pulse was absent for over three minutes. An electrocardiogram, recorded shortly after this seizure, illustrated the presence of complete heart-block. The heart was not enlarged. The vessels were arterio-sclerotic. The blood pressure was 160 systolic, 60 diastolic. The ventricular rate was 34 per minute. He commenced taking half a grain of ephedrine 4 hourly by mouth that night, and had one further attack a few hours later. A further electrocardiogram on 22nd June demonstrated that normal rhythm had been restored at a rate of 74 per minute, with a P-R interval of 0.205 sec. There have been no seizures since and he remained comparatively well when last seen in August 1932.
CASE 10.

Mrs. M.C. Aged 84

Occupation, Housewife.

Admitted to Royal Infirmary 13th January, 1932 and died 6th March, 1932. (This patient was seen in consultation with Dr. P. Martin Brodie of Edinburgh, and thereafter admitted to Ward 21 of the Royal Infirmary where Professor Murray Lyon kindly gave me every facility to investigate her condition. I am much indebted to Dr. Brodie who has placed his carefully compiled notes at my disposal).

COMPLAINT.

Flatulence 21 years.

Repeated fainting attacks during 1 year and 8 months.

HISTORY OF ILLNESS.

As the patient's memory was defective and as she was often confused mentally the following history has been constructed largely from Dr. Brodie's careful clinical records and from the statements of her daughter - a trained nurse - who was her mother's constant companion.

The patient first came under Dr. Brodie's care in October 1926 on account of increasing abdominal discomfort. As a child she was delicate and had been difficult to feed. In 1910 she began to have dyspeptic symptoms, frequently suffering from flatulent distension, but in spite of this she remained in fairly good health until 1926, when her digestion began to give rise to increasing discomfort. In October of this year her symptoms were severe enough to make her seek advice. She complained of distension in the upper abdomen after food, felt her clothes too tight for her, and gained relief by frequent eructations of wind. She had no actual pain and her appetite was generally good but she was troubled by infrequent actions of the bowels. The pulse was perfectly normal/
CASE 10.

normal in rate and rhythm at this time. By the use of a dry diet and simple aperients her condition in the course of a month improved materially. She was able to do all that she wished in household work.

In December 1927 she again complained of distension of the abdomen, and in addition had, to use her own words "difficulty in taking breath!" She was kept in bed and it was then noted for the first time that, to breathe with comfort, she insisted upon being propped up with several pillows. She had however no particular breathlessness on exertion and ascribed her difficulties to fullness in the pit of the stomach. Examination at this time revealed a generalised flatulent distension of the abdomen, the absence of ascites and the lower edge of the liver a full inch below the costal margin. The bowels were irregular. There was no melaena and rectal examination was negative. Examination of the pulse suggested the diagnosis of heart block, for Dr. Brodie then found for the first time that the rate was 36 to 38 per minute. The heart rate corresponded to this and was perfectly regular. Careful examination of the heart showed that it was not enlarged and that the heart sounds were pure. There were no murmurs. She was unaware that her pulse had become slow since she was last examined. She had had no faints, "weak-turns", or fits. Dr. Brodie had her under his care for two months at this time and on no occasion had she any symptoms to suggest a Stokes-Adams attack. On one occasion, while feeling the pulse, the rate suddenly quickened from 34 to 60 per minute. This rate continued for at least 3 minutes without her being aware of it. When Dr. Brodie left the house, the heart had returned to its usual rate of 36. This slow rate of the heart continued until the end of January 1928 when it was noticed to be beating at an average rate of from 58 to 64 per minute.

Between 25th. April and 6th. June, 1929 she had an exacerbation of the flatulence and abdominal distension. Her general health had remained good, and she had not lost weight and her breathing/
breathing caused her little or no difficulty. She was able to do her household duties and successfully nursed her daughter during a long illness in the latter part of 1928 and early 1929. The pulse rate was about 60 and the heart on physical examination appeared perfectly normal. She was not confined to bed for more than a day or two at this time.

On 2nd. March 1930, the patient became suddenly ill during the night, suffering from severe abdominal pain of a colicky nature. Her daughter observed that the abdomen was much swollen and administered an enema. A distinct swelling was found in the abdomen on the outer side of the right rectus muscle extending from below the costal margin down to near the right iliac superior spine and deeply seated within the abdomen. It was firm with rounded margins and of greater length than breadth. The heart-block was again recognised to be present - the heart rate being 36. Some days later the patient was seen in consultation with a surgeon who considered that a carcinoma of the hepatic flexure was the cause of the subacute obstruction. In view of her age and the state of her heart, operation was deemed inadvisable. On certain days at this time the pulse rate fell to 28. She still had no faints or convulsive seizures.

On the 5th. April the patient had the first of a long series of Stokes-Adams attacks. Between 5th. April, 1930 and the 13th. January, 1932 (when she was admitted to the Royal Infirmary) she suffered from no less than 352 fully developed typical Stokes-Adams attacks. Milder attacks, which were of fairly frequent occurrence and consisted in a trifling degree of faintness with cessation of the ventricular beat for a few seconds, are not included in this figure. Each of the 352 seizures were accompanied by complete loss of consciousness for varying periods of time. Each attack was accurately recorded by her daughter or other member of the family, one of whom was constantly with her night and day. These symptoms were necessarily distressing to her relatives and most alarming to those attending her, but gradually became/
<table>
<thead>
<tr>
<th>SERIES</th>
<th>DATES INCLUSIVE</th>
<th>NUMBER OF STOKES-ADAMS ATTACKS</th>
<th>DURATIONS OF SERIES</th>
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<tr>
<td>1</td>
<td>April 5 to April 17</td>
<td>17</td>
<td>13</td>
</tr>
<tr>
<td>2</td>
<td>May 28 to June 8</td>
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<td>June 26 to July 4</td>
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<td>July 18 to July 24</td>
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<td>November 16</td>
<td>3</td>
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<td>6</td>
<td>December 20</td>
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<tr>
<td>7</td>
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<td>6</td>
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<td>8</td>
<td>April 13</td>
<td>1</td>
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<td>9</td>
<td>July 18</td>
<td>1</td>
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<td>10</td>
<td>August 28</td>
<td>5</td>
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<tr>
<td>11</td>
<td>Sept. 24 to Sept. 27</td>
<td>5</td>
<td>4</td>
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<td>Dec. 3 to Dec. 6</td>
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<tr>
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<td>Dec. 27 to Dec. 30</td>
<td>131</td>
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<td>January 13</td>
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**TOTAL NUMBER OF STOKES-ADAMS ATTACKS = 352**
became by their mere repetition almost commonplace. In one period of 24 hours (between 1 p.m. on the 29th. to 1 p.m. on the 30th. December, 1931) she had exactly 114 seizures, each accompanied by complete loss of consciousness and temporary arrest of breathing.

The Stokes-Adams seizures tended, as a rule, to come in series for a few days to be followed by comparative freedom from attacks for a few weeks or longer. The intervals of freedom varied considerably. To indicate the incidence, the number and dates of various attacks are here tabulated.

It will be noted that from the 25th. July, 1930 until 26th. December, 1931 there were only 24 seizures. This long period of comparative freedom from attacks was followed by the most acute exacerbation. During four days, (December 17th. to December 30th.) she had 131 seizures, 114 of which occurred in the 24 hour period ending 1 p.m. on 30th. December. She was then free of all attacks until she was admitted to the Royal Infirmary, when a single seizure occurred. I was fortunate to secure a long and continuous film electrocardiogram of this attack. It showed the presence of ventricular fibrillation followed by arrest of the heart. This was the last attack which she was to experience. She died two months later on 6th. March, 1932.

In the great majority of the seizures the pulse was absent for about two minutes. Slight attacks - "threatenings" the patient called them - were present on those days on which severe attacks of unconsciousness were also being experienced. They were absent when she was free of fully developed seizures.

Dr. Brodie, who witnessed a large number of seizures has described them as follows:- "In the middle of a sentence the patient would stop talking, become pale, the eyes become staring and vacant, rolling slightly from side to side, and later fixed and deviated to the left or right. Consciousness/
Consciousness was lost early after three to five seconds. The hands, if previously raised, would fall to the bed, the breathing become shallow and feeble and there would be neither pulse or heart beat. In the more prolonged attacks the patient appeared to be dead. After a period, often of a minute to two minutes, a feeble contraction of the heart, registered by a flicker of the pulse, would be felt and during the next minute several similar contractions might occur - not in any regular rhythm and often not more frequently than seven in the minute, sometimes of even less frequency. Just before the pulse returned the head would often lean to one side - usually the right - the arms and legs become moderately rigid and the muscles of the neck and arms suffer spasmodic contractions. The skin, previously cyanosed and pale, would commence to flush over the neck and face. The respirations would become deep, rapid and noisy, as the breath was sucked in through the clenched jaws and retracted cheeks.

