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A CLINICAL STUDY OF EIGHT CASES OF HEART-BLOCK

SUBMITTED AS A THESIS
FOR THE DEGREE OF M.D. EDINBURGH.

BY

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JULY 1921
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I. HISTORICAL.

The credit for describing the first case of Heart-block is generally given to Robert Adams of Dublin, who described one such in 1827. (1) Stokes in 1842 (2) gave a more detailed account of some cases, and the syndrome of slow pulse accompanied by epileptiform convulsions has been known since as the Adams-Stokes Syndrome.

It is not so well known that in the year 1702, an Edinburgh Physician, Dr. Thomas Spens, gave a "History of a case in which there took place a remarkable slowness of the pulse." (3)

I make no apology for inserting this description here for it helps one to realise how accurate were the observations made by the old physicians, and also how little we have advanced in a century except in the knowledge of the mechanism of the production of this syndrome.

Therapeutically we can do little more today for our cases of heart-block than Dr. Spens did century before last.

"On the 16th May 1702 about 9 o'clock in the evening, I was sent for to see a T.R., a man in the 54th year of his age, a common labouring mechanic."
After having heard from him some account of his complaints, I was much surprised, upon examining the state of his pulse, to find that it beat only 24 strokes in a minute. These strokes however as far as I could judge, were at perfectly equal intervals, and of the natural strength of the pulse of a man in good health.

He informed me that about 3 o'clock in the afternoon, he had been suddenly taken ill, while standing in the street, that he had fallen to the ground senseless, and that according to the accounts given him by those who were present, he had continued in that state for about 5 minutes.

His face was slightly cut in two different places by the fall, but his head did not seem to be in any way materially injured. From the time of this first attack till I saw him he had been affected with three other fits of a similar nature. These however were attended with some convulsive motions of his limbs and with screaming during the fit.

When I saw him he was somewhat drowsy but perfectly recollected and distinct and his voice was as strong as when he enjoyed a state of perfect health, nor had he at that time any other complaint, besides being uncommonly thirsty, during the former part of the day he was frequently affected with sickness at the stomach.

Upon visiting him on the morning of the 17th. I found
that he had been attacked with several fits during the night and had vomited up his dinner soon after it was taken.

Upon examining his pulse, I found that it beat only 23 strokes in the minute, nor was any change produced upon it by his drinking a tea-cupful of wine and a glassful of whiskey. In the morning of the 18th. I was informed that he had been frequently faint, his pulse beat only 26 strokes to the minute."

During the doctor's visit the patient had a fit. "He apparently recovered in a few seconds but hardly any pulse could be felt for a good many seconds.

On the morning of the 19th. I learned that he had been attacked with frequent fits attended with violent convulsions .................. At three in the afternoon I found that the pulse beat only 10 strokes a minute, though it still continued equally strong and regular as before. He expired on the 20th. A post-mortem revealed nothing."

Galabin in 1875 was the first to describe the actual condition of heart-block remarking, "We have here a heart the auricle of which sometimes contracted twice in the interval between two ventricular pulsations and sometimes singly in the midst of a long pause instead of just before the systole of the ventricle." He showed curves taken from the heart's apex.
Later the work of Chauveau, Wenckebach, His, and Mackenzie advanced our knowledge of the anatomy and pathology of this condition.
Case I. seen Sept. 2nd 1920, a doctor, male, age 57. Complained of shortness of breath on exertion only, worse some days than others. Sometimes even on exertion he did not feel breathless. The breathlessness is worst after a meal and is accompanied by a feeling of discomfort which may come directly after eating or be delayed for some hours. He feels inclined to break wind and when successful feels relieved. He eats slowly and takes his food dry. Exertion in his case is limited always by breathlessness, which is eased as soon as he rests. He can walk on the level 4 miles or more at 3 miles an hour when he feels well.

Associated with the breathlessness at times he has "giddy turns" while walking. "Seems as though the heart stopped for a moment." Palpitation is also associated with breathlessness on exertion as a rule, but it comes on occasionally quite apart from exertion.

Sitting still, sometimes he feels conscious of the more rapid action of his heart for a few minutes. He may pass weeks without this sensation.

He sleeps well at night, requires only two pillows and can sleep on either side. His appetite is good and his bowels are open regularly daily.

His complaint dated back to 1914 when he was overworked. It began by his feeling that he could not do
the hills as he used to. One day he collapsed on the stair in the hospital he was visiting in his professional capacity. He was short of breath then and had some oedema of the ankles.

He has always had a slow pulse. From infancy he was regarded as phenomenal on this account, and was taken by his father to Edinburgh to be examined by a cardiac expert over fifty years ago. His father always chaffed him as being only half-a-man as he had only half the pulse rate. (4)

As far as his previous health is concerned he has had influenza several times. He has never had rheumatic fever nor chorea, nor has he suffered much from tonsilitis.

There is no history of Syphilis. He is a tall well nourished man. On walking into the room he is cyanosed about the ears and lips. After sitting for half-an-hour his colour is good.

The apex beat of the heart is not visible nor palpable. Cardiac dullness extends slightly beyond the nipple line. At the apex of the heart a rough systolic murmur is heard conducted over most of the precordia and heard loudest just inside the nipple.