Sometimes attacks came in rapid succession. She would recover from one only to pass into another in a few minutes times. If an attack was not one of rapid sequence the patient would slowly regain consciousness, and in two or three minutes, be able to answer questions, though often remaining dazed for 10 to 15 minutes. Very often there was a little bout of vomiting at this stage. When the attack was a long one, consciousness was not as a rule recovered for five to thirty minutes, and during periods of frequent attacks, the patient remained semiconscious, confused and dazed over hours and even days.

The general condition of the patient deteriorated markedly during the period April to June 1930. The frequent interruptions in the circulation, the loss of sleep, and the difficulty in securing adequate assimilation of food caused her to become very emaciated. It was noticeable, however that in the short intervals of from two to five weeks between the various series of attacks at this time she picked up strength, gained flesh and grew stronger.

Towards the end of June 1930 the swelling of the abdomen was less evident and not so firm.
CASE 10.

Daily examination revealed that the swelling fluctuated in size, occasionally being absent altogether. This suggested the possibility of hypernephrosis. A consulting urologist, who was called to see her at this time, came to the conclusion, that as a loose freely moveable kidney was present on the right side, the swelling was probably due to an intermittent hydronephrosis, and that the flatulence and difficulty in getting the bowels to act were secondary effects.

From the 18th. to 24th. July, 1930 the patient had 35 Stokes-Adams and during this week the swelling in the loin was constantly and very distinctly present. Treatment was chiefly directed to the abdominal condition. Daily enemata, followed by abdominal massage and a firm binder with the foot of the bed raised in blocks proved very efficacious, not only for the relief of the distension but also for the cardiac condition. With the passage of time the conclusion was reached on purely clinical grounds that the abdominal condition had an influence on the production of the heart attacks. If the abdominal tumour was allowed to grow in size the likelihood of Stokes-Adams seizures was greatly increased. On the other hand if no tumour could be felt it was possible to be reasonably sure that no heart attacks were imminent. This conclusion held good for over sixteen months - up to December 1931. From the table to attacks (page ) it can be seen that while this treatment was in progress the frequency of the attacks greatly diminished. When attacks did occur the tumour was easily palpated for a day or two beforehand and the urine output appeared to fall to 14 or 20 ozs. as compared with 30 to 40 ozs. while the tumour was absent. No very large amounts of urine were passed, nor did the swelling disappear suddenly. The urine contained a small amount of albumen from time to time.

In spite of occasional attacks she remained fairly well until 27th. December, 1931. She was able to be up and about in the afternoons, did/
CASE 10.

did a little household work, and showed an interest in the family affairs. As might be expected for one of her age, she was easily tired, frail and had to be careful about her diet. In spite of careful and daily treatment along the lines described above, the swelling reappeared, became large and tense, and a series of attacks of great severity then developed. They were identical with those from which she had previously suffered but much more frequent. In four days she had 151 attacks. In the last 24 hours of this series she had 114 seizures. Many occurred in such rapid succession that she was quite unconscious for hours at a time. Two days after the actual attacks had ceased, she was still confused, very weak and had to be propped up in bed with pillows. In the course of a fortnight her strength began slowly to improve and she was admitted to the Royal Infirmary for further investigation.

PREVIOUS ILLNESSES.

She had apparently the usual childhood's illnesses. She had not had diphtheria as far as is known. She had a mild attack of influenza five or six years ago. "Indigestion" had troubled her, more or less, all her life.

FAMILY and SOCIAL HISTORY.

The cause of death of her father and mother is unknown. Her husband is alive and suffers from rheumatism. She has a daughter and a son both alive and well.

STATE on EXAMINATION.

The patient is an old wizened woman of 84 who looks her age. She sits up in bed, supported by four pillows. The head, neck and shoulders are bent forward, and movements of the trunk are made slowly and stiffly. The skin of the face is sallow in colour, wrinkled and drawn. The expression is a little anxious. The eyes are sunken, the cheeks are drawn, and the hair is dry and scanty. Breathing is shallow and increased in rate - 28 per minute - often irregular but causes her no apparent embarrassment. There is no pallor or cyanosis/
cyanosis. She is very emaciated, and has a tired and exhausted look. She is too weak to have her height and weight recorded.

CIRCULATORY SYSTEM.

The Pulse: The pulse rate is 27 per minute and the wave is regular in time and force. The upstroke is abrupt, well sustained, and falls away gradually. The pulse wave is full, and the tension is raised. The vessel wall is greatly thickened, hard and extremely tortuous. It stands out under the emaciated superficial tissues as a distinct and twisted cord. The pulse is visible to the eye over a considerable stretch of the forearm as a regularly recurring wave. The brachial artery is also visible, hard and tortuous. The Blood Pressure is 170 systolic and 80 diastolic. There is no capillary pulsation and no clubbing of the fingers. The skin of the arms and hands is wrinkled and melastic. The superficial veins are a little swollen. The walls of the veins are thickened and harder than usual. The veins in places are also tortuous.

The chest and neck are thinly covered. The skin is wrinkled and lies in small folds over the intercostal spaces. The thyroid gland is not enlarged. The veins in the neck are not unduly prominent. No regular venous pulsation can be detected even by altering the position of the head.

The praecordium is of normal form. The interspaces are indrawn, and the chest is poorly covered. The apex beat is visible in the 5th interspace, just within the midclavicular line. On palpation the apex is easily felt and makes unusually close contact with the palpating hand. It is a strong localised and sustained thrust falling away gradually. The lowest and outermost point is exactly in the midclavicular line 3½ inches from the middle of the sternum and just above the upper margin of the sixth rib. On percussion the right border of the heart is 1 inch from the mid-sternal line in the 4th right interspace. The area of supracardiac/
CASE 10.

supracardiac dullness in the 2nd spaces is not increased. Auscultation reveals that the heart sounds are pure in character and of a constant quality. Both sounds in all areas are better heard than usual. The second aortic sound is definitely accentuated. There are no murmurs heard. No sound of auricular systole is heard.

RESPIRATORY SYSTEM.

The thorax is of normal form, but very thin. The muscles are poorly developed. Expansion is deficient, but equal on both the two sides. She has some difficulty in taking a deep breath. Vocal fremitus is distinct and easily elicited all over both lungs. The percussion note is resonant throughout. There are no moist sounds heard.

ALIMENTARY SYSTEM.

The abdominal wall is very thin and sunken in the epigastric region. The skin is lax, wrinkled, and in places thrown into loose folds. The anterior wall moves freely with respiration. There is no tenderness detected except on deep palpation in the right loin. There is no muscular rigidity. The lower abdomen is a trifle distended and the overlying muscles are lax and atrophic.

The liver edge can be felt a finger's breadth below the right costal margin in the nipple line. The spleen is not enlarged. The lower pole of the right kidney can be felt indefinitely. There is some resistance to deep palpation in the right loin, but no tumour mass can be defined.

URINARY SYSTEM.

She has occasionally frequency of micturition and may pass water twice during the night. This symptom is irregular and not always present. She has sometimes voided urine involuntarily immediately after a heart attack.

The/
CASE 10.

The daily urine output amounts to 1500 to 2000 cc. The specific gravity is in the neighbourhood of 1012, sometimes being as low as 1005. A faint trace of albumen is occasionally present. There are no other abnormal constituents of the urine. The reaction is generally faintly acid, or neutral.

NERVOUS SYSTEM.

Her memory for recent events is poor. In talking with her, she generally becomes confused and rambles in her train of thought after a few minutes' conversation.

There is no abnormality of the cranial nerves. The pupils are of average size, equal, round in shape and react to light and accommodation. The eye movements are free and equal. There is a very marked arcus senilis. The muscles are poorly developed. The arms and legs are very thin. There is no paralysis. The knee, ankle and arm jerks are present and equal on the two sides. The umbilical reflex is not elicited. The plantar reflex yields a flexor response on both sides. There is no disorder of sensation.

BACTERIOLOGICAL REPORT: 14-1-32. The blood Wassermann reaction is ?

BIOCHEMICAL REPORT: 15-1-32.

Blood Urea N₂ is 19.0 mgms%
Blood Creatinine is 3.5 mgms%
CO₂ combining power of the blood is 63.0 vols.%

11-2-32 The blood Calcium is 10.0 mgms%

COURSE in HOSPITAL.

She/
She was admitted to Ward 21 on the morning of 13th. January 1932. That afternoon she had a Stokes-Adams attack in the electro-cardiograph department, where she had been taken in order to test her response to a dose of atropine. After lying on the examination couch for twenty-five minutes, during which time a series of tracings of the heart beat were recorded, a dose of atropine (gr. 1/30) was administered. Up to that time the patient was perfectly well. The ventricles were beating regularly at a rate of 30.1 per minute save for the presence of an occasional extra-systole. The auricles were fibrillating.


Three electrocardiograms had been recorded and preparation was made to administer 1/30 grain of atropine sulphate intravenously. Difficulty was experienced in entering the point of the needle within one of the superficial veins at the bend of the right elbow, on account of sclerotic condition of the vein wall. In spite of a sharp needle the vein was very resistant and several attempts were made before it was successfully pierced. This procedure caused her a little discomfort, and no sooner had the needle point entered the vein and the injection commenced, than she turned pale, her head fell back, and we realized that a Stokes-Adams attack had started.

The camera was not running at the instant of the injection, but the movements of the electrocardiograph fibre were under observation. These did not cease. A rapid series of ventricular extra-systoles at a very high rate, probably in the neighbourhood of 150-200 per minute, immediately preceded the loss of consciousness. By the time the film camera was started they had ceased, and the subsequent record shows (fig.) that the series of extra-systoles - (probably ventricular tachycardia) - had been replaced by ventricular fibrillation. This continued for 1½ minutes when the heart suddenly ceased to beat. Listening over the heart during the ventricular fibrillation/
CASE 10.

fibrillation nothing was heard. No movement of the heart could be palpated. The left border was outside the mid-clavicular line. Half-way through the course of the ventricular fibrillation, the breathing, which had been stertorous, slow and irregular, gradually ceased. She became a deep, livid colour, with glassy eyes and widely dilated pupils. The limbs jerked violently and in an instant fell immobile. She appeared to be dead.

I filled a syringe with adrenalin and then commenced artificial respiration. This was performed with one hand over the heart in the hope of stimulating it. Artificial respiration was done for fully a minute - the chest was compressed eighteen times - and as there was no sign of even a flicker on the electrocardiograph fibre, and as the breathing was still in abeyance, I injected with a long needle through the 4th. left intercostal space at a point about 1½ ins. from the middle of the sternum, 0.5 c.c. of adrenalin into the heart. When withdrawn, the needle contained dark venous blood and the adrenalin presumably entered the left ventricle. Within five seconds of administering this intracardiac dose of adrenalin the ventricle recommenced beating at a rate of (Fig.). The pulse wave was absent for nearly 4 minutes. Cheyne-Stokes breathing commenced a few seconds after the heart had resumed beating, and continued, in greater or less degree, for some hours.

The attack occurred at 4 p.m. and the Cheyne-Stokes respiration ceased at 10 p.m. She remained deeply comatose until the following morning. She gradually recovered consciousness during the day, but 24 hours after the attack was confused. She slept well that night, and the next day (15th. Jan.) was quite recovered. She never at any time had any recollection of the attack and could not even remember leaving the ward to go to the department for the original examination of the heart.

Atropine, in the same dose, was repeated some days later with no ill effects. She had several/
several venous punctures done on other occasions, and no seizure ever occurred again. It is true that on these occasions a particularly sharp and obliquely bevelled needle was used and that no great difficulty was experienced in entering the vein.

She gained in strength for some weeks, but towards the end of February she became restless at night, confused during the day, and suffered from delusions regarding the care which the nurses were wont to bestow upon her. This was the beginning of the end. She began to suffer from hypostatic congestion of the lungs which increased rapidly in the course of a few days, and she died peacefully on the night of the 6th March 1932, without again suffering from a Stokes-Adams attack.

On the day of death the temperature rose to 101.6° and the pulse rate to 60 - the block remaining complete.

THE CLINICAL DIAGNOSIS WAS:

<table>
<thead>
<tr>
<th>Etiology:</th>
<th>Arterio-sclerotic: Coronary artery sclerosis.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Structural:</td>
<td>Chronic interstitial myocarditis. Cardiac enlargement, ? dilatation.</td>
</tr>
<tr>
<td>Rhythm:</td>
<td>Complete heart block, auricular fibrillation.</td>
</tr>
<tr>
<td></td>
<td>? Paroxysmal ventricular tachycardia.</td>
</tr>
<tr>
<td></td>
<td>Ventricular fibrillation.</td>
</tr>
<tr>
<td>Functional:</td>
<td>Stokes-Adams syndrome.</td>
</tr>
<tr>
<td></td>
<td>Hypostatic congestion of the lungs.</td>
</tr>
<tr>
<td>Grade 3 - Death.</td>
<td></td>
</tr>
</tbody>
</table>

POST-MORTEM EXAMINATION.

An examination of the body was made on the day after death.
CASE 10.

The Heart: The pericardial sac contained a small quantity of pale yellow serous fluid. The pleural and peritoneal sacs were healthy. The pericardial surface was smooth, glistening and transparent. The heart was only slightly larger than usual. All the chambers, especially those on the right side, were dilated. The valves, however, were of normal dimensions and the endocardium was healthy. The inner half of the wall of the left ventricle was distinctly paler than the outer half, and seemed to be the seat of fibrous change. The line of demarcation between the outer and inner halves was fairly well defined. The Aorta: The ascending aorta was healthy. The arch showed a fair number of calcified atheromatous patches, so also did the ascending aorta. The Coronary Arteries: near their origin were a little tortuous. On section the walls were thickened and the lumen reduced in size. There was some spotted atheromatous change on the inner surface. The Larynx, Trachea, and Bronchi were healthy. Lungs: Both pleural surfaces were smooth, glistening and transparent. Each organ was of average size, shape and consistency, and, on section, presented a moderately marked degree of hypostatic congestion. The pulmonary tissue was slightly oedematous. The Oesophagus and Stomach presented no pathological abnormality. The Liver was a little reduced in size but of average shape and consistency. On section, it presented a rather pale surface with some degree of vascular mottling. The Spleen was of average size, shape and consistency. On section, it presented a normally coloured surface, except in one area, which was the seat of a recent infarct. The Intestines were healthy. Sprunging from the anti-mesenteric border of the ileum about two-and-a-half feet above the ileo-colic sphincter there was a Meckel's diverticulum. It was about three inches in length, about an inch or more in width at its point of origin, and the tip appeared to be loosely adherent to the peritoneum lining the anterior abdominal wall. It contained fluid faecal matter and the wall towards/
towards the tip was a trifle congested. The Kidneys were each slightly reduced in size and presented a number of irregular depressions. On section, it was found that the depth of the renal parenchyma was definitely decreased, both cortex and medulla sharing in the reduction. The capsule stripped comparatively easily, exposing in each case an irregular pitted surface. The kidneys were clearly of the arterio-sclerotic type. The renal pelves, ureters and bladder were healthy. Permission to examine the cranium was not obtained.

MICROSCOPICAL EXAMINATION.

The heart has been preserved for further pathological investigation. A block of tissue has, however, been cut from the lateral wall of the left ventricle about an inch from the apex in its entire thickness. In one area in the middle of the section the heart muscle fibres have undergone a marked degree of atrophy, and many have disappeared altogether. This has been accompanied by a proportional overgrowth of stroma, and here and there fairly broad bands of fibrous tissue have developed. The small vessels are the seat of definite arterio-sclerotic thickening.

THE PATHOLOGICAL DIAGNOSIS WAS:

Arterio-sclerosis.

Chronic interstitial myocarditis with
Sclerosis of the Coronary arteries.
Cardiac dilatation.
Arterio-sclerotic kidneys.
Hypostatic congestion of the lungs.

SUMMARY of CASE 10 (Mrs. M. C.).

A female aged 84. Suffered from flatulence for 21 years and repeated fainting attacks over a period of twenty months. In December 1927...
CASE 10.

In December 1927 it was observed for the first time that the heart rate was 36-38 to the minute, occasionally doubling in rate. The rate was maintained again until March 1930 when it was noted that its usual rate was in the neighbourhood of 25. A possible explanation of the phenomena just noted is that up to this time she suffered from an intermittent 2 to 1 or 3 to 1 block, all beats being conducted when the faster rates were observed. In April 1930, she had the first of a long series of Stokes-Adams attacks, which were to continue intermittently until her death on 13th January 1932. In the intervals between attacks the heart rate averaged 36 per minute. It would seem justifiable to assume that the first seizure of 5th April 1930 marked the onset of complete heart block. In other words the defect in conduction had by April 5th reached such a pitch that the circulation was dependent upon an idioventricular rhythm. From that time until death she had at least 352 typical Stokes-Adams seizures. These occurred in groups over a period of a few days at a time. In the intervals she remained comparatively well and not greatly handicapped in her activities in spite of the slow heart action. A precipitating cause for the seizures was suggested by the discovery of localised abdominal tumour, at one time thought on clinical grounds to be a hydronephrosis. When enlarged, tense and firm it heralded the onset of the heart attacks. When the swelling was impalpable no attacks occurred. Post mortem evidence suggested that the abdominal distension was of the nature of a subacute obstruction produced by a Meckel's diverticulum. No hydronephrosis was found. She had only one Stokes-Adams seizure while under observation in the hospital, though a fortnight previously she suffered from 114 attacks in a single 24 hour period. The seizure in hospital was particularly severe. It was heralded by peroxysmal ventricular tachycardia. Consciousness was lost with the onset of ventricular fibrillation. This rhythm continued for 1½ minutes. The ventricles then stood still. The pulse was absent for over 3 minutes/
minutes and the heart's action was apparently restored by an intracardine injection of adrenaline. The heart resumed beating at a rate of 68 per minute. Consciousness was not regained after the attack for 24 hours. It is possible that a similar arrhythmia was present in all the severe seizures which were of unusually frequent occurrence. During succeeding weeks she gradually lost strength had no further attacks and the heart-block remained complete and was associated with auricular fibrillation. The heart was a trifle enlarged. The blood pressure was 170 systolic and 80 diastolic. The blood Wassermann reaction was negative. As the fatal termination approached she became more and more confused, suffered from delusions, and finally developed hypostatic congestion of both lungs. The rise in temperature during the last day of life was accompanied by an acceleration of the ventricular rate.

Post mortem examination revealed a generalised arterio sclerosis affecting particularly the heart and the kidneys. The muscle of the ventricles was largely replaced by scattered bands of fibrous tissue. The finer branches of the coronary arteries were grossly thickened. A Meckel's diverticulum was discovered at the post mortem examination.
CASE 11.

Mrs. Jessie D.  Aged 72 (April 1931).

Occupation, housewife.

Admitted Royal Infirmary 20th. April, 1931 and discharged 29th. May, 1931. Died at home 26th. October, 1931. (This patient was seen at the Dietetic Out-Patient Department of the hospital and I am much indebted to Professor Murray Lyon who arranged for her admission to Ward 21 where I was given the opportunity of investigating her condition.)

COMPLAINT.

Shortness of breath for two years.

"Faint turns" one year ago.

Swelling of the feet for two months.

In the spring of 1929 the patient's health began to fail. Her family noticed that with even slight exertion, fatigue readily set in and that she was able to do less and less in the house. Previous to this she had always been an active person, and it was noticed that fatigue and breathlessness were constantly handicapping her activities. About the same time she began to lose weight and suffered from a boil in the left groin. This healed quickly and though her appetite was good she continued to decline in strength and weight.

In April, 1930 she experienced two severe fainting attacks. Her memory is poor and she is neither able to recall the exact dates, nor is she able to give a clear account of the exact sequence of events at this time. The attacks were separated by an interval of a day or two, and after each she was unconscious for over an hour. Both attacks came on suddenly, the first while sitting in a chair and the second in bed when convalescing after the first one. She was found lying on the floor on the first occasion. Her doctor, who saw her some hours after this attack made no remark to her about the rate of her/
CASE 11.

her pulse, but told her family that her heart was affected.

She had no further attacks, but on again commencing to go about the house in September 1930 she was even more easily breathless. The slightest exertion was difficult for her and she says that, since she had the "faints", life has been a burden to her.

In February 1931 she began to suffer from swelling of the feet. Though she was only sitting at the fire for a few hours at a time the ankles were always puffy and swollen by the time she returned to bed. The swelling as a rule had disappeared by the morning. Apparently increased during the past few months, the amount of swelling present in the evenings is much the same as when it was first noticed.

Since March 1930 her weight has fallen by two stones or more. On account of this she was taken to see her doctor, who then discovered a large amount of sugar in her urine.

PREVIOUS ILLNESSES.

She has always been a healthy active woman until quite recently. She has had the usual illnesses of childhood. She has not suffered from diphtheria or rheumatic fever. At the age of 68 she suffered from sciatica in the left leg and was confined to bed for some weeks.

In May 1930 her sight began to fail and her doctor told her she had cataract. Diabetes was diagnosed a month ago.

FAMILY HISTORY.

Her Mother died of dropsy many years ago. Her Father died of cause unknown. One sister is alive at the age of 60 but is an invalid from paralysis of both legs, which developed in infancy. The patient married at the age of 22 and has had eleven children, three of whom died when/
when babies. The remainder are alive and well. She has had no miscarriages.

SOCIAL HISTORY.

The patient lives very quietly at home. Her daughter keeps house for her. She goes out of doors very seldom. She does not smoke or take alcohol.

STATE ON EXAMINATION.

The patient is an elderly woman who looks some years older than her age. She is propped up in bed with three pillows and at rest is quite comfortable but a little orthopnoeic. The face is haggard and worn, the hair scanty and dry and the skin furrowed and wrinkled. There is no pallor, but a slight tinge of cyanosis in the malar regions and about the lips. She has a sallow yellow tint. The eyebrows are thinned in their outer halves. She is 5 ft. 1 inch in height and now 9 stone 9 lbs. in weight (having lost approximately 2 stones in the past year).

CIRCULATORY SYSTEM.

The pulse is 40 per minute, regular in rate and force. The tension is raised and the pulse is full and bounding. The pulse wave rises abruptly, is well sustained and falls away quickly. The arterial wall is thickened but not tortuous. The Blood Pressure is 200 systolic and 115 diastolic. There is no capillary pulsation nor clubbing of the fingers. The fingers are short and "stumpy". The skin of the hand and forearm is dry, wrinkled and inelastic.

The Chest and Neck. The thyroid gland is not enlarged. In the supra-clavicular regions there is some slight excess of fat, the normal hollowing being obliterated. The veins in the neck are a little more prominent than usual, and a regular succession of venous waves at a rate of approximately 85 can be seen over the jugular bulb and extending upwards into the neck along the venous channels. The carotid beat is more readily seen as a slow and powerful pulsation, particularly when the head is inclined to one side.

The/
CASE 11.

The praecordium is well covered and of normal form. No abnormal pulsation is visible. The apex beat is not seen. On palpation the apical thrust is found in the 5th. left interspace. The lower and outermost point of pulsation lies less than a half inch outside the mid-clavicular line and impinges behind the 6th. rib. Percussion confirms that the heart is enlarged. The area of supra-cardiac dullness in the second interspace is not increased. On auscultation, no murmurs are heard. The sounds are pure in character, but the first mitral has a booming character and the second aortic is accentuated and occasionally reduplicated. The sounds both first and second vary in character from time to time particularly in the mitral area, but no murmur can be detected. The changing character in the sounds give rise to a suspicion that it might be possible to hear an auricular sound during ventricular diastole. On one occasion it was thought that a regular muffled sound at short intervals could be heard, but repeated and prolonged auscultation at various sites on many different occasions failed to confirm this impression.

ALIMENTARY SYSTEM.

She has been very constipated until about three weeks ago when she had an attack of diarrhoea. This has now subsided and she is again having difficulty in regulating the bowels. Flatulence has troubled her during the past year or two. She complains of feeling her clothes too tight for her after meals and this is accompanied by a feeling of tightness in the chest, sometimes extending up to the right shoulder.

The mouth appears healthy. She has no teeth. The tongue is coated, has a raw appearance and is distinctly dry. The throat is healthy. The abdomen is well covered and pendulous in the lower half. The flanks bulge on either side. The abdominal wall moves freely. There is no tenderness detected. The liver and spleen are not enlarged. The kidneys are not felt. There is no ascites.

RESPIRATORY SYSTEM.
CASE 11.

RESPIRATORY SYSTEM.

She has no cough but is easily made breathless. The chest is of normal form, well covered, and expansion is equal on the two sides. Vocal fremitus is unaltered. The percussion note is resonant. The breath sounds are vesicular.

GENITO-URINARY SYSTEM.

She has always drunk a lot of water and has been passing more than formerly during the past six months. She has no desire for sweet cakes, or sweets but craves for fruit. Her sight has been failing and for two and a half years she has been gradually losing weight. She has had no skin cimitation or pruritus, but suffered from a boil 2½ years ago.

ANALYSIS OF URINE.

The daily output ranges from 1000 to 2000 c.c.s. It is acid in reaction, with a specific gravity of 1020, contains sugar, a trace of acetone, and a small amount of albumen. Microscopic examination reveals hyaline and granular casts.

NERVOUS SYSTEM.

Her memory is poor. She has tended to be irritable and rather unstable emotionally during the past few months. Fits of depression occasionally occur. At other times she is unduly excitable. The cranial nerves are perfectly normal. The pupils are equal and react to light and accommodation. She has some cataractous changes in the lens in both eyes and her sight is defective. The tendon reflexes are all present and equal. There is no paralysis and no disorder of sensation.

LOCOMOTOR SYSTEM.

The movements at the joints are all a little restricted. She has some pain occasionally in the left leg. There is a distinct degree of oedema around the feet and ankles. Pitting on/
CASE 11.

on pressure is easily elicited. The fingers are rather short and "stumpy". The superficial veins of the arms are a trifle more prominent than usual.

BACTERIOLOGICAL REPORT.

The Blood Wassermann reaction is negative.

BIOCHEMICAL REPORT.

The Blood Urea nitrogen is 17 mgms% and the Non-protein nitrogen is 35 mgms%.

COURSE IN HOSPITAL.

The patient was admitted to hospital and, with dietetic treatment combined with two small doses of insulin, the urine was rendered free of sugar and acetone. With rest in bed she lost all evidence of oedema and her weight declined. She was discharged from hospital at the end of a month considerably improved in health, on a suitable diet of 2010 calories with a single morning dose of 5 units of insulin.

THE CLINICAL DIAGNOSIS WAS.

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Arterio-sclerosis (associated with diabetes mellitus and slight myxoedema). Chronic nephritis.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Structural</td>
<td>Cardiac hypertrophy.</td>
</tr>
<tr>
<td>Rhythm</td>
<td>Complete heart-block.</td>
</tr>
<tr>
<td>Functional</td>
<td>Grade 3.</td>
</tr>
</tbody>
</table>

SUMMARY OF CASE 11. (Mrs. J.D.)/
CASE 11.

SUMMARY OF CASE 11. (Mrs. J.D.).

A female aged 72, suffered from shortness of breath with increasing fatigue for 2 years. Twelve months previous to admission to hospital had two severe fainting attacks with loss of consciousness for over an hour on each occasion. Since then breathlessness has got worse and has greatly limited her activities. Two months ago began to suffer from oedema of the feet. Diabetes diagnosed one month before admission to hospital. The heart was enlarged. No valvular disease. Heart rate in the neighbourhood of 40. The Blood Pressure was 200 systolic and 115 diastolic. Vessels thickened. Urine contained albumen, sugar at times and a trace of acetone. With rest in bed and suitable dietetic treatment she made a good but limited response to treatment. She has not been able to attend at the hospital since her discharge on account of difficulty in going about. She has not been seen since August 1931. She died
CASE 12.

James D. Male age 50 (1931)

Occupation Foundry labourer.

Admitted Royal Infirmary 21st. March 1931 and discharged 17th April 1931. (I am indebted to Professor Murray Lyon who granted me all facilities to make observations on this patient in Ward 21 of the Royal Infirmary.)

COMPLAINT.

Exhausted and breathlessness on exertion for six years.

The patient was regularly employed at his work until about six years ago when he began to notice that at night he was much more exhausted than had been customary for him. At first he paid little attention to this symptom and was inclined to attribute it to the unusually heavy work he had been doing. A few weeks later his activities began to be checked by breathlessness and when he got home at night he was so fatigued that he used often to retire to bed as soon as he had his evening meal. His doctor, who saw him at this time advised him to rest and after a month's quiet, he was able to return to his work feeling much stronger. Every year since then he has been forced on account of similar symptoms, to give up his work for some weeks. With successive years the duration of his absence from work has gradually increased. Last year, he was at home for six months and when he returned to the foundry, it was noticeable that his improvement had not been so pronounced as in former years. There were certain tasks he was quite unable to under-take and in spite of endeav-ouring to save himself as much as possible he again had to give up work in January 1931. after only two months of regular duties. He was by this time made breathless merely by walking to his work. He consulted his doctor who told him that his heart was strained and that he was anaemic. Acting on the advice he received he went to the slaughter house each week and drank half a pint of fresh blood. He/
He much preferred this to eating liver and he thinks that the large amount of blood which he consumed improved his health considerably. In February 1931, however he had a transient fainting attack in which he became lightheaded, staggered but did not lose consciousness, not fall to the ground and was perfectly well again in a few seconds. He volunteers the remark that it seemed to him as though his heart had stopped beating. He has once or twice in the past - even five years ago - had a similar sensation but the actual number and dates of these have been forgotten and he has not paid much attention to them. Within the last month, in spite of long hours in bed, the sense of exhaustion and the discomfort due to the breathlessness have both been decidedly worse. He has only been able to go about the house with difficulty. He has had no palpitation, no pain of any kind and has never suffered from swelling of the feet. Less than a week ago he says that he was seized with a sudden "spasm" in which he felt giddy, could not get his breath and lost his voice. In a few seconds the discomfort passed off. On account of the increasing breathlessness and weakness his doctor sent him to the Royal Infirmary. He was admitted to the ward a few days later.

PREVIOUS ILLNESSES.

Thirty two years ago he was rejected for admission to a Friendly Society on account of "a peculiar murmur at my heart". The doctor who examined him told him that, though he had no symptoms then, he might well suffer from heart disease at a later date.

He had an operation for acute appendicitis in Leith Hospital 30 years ago.

As a young man he had an attack of pleurisy and a second attack 7 years ago. No fluid was removed from the chest on either occasion.

He has never suffered from any of the rheumatic manifestations and he has had no diphtheria. He has never suffered from any form of venereal disease.

FAMILY HISTORY.

His father died at age 48 after suffering from a number of years of some form of heart disease.
disease.

His mother died some years later of syphilis.

One brother is alive and well. One brother died of "decline" at age 7.

Two sisters are alive and well. One sister died of "swollen glands" - actual cause of death is not known to him. Two aunts, his father's sisters, died of heart disease, each at age 60. One uncle is troubled with heart disease. He is alive but incapacitated, at age 65.

His family consisted of three daughters. One daughter was killed in a street accident. Another suffered from meningitis as a child and has never really been strong since. She occasionally suffers from fits at long intervals. The remaining girl is alive and healthy. His wife has had one miscarriage.

SOCIAL HISTORY.

The patient has always worked as a foundry labourer. For the past six years he has been a teetotaller, though formerly his consumption was at times excessive. He has not smoked for five years.

STATE ON EXAMINATION

The patient looks a healthy man perhaps ten years younger than his age. His colour is good and he lies comfortably in bed in no distress. There is no cyanosis and no pallor. The breathing is effortless. He is 5 feet 3 inches in height and weighs 8 stone 2 lbs.

CIRCULATORY SYSTEM

The rate of the pulse is 50 per minute. It is regular in time and force. The vessel wall is just palpable but not unduly thickened. The artery is not tortuous. The tension is raised. The pulse wave is of average volume, well sustained and falls away gradually. There is no capillary pulsation. The fingers are clubbed to a slight extent, the finger tip being a trifle enlarged and the nail curling over the end of the finger. Dimpling of the skin at the insertion of the nail is/
is produced by a slight degree of pressure exerted on the nail. The Blood Pressure is 192 systolic and 120 diastolic.

The neck is short and thin. The thyroid gland is not enlarged. The veins in the neck are not distended but a regular succession of small waves can be seen over the jugular bulb. At regular intervals a prominent carotid beat occurs.

The praecordium is well covered. No abnormal pulsations are detected over the praecordium. There is no pulsation is the supra sternal notch. Pulsation can be seen in the epigastrium but it is not prominent.

On inspection the apex beat is not visible and on palpation it is located with difficulty. There is no thrill at the base or apex. A diffuse shock is imparted to the fingers over a wide area but a localised thrust can not be detected. The lower and outermost point of pulsation is $4\frac{1}{4}$ inches from the midsternal line. The mid-clavicular line is 4 inches to the left.

On percussion the left border of the heart in the 5th interspace lies between $4\frac{1}{4}$ and $4\frac{1}{2}$ inches to the left of the mid-line. The right border cannot be percussed. The heart is enlarged.

Auscultation reveals that at the mitral area there is a soft blowing systolic murmur which accompanies the latter part of the first heart sound. It is not conducted to the axilla and can be heard with increasing difficulty towards the sternum. It is not heard at the base. The murmur is occasionally absent - in fact at the apex it varies from beat to beat. Sometimes no murmur is heard. The first sound then has a booming accentuated quality. At other times the first sound is reduplicated. In spite of these striking variations in the character of the first mitral sound, yet auricular systoles in the period of ventricular diastole, give rise in themselves to no appreciable sound. The second mitral sound is pure.

Over the base the sounds are faint and distant. The first is heard with difficulty except on certain occasions, when, for a single cycle, it becomes a trifle louder. The second sound is closed and pure. It is not accentuated.

ALIMENTARY SYSTEM.

He has suffered from heart burn occasionally during the past few years. The abdomen is well covered.
covered and moves freely with respiration. There is a former healed scar of an appendix operation in the right iliac fossa. On palpation no tenderness is found. The liver and spleen are not enlarged. The kidneys are not felt and there is no ascites.

RESPIRATORY SYSTEM

He has had no cough and no sputum. The thorax is well covered and of normal form. Expansion is equal on the two sides. He has no difficulty in taking a full deep breath. Vocal fremitus is easily felt and of similar intensity on the two sides. The percussion note is resonant throughout. The breath sounds are vesicular and there are no accompaniments.

NERVOUS SYSTEM

His memory is good, and he is an intelligent man. Headaches are not complained of. The cranial nerves are healthy. The pupils are equal and react briskly to light and accommodation. The arm jerks are brisk and equal on the two sides. The knee and ankle jerks are easily elicited. They are equal on the two sides. The plantar reflex yields a flexor response. The muscular power is good. There is no weakness or paralysis. Sensation is perfectly normal throughout.

URINARY SYSTEM

He has no urinary symptoms. He does not rise at night to micturate.

ANALYSIS OF URINE

The average daily output amounts to about 1800 cc's. The reaction is acid and the specific gravity is 1008. It does not contain albumen, sugar, acetone or other abnormal constituents. Microscopical examination was negative.

LOCOMOTOR SYSTEM

The muscles are well developed. Joint movements/
movement are free. There is no oedema of the extremities.

BACTERIOLOGICAL REPORT (23-3-31)

Wassermann reaction is negative.

COURSE IN HOSPITAL.

After a day or two's rest in bed the patient felt very much better and during the remainder of his stay in hospital certain investigations were carried out. He had nothing in the nature of a Stokes-Adams seizure and complained of no discomfort at any time.

THE CLINICAL DIAGNOSIS WAS:

**Etiological** Unknown.
**Structural** Cardiac hypertrophy. Mitral insufficiency.
**Rhythm** Complete Auriculo-Ventricular dissociation with interference.
**Functional** Grade 2a.

SUMMARY OF CASE 12. (J.D.)

A male aged 50, accustomed to do heavy work until six years ago. At the age of 16, he was rejected for admission to a Friendly Society on account of a peculiar cardiac murmur then detected. He had no symptoms until 44 years of age. Since then has suffered from breathlessness and undue fatigue on exertion - greatly relieved by periodic spells of rest. He has had one or two giddy attacks but nothing to suggest the presence of the Stokes-Adams syndrome. On admission to hospital the pulse was 56 per minute and electrocardiograms revealed the presence of complete heart block. The heart was a little enlarged. Arterio-sclerotic changes were no more advanced than might be expected for his age. The blood pressure was 192 systolic and 120 diastolic. The Wassermann reaction was negative. The urine was of low specific gravity but there was no other abnormality/
abnormality detected. The etiology of the cardiac condition remains obscure.

Electrocardiograms revealed dissociated auricular and ventricular rhythms. The block, which could be temporarily relieved by adrenalin, is therefore not entirely of organic origin. It is of the form described by Mobitz as "dissociation with interference". He has given up his work since his discharge from hospital and has kept in very good health up to the present time (October 1932). He is able to go about, is breathless on severe exertion but is not so easily exhausted as before.
CASE 13.

Mrs. H. Age 57 (1932) Female.

Occupation, housewife and charwoman.

Admitted to the Royal Infirmary 11th. November 1932, and discharged 2nd. December 1932. (This patient came under observation in the Dietetic Out-patient Department and I am much indebted to Professor Murray Lyon who gave me the opportunity of having her under observation in Ward 21 Royal Infirmary.)

COMPLAINT:

Pains in the right knee and stiffness - 7 years.

"Weak turns" 7 years.

Attacks of giddiness. 7 years.

The patient states that until 1925 she was in very good health and able to undertake all her usual physical activities. About that time she began to be troubled with pains, accompanied by stiffness in the right knee particularly on rising from bed in the morning. She consulted her doctor, who told her that she had synovitis. The knee became swollen and intensely painful with the result that she was confined to bed for five weeks, at the end of which time the condition had subsided sufficiently to allow her to go about. She had not returned to her work for more than a week or two when she began to be troubled with what she calls "weak turns". The first of these she can remember quite distinctly. She was standing in her living-room one afternoon, after her return from work, when quite unexpectedly and without warning she felt her strength gradually growing less in the course of a few minutes. She was so distressed by this symptom that she was forced to lie down. She felt powerless, helpless and quite exhausted. An hour or two/
two passed before she was able to get up from bed and attend to her duties. She did not become unconscious, but felt all the time in a dreamy state. Similar attacks have occurred at frequent intervals. Up to the time of her admission to hospital she might experience two or three in a fortnight at quite unexpected and irregular times, and then, for some reason unknown to her, she might have a period of freedom lasting for two or three months. She states emphatically that she has never lost consciousness in an attack, nor has she ever fallen and hurt herself. At the time of the peculiar weak feeling she does not experience a sensation of giddiness or faintness. To her the alarming aspect of the attack is the absolute loss of strength, which comes on suddenly and from which she recovers as abruptly, after a varying interval of perhaps ten or twenty minutes or even two and a half hours. Attacks generally occur in bed and she has wakened through the night feeling utterly helpless.

For about the same period of time she has also suffered from attacks of giddiness. These are not directly associated with the "weak turns". They do not occur at the same time, and each lasts for only two to three minutes, occurring perhaps twice a month or thereabouts. In the giddy attacks, of which she has no warning, her gait becomes unsteady, and more than once, she has been glad to hold on to nearby railings or other support, in order to steady herself and prevent herself falling. She has never lost consciousness in one of these attacks, and though she has felt faint and giddy she has never fallen. Sudden giddiness of this nature has never occurred while she has been lying in bed.

Since 1925, she has also been a trifle breathless on exertion, particularly if climbing stairs or on hurrying along the street. This symptom varies a good deal from week to week. Some days she feels very much better and has no breathlessness; other days a like amount of work is a little exhausting to her. She has never wakened short of breath through the night; nor/
nor is she short of breath during a "weak turn" or giddy attack.
She has never had swelling of the feet, though in former years after a heavy day's work the right ankle has been painful and a trifle puffy at night. She has had no cough and no other complaints.

PREVIOUS ILLNESSES:
She has not suffered from diphtheria nor scarlet fever.
At the age of 18 she had a severe attack of rheumatic fever and was confined to bed for over 4 months, during the first three of which the joints were so swollen and painful that she was unable to feed herself. One year elapsed before she was permitted to return to her occupation.
In the spring of 1925 (age 50), about three months before the onset of her present symptoms she had an attack of tonsillitis, which was labelled "an influenza throat". She was in bed for 10 days at this time.
These are the only illnesses known to her.

FAMILY HISTORY:
Father died of "dropsy" about age 60. Mother died a few months after patient's birth - cause of death unknown. Her husband died 10 years ago of lobar pneumonia.
The patient has had three full-time children and no miscarriages. Of her three sons, one was killed on active service in France. The two remaining are in good health at the present time and have had no serious illnesses.

SOCIAL HISTORY:
The patient is a keen intelligent woman accustomed all her life to engage in hard physical work. She was originally employed as a/
CASE 13.

a laundry-maid (at the time of the attack of rheumatic fever) after which she married. Since the death of her husband she has supported herself by working as a charwoman and for the past few years she has been in regular employment as a cleaner, doing heavy work in an Edinburgh Nursing Home. Her occupation involves a good deal of scrubbing and the arthritis of the knee-joint has been a much greater handicap to her than her cardiac symptoms.

She does not smoke, has been a teetotaller all her life, and lives by herself in healthy surroundings.

STATE ON EXAMINATION: (12th. Nov. 1932.)

The patient is of healthy appearance, has a good colour, looks her age, and lies in bed in no distress. Breathing gives rise to no difficulty. Her weight is 12 st. 7½ lbs. and her height is 5 ft. 4½". The correct weight for her age and height is 10 st. 6 lbs.

CIRCULATORY SYSTEM:

The pulse is regular in rate, 40 per minute, and the beats are of equal force. The pulse wave is full and well sustained. The artery wall is not thickened. There is no tortuosity of the vessel. The blood pressure is 158 systolic and 70 diastolic. There is no capillary pulsation and the finger tips are not clubbed.

The chest and neck are of normal form. The thorax is well covered and no deformity is detected. The breasts are pendulous. The veins in the neck are not distended and pulsation in the region of the jugular bulb is not detected. The thyroid gland is not enlarged.

There is no pulsation seen over the praecordium, and none in the supra-sternal notch. The apex beat is not visible.

On palpation the apex is found in the fifth interspace. It is a slow, steady, regular thrust localised to an area about the size of a shilling. To the palpating hand the pulsation at the apex at certain times, appears to be stronger/
stronger than others. This variation in strength is independent of respiration and occurs at intervals of about every tenth or twelfth cycle. The apex beat is in the fifth interspace impinging on the upper border of the 6th rib, immediately outside the mid-clavicular line.

The right border of the heart cannot be percussed. The left border, owing to the excess of adipose tissue is percussed with difficulty. The area of supra-cardiac dullness does not appear to be increased.

On auscultation the heart sounds are easily heard. Over the base the second sound is much the louder, though the first is heard distinctly. Both sounds are of the usual quality. No murmurs are heard at the base, but the second aortic sound is definitely accentuated. The sounds are of constant character from cycle to cycle. At the apex the sounds are also clearly heard, but each varies in character from time to time. The first is much the louder of the two, and has a booming quality. The second sound is as a rule short and thudding. From time to time, about every tenth cycle, the first mitral sound is increased in intensity. This is accompanied by a more powerful apical thrust. Three or four cycles later, the second mitral sound is occasionally reduplicated or louder and a trifle longer than usual. Distinct and separate sounds attributable to auricular systoles cannot be heard over the praecordium.

ALIMENTARY SYSTEM:

She has had no digestive disturbance. The tongue is clear and moist. The teeth are artificial, the gums healthy. The tonsils are small. The abdomen is well-covered and moves freely with respiration. No tenderness is detected and the spleen and kidneys are not felt. The liver is not enlarged. Its lower edge can be felt under the costal margin in the mid-clavicular line.

RESPIRATORY/
CASE 13.

RESPIRATORY SYSTEM.

The chest is well covered and moves freely with respiration. Expansion is equal on the two sides. The percussion note is resonant throughout. On auscultation the heart sounds are heard distinctly and no accompaniments are detected. The breathing is of the vesicular type.

NERVOUS AND LOCOMOTORY SYSTEMS.

The patient is inclined to be a little nervous about herself, and dreads the occurrence of further "weak turns". She is inclined to be introspective and anxious. She is an intelligent woman, better educated than many, and looks forward to returning to her work. Her memory is good.

The cranial nerves are healthy. The pupils are equal and react briskly to light and accommodation. They are round and equal in size. The tendon reflexes are equal and present on the two sides. The plantar reflex yields a flexor response in each foot. Sensation is perfectly normal throughout.

The muscles are well developed. The limbs are strong. There is no muscular weakness or paralysis.

The right knee joint is a little swollen and the tissues round about are somewhat thickened. On moving the joint surfaces, marked creaking and grating is felt. Full flexion at the knee produces pain in the region of the joint. The joints elsewhere are healthy.

There is no oedema of the extremities or sacrum.

GENITO-URINARY SYSTEM.

She has no urinary symptoms. The menopause occurred ten years ago. The urinary output averages 1400 c.cs. The specific gravity is 1015. The reaction is acid. No abnormal constituents/
CASE 13.

constituents are found in the urine.

BACTERIOLOGICAL REPORT. (1:12:32.)

The blood Wassermann Test yields a weak positive reaction. (Ten days after a provocative dose of '914' the test was repeated and found to be negative).

RADIOLOGICAL REPORT. (15:11:32.)

The transverse diameter of the heart in the antero-posterior view is a trifle increased in size. The aorta is of normal size. The posterior mediastrium is clear. Screen examination revealed dissociated activity of auricles and ventricles.

COURSE IN HOSPITAL.

This patient, when attending the Dietetic Department on account of obesity was observed to have an unusually slow pulse. Electrocardiograms demonstrated the presence of complete block and she was admitted to Ward 21 for observation. She had no "weak turns" or fainting attacks while a patient in the hospital, and in the three months which have elapsed since then she has had only one mild attack of loss of power, in spite of no particular treatment.

THE CLINICAL DIAGNOSIS WAS

Etiology  ? Rheumatic heart disease.
           Obesity.
Structural Cardiac hypertrophy.
Rhythm    Complete heart-block.
Functional Grade 1.

SUMMARY/
SUMMARY OF CASE 13 (Mrs. H.)

At the age of 18, this patient suffered from a severe attack of rheumatic fever which incapacitated her for 12 months. At the age of 50, she suffered from an attack of tonsillitis followed some months later by arthritis of the right knee. She was in bed for five weeks and after returning to her work began to suffer from "weak turns", giddy attacks and slight dyspnoea on strenuous exertion.

The heart was a trifle enlarged with no evidence of valvular disease. The blood pressure was 154/70. The pulse rate was generally in the neighbourhood of 40 per minute. She had no fainting attacks or "weak turns" while under observation. The knee joint was her main handicap. On a diet of 1000 calories she lost 9 lbs. in weight in three weeks and, on her discharge from hospital she was able to lead a reasonably active life. Dyspnoea was only induced by prolonged exertion of a heavy nature. Her incapacity was slight.
CASE 14.

Mrs. M.D. Age 63 (1932). Female.

Occupation, housewife.

Admitted to the Royal Infirmary on 8th. November, 1930 and discharged 4th. December, 1930 was again admitted 25th. June, 1931 and discharged 18th. July, 1931. A final period of hospital treatment extended from 23rd. April, 1932 until 15th. June, 1932. (Professor Murray Lyon kindly drew my attention to this patient in the Dietetic Department and arranged for her admission to his Ward where I was kindly given the opportunity of having her under observation).

COMPLAINT.

Breathlessness on exertion, 7 years.

Flatulence, 3 years.

"Faint turns" intermittently 1 year.

A sense of discomfort on exertion has been experienced by this patient for at least ten years but she did not suffer from any undue amount of breathlessness until approximately seven years ago (1925). The discomfort in getting about she attributed to the fact that her weight has gradually tended to increase for the past twenty-five years, but that she experienced no inconvenience until 7 years ago when breathlessness on exertion very gradually began to be a check to her activities in the house. It has steadily increased in amount and has been accompanied by a sense of undue fatigue. This has so handicapped her that within the last few years she has been able to do less and less, with the result that she has become even stouter. She has had to give up practically all her household work spends a great deal of her time resting, and is unfit to carry on any physical work. Within the last six weeks even in conversation she has had to pause in order to recover her breath.

For three years (since 1929) she has been troubled with flatulence. This amounts to a sense of discomfort in the epigastrium after food. She feels her clothes too tight for her even/
Case 14.

Even after small meals and has been in the habit of taking baking-soda when the discomfort was at its height. Within the last year, the flatulence has been definitely worse. She describes it as a sensation of fullness over the chest accompanied by a sinking feeling. She is relieved by eructations. This peculiar sensation which she has some difficulty in describing has given her a great deal of distress. It is accompanied by such swelling of the abdomen that she has often to lie down and loosen her clothes until "the wind breaks". This as a rule gives her relief. It has not given rise to palpitation, from which she has never suffered, but she has noticed that it appears to make her perspire freely. Much anxiety is caused until she gets relief.

One year ago (June 1931) while endeavouring to perform some of the lighter household duties, she was suddenly seized by a feeling of faintness and giddiness. She had never experienced a similar sensation. During the succeeding three weeks similar attacks occurred at short intervals and became so incapacitating that she was forced to retire to bed. Sometimes attacks came in rapid succession, three or four in the course of ten minutes, and each lasting a few seconds during which time she was observed to become very pale and then blue about the mouth and ears. She never actually lost consciousness, but experienced a sensation of great weakness, complete loss of power and utter helplessness. In each instance she recovered very rapidly, but the frequency with which one attack sometimes followed another, and the fact that she could do nothing to prevent them made her very alarmed and extremely nervous. She was admitted to the Infirmary at this time and electrocardiographic investigations proved that the attacks were mild examples of the Stokes-Adams syndrome. After three weeks in hospital the attacks ceased and she was discharged and able to go about in a reasonable amount of comfort provided she did not over-exert herself.

She was readmitted to the Ward in April 1932 on account of a return of the breathlessness.
CASE 14.

on exertion, frequent headaches, and great exhaustion.

PREVIOUS ILLNESSES.

In childhood she suffered from measles, chickenpox and whooping cough. She has never had diphtheria, rheumatic fever, rheumatism or chorea.

About the age of thirty a curettage was performed on account of dysmenorrhoea.

The slow pulse was first recognised in July, 1930 when she came under observation in the Dietetic Department. The rate then was 48 per minute.

FAMILY HISTORY.

Her father died in senility at age 77. Her mother died - age unknown - of heart disease. She was very obese. The patient has had no children nor miscarriages. Her husband is alive and well.

SOCIAL HISTORY.

The patient was married in 1914 at the age of 46. She has a good home and lives in congenial surroundings. She does not smoke, is a teetotaller, but drinks strong tea, perhaps six or eight cups each day.

STATE ON EXAMINATION. (23rd. April, 1932).

The patient is a stout elderly woman. Lying at rest in bed the breathing is comfortable and she is in no distress. There is a faint degree of cyanosis about the lips and finger tips. There is no undue pallor. She is 5 ft. 1½ inches in height and weighs at present 10 st. 4 lbs. In July, 1930, when she first came under observation her weight was 14 st. 9 lbs. Her correct weight for her age and height is 9 st. 12 lbs.

CIRCULATORY SYSTEM./
CASE 14.

CIRCULATORY SYSTEM.

The Pulse. is slow in rate 28 per minute and regular in time and force. The volume is somewhat reduced and the artery is palpated with difficulty. The wave is of poor quality being thin and of wiry consistency. The artery is slightly thickened, hard but not tortuous. The tension is raised. The Blood Pressure is 300 systolic and 120 diastolic.

Chest and Neck. The neck is short, thick-set and obese. The veins do not appear distended. The thyroid gland is not enlarged. Regular small venous waves can be seen at a rate of approximately 90 per minute over the jugular bulb with the head turned in certain positions. The carotid beat can then also be observed at about one third of this rate.

The shoulders are richly covered with fat and the rib outlines and clavicles are obscure. The thorax is also somewhat obese and the breasts are pendulous. There is no pulsation visible in the supra-sternal notch and the apex beat is not seen. There is no abnormal praecordial pulsation.

The Heart. On palpation the apex is found to be half an inch outside the left mid-clavicular line. The apical impact is a slow regular prolonged and forcible thrust confined to a small area less than the size of a shilling. There is no thrill at the apex over the base. There is no diastolic shock.

On percussion the area of cardiac dullness cannot be defined with accuracy owing to the thickness of the chest wall. In the fifth space the left border appears to be 4½ inches from the mid-sternal line. The right border cannot be percussed. The supra-cardiac dullness is not increased. The heart is slightly hypertrophied.

On auscultation the sounds are distinctly heard at the apex. The first is loud and has a
CASE 14.

a dull thudding character. It is accompanied by a soft blowing systolic murmur, maximum at the apex, constant in character, and heard over an area about the size of a five shilling piece. The second mitral sound is pure in character, but rather louder than might be expected in the presence of such a thick chest wall. At the base the sounds are pure in character. No murmurs are heard. The second aortic sound is a trifle accentuated. No sounds attributable to auricular systole are heard.

ALIMENTARY SYSTEM.

She has suffered from flatulence for three years. The bowels have tended to be irregular and she is accustomed to take a saline purge two or three times a week.

The mouth is healthy. The tonsils are not enlarged. The tongue is clean. The teeth have all been extracted. Artificial dentures are worn. These fit well. The abdomen is well covered, a little pendulous and tends to bulge outwards in each flank. There is no tenderness and no rigidity. The abdominal wall moves freely with respiration. The liver and spleen are not enlarged and there is no ascites.

RESPIRATORY SYSTEM.

She occasionally suffers from colds and has once or twice suffered from a mild attack of bronchitis. At the present time she has no cough or expectoration. She is easily made breathless on exertion.

The thorax is well formed and well clad with adipose tissue. The respiratory movements are free and symmetrical. Vocal fremitus is of average quality throughout. Percussion gives a healthy resonant note in all areas. The breath sounds are of the usual vesicular character. No accompaniments are detected.

THE NERVOUS SYSTEM.

The patient is an intelligent woman whose memory is good. She suffers from headaches when/
when tired and exhausted. She had a succession of "fainting turns" without loss of consciousness in June, 1931 and was admitted to the Royal Infirmary on this account. They have not recurred since then.

She has become very nervous, excitable and emotionally unstable during the past few months. She says that her "nerves" get the better of her. She considers that this has been brought about by the dread of further fainting attacks, and also by the repeated discomfort caused by flatulence. At times she is depressed and worried about her personal welfare.

The cranial nerves are healthy. The pupils are equal and react briskly to light and accommodation. They are perfectly regular in shape. The eye movements are free, brisk and a trifle exaggerated but equal on the two sides. The arm, knee and ankle jerks are easily elicited. The plantar reflex yields a flexor response in each foot.

The muscles are well developed and the limbs are powerful. There is no muscular weakness or paralysis. Sensation is perfectly normal throughout.

GENITO-URINARY SYSTEM.

She has no urinary symptoms. The menopause occurred at the age of 50. Formerly menstruation was perfectly regular of the 28 day type the flow lasting 3 days. She had dysmenorrhoea at the age of 30.

ANALYSIS OF URINE.

The output averages 1500 c.cs. The specific gravity is 1010. There is a trace of albumen present, but no other abnormal constituent. A microscopic examination reveals the presence of many pus cells, a few epithelial cells. There are also many hyaline casts and a few granular casts.

LOCOMOTORY SYSTEM.

The limbs are strong, richly covered in fat.
fat and the joint movements are free. There is no oedema. The superficial veins are not unduly prominent.

BACTERIOLOGICAL REPORT  
(27-11-30).
The Blood Wassermann reaction is negative.

PATHOLOGICAL REPORT  
(23-5-32)
The Urine: Direct films show a few epithelial cells, but no organisms are seen and no growth is obtained on culture.

BIOCHEMICAL REPORTS:  
(27-4-32)
The blood urea nitrogen is 12 mgms%  
Creatinine is 3.3 mgms%  
Calcium is 9.2 mgms%  
The CO₂ combining power is 71 vols%  

(6-5-32) Urea Concentration Test:—  
Fasting specimen contains 0.94 grms% urea.  
After 15 grms, urea by mouth specimen at hourly intervals. (1) 1.54 grms. % urea.  
(2) 2.01 " " "  
(3) 2.23 " " "

COURSE IN HOSPITAL.
This patient was referred to the Dietetic Department of the Royal Infirmary in July 1930. She has been under observation since then as an out-patient, attending regularly at weekly or fortnightly intervals, save for the three occasions when she was admitted to the hospital for fuller investigation and treatment. When first seen her weight was 14st. 9 lbs. and by use of a restricted diet of 1200 calories she lost 33 lbs. in weight in four months. There was not however as much improvement in her cardiac symptoms as might have been anticipated. Flatulence and dyspnoea continued to trouble her. Her pulse rate counted after resting was generally in the neighbourhood of 48 - the highest rate recorded being 66 and the lowest 34. An electrocardiogram recorded on 4th November 1930 revealed the presence/
presence of 2 to 1 heart block with P-R interval 0.180 sec. She was thereafter admitted to the Ward. During this, her first period in hospital (6th November 1930 - 4th December 1930) the degree of block varied between either full conduction of all auricular impulses (Ltt rhythm) or else half conduction (i.e. 2 to 1 block). Certain experimental investigations were made under these conditions.

After her discharge from hospital she continued to attend as an out-patient until June 1931 when she was readmitted on account of repeated syncopal attacks. By this time a further degree of block had developed it being frequently 3 to 1 but generally of the 2 to 1 type. Full conduction (1 to 1) in repeated daily records was never observed. The P.-R intervals were respectively, 0.165 sec. and 0.185 secs. for 3 to 1 and 2 to 1 block. Certain investigations were recorded and an explanation for the fainting turns was discovered by the demonstration of short periods of ventricular asystole by the electrocardiograph. No attacks occurred while ephedrine was taken by mouth.

She again continued to attend as an out-patient after her discharge from hospital on the 18th July 1931, until she was readmitted on 23rd April 1932. By the latter date her weight had fallen to 10 st. 4 lbs. and the pulse rate was 28 per minute. On this occasion electrocardiograms revealed that the block was either complete. 3 to 1, or 3 to 1 accompanied by the phenomenon of ventricular escape. She complained of repeated headaches general weakness and great exhaustion on the least bodily activity. The cardiac response to a number of drugs was tested. Within twelve hours of discontinuing a course of treatment with ephedrine the ventricular rate declined to 7½ beats per minute. This was accompanied by profound weakness, marked frontal headache, great pallor shock and clammy perspiration. At 11 a.m. the heart rate was 16, at 11-55 11 per minute. At 12-35 p.m. the pulse was 7½ per minute and the patient had a Stokes-Adams attack without the heart actually stopping. She had five similar attacks between 1 p.m. and 2 p.m. without fully regaining consciousness in the intervals. A dose of adrenalin (0.5 c c) increased the heart rate from 11 to 23 per minute and ephedrine was recommenced the same day. This appeared to have/
have the effect of fixing the block at 3 to 1. In its absence the ventricular rate fell to an unusual extent with the production of alarming symptoms but no congestive heart failure ever occurred. She continues to take ephedrine and still keeps remarkably well (Nov. 1932) since her discharge from hospital.

THE CLINICAL DIAGNOSIS WAS:

- **Etiological**
  - Obesity
  - Arterio sclerosis

- **Structural**
  - Cardiac hypertrophy
  - Mitral insufficiency

- **Rhythm**
  - Partial heart block
    - (a) 2 to 1 rhythm with shut P R intervals.
    - (b) 3 to 1 rhythm with shut P R intervals
    - (c) Complete heart block.

- **Functional**
  - Grade 2b.
  - Stokes-Adams seizure.

**SUMMARY OF CASE 13. (Mrs M.D.)**

A female aged 63 (1932) has been under observation for 2 years during which time a gradually increasing degree of heart block has developed. She was very obese, suffering from dyspnoea and flatulence, and has a slight degree of cardiac hypertrophy without valvular disease. When first seen the block was of the 2 to 1 type and with the passage of time the defect in conduction has increased. The blood pressure was 300 systolic and 120 diastolic. There were slight arterio sclerotic changes with albuminuria. At one time she had mild Stokes-Adams seizures at short intervals. Later she had fully developed fits in the presence of heart rate of 73 per minute. She responded well to adrenalin and ephedrine and was discharged from hospital able to go about but with a limited range of activities.