The second sound is closed and well-struck. At the base of the heart the systolic murmur is heard faintly. The second sound is closed. The rate of the heart as judged by the sounds is about 36 to the minute with an
occasional beat like an extra systole. The radial pulse is 39 to the minute. There is some irregularity seemingly long pauses. During these pauses the heart sounds are heard. The wall of the radial artery is not thickened. The systolic blood-pressure in the right arm is 140 the diastolic 80.

A polygraphic tracing shews a partial heart-block. The auricular rate is 75 to the minute, the ventricular is 34.

The pulse rate rises on exertion. Before climbing a 20 foot staircase the pulse was 17 to the half minute at the top of the stairs it was 22 to the half minute. In 5 minutes time it was 18 to the half-minute.

The staircase was climbed in 20 seconds. A sensation of palpitation was produced by the exertion.

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Case II.

(Copy) Tracing taken 2nd Sept. 1920, Shewing Heart-Block. As 75 to minute Vs. 34 to minute. Radial beats 2, 3, 6, are of equal length, 4 and 5 are longer, most of this lengthening may be accounted for by the quicker rate of the paper roll. That it is not entirely due to this is evidenced by comparison of carotid beats 3, 4, with 5, 6, where it will be seen that the carotid element in the wave is delayed in the latter two, so that it coincides with the auricular wave.

(Copy) Tracing taken 23-7-17 from the same patient Shewing partial heart-block.

As = 100

Vs = 40.
Case 2.

July 18th. 1920.

Patient "O", age 59, male. Complains of being easily tired, physically not mentally. Can walk about 2 miles on the level. On going rapidly up a slope feels tired, legs feel heavy. Has a sense of oppression in the chest and he may feel giddy. If he rises up quickly from bed he feels giddy and faint.

Three months ago he lost consciousness. About 20 months ago he noticed that lying on his left side made him feel uneasy and uncomfortably conscious of the heart action, which was slow, beating about 30 times to the minute.

This uneasiness would last about half an hour almost every evening. Gradually it disappeared until about 8 months ago, when one day after lunch he had occasion to go quickly up the stairs to his office. He felt faint at the top, sank into a chair and lost consciousness for a few seconds. He woke to find brandy being poured down his throat. He felt dazed and uncomfortable, weak and cold, and his vision was blurred. He felt cold for some days after. A week later he had a similar attack in his motor car and that day he had frequent attacks almost hourly, each lasting only a few seconds.

His doctor saw him that afternoon when his pulse was 22 to the minute, and saw him in several attacks and noted that his pulse disappeared just before the attack began.
43 hours later he lost consciousness in bed.
2 weeks later he had another attack.
2 days later the pulse was 66 to the minute.

The last attack over 7 months ago when his pulse had been 66 for four days, was a severe one lasting over one minute and was accompanied by convulsions. He has had no more attacks since then, but has felt giddy and had a slight faint feeling 3 months ago. There is no history of venereal disease.

The Wassermann reaction is negative.
Heart dulness extends to the nipple line. The sounds are faint.

There is no murmur. A polygraph tracing shows heart block with a 3.1 rhythm.

Frequently a ventricular beat comes through at the normal auricular interval. (See Fig.1.) There is no widening of the A-C interval.

These two facts suggest that the conducting tissue of the heart is not so much at fault as that the heart-block may be due to vagal influence.

A hypodermic injection of 1/50 grain Atropine Sulphate was given and the effects observed on his pulse. (See Fig.s 1,2,3,4, ) Within 20 minutes the ventricle was responding more frequently to the auricle and in 60 minutes time the rhythm had become normal at the rate of 60 to the minute. The polygraph tracing shewing normal a.c.v. waves.
For two months the patient was placed on 1/100 gr. of Atropine Sulphate 6 hourly taken by the mouth.

His condition during this time was much improved. He had no syncopal attacks. Tracings taken during this period show the condition of partial block still present. (See Fig.s 5 and 6).

The improvement could therefore not be attributed to the Atropine which was now withheld.

The patient continued in fair health accomplishing much mental work until a month ago when he died suddenly.
Fig. 1. The slower beats are at the rate of 75 to the minute. The auricles are beating at 60 to the minute. Numerous beats of the ventricle are occurring at 33% of the minute. 19 minutes after injection with 1/50 grain of Atropine Sulphate.

Fig. 2. Before injection, a regular 2:1 rhythm. After injection with 1/50 grain of Atropine Sulphate, the auricles are beating at 60 to the minute. The ventricles are beating at 33% of the minute. Numerous beats of the ventricle are occurring at the rate of 75 to the minute.
Fig. 3

35 minutes after injection with Atropine Sulphate 1/50 grain.

Again note that there is no widening of the a-c interval.

In a normal manner at the rate of 75 beats to the minute.

Excerv the auricle and ventricle are acting through to the ventricle, auricular and ventricular beats have not come except for three pauses where the auricular beat has not come.

25 minutes after injection with Atropine Sulphate 1/50 grain.
the same, the rate being 60 to the minute.

A normal tracing showing a. v. waves in the jugulo-carotid. 60 minutes after injection with 1/50 grain Atropin sulphate.

Case 1. 2.

Fig. 4

60 minutes after injection with 1/50 grain Atropin sulphate.

30 minutes later the rhythm was exactly the same, the rate being 60 to the minute.

The auricle and ventricle are beating at the rate of 60 to the minute. The auricle and ventricle are beating at the rate of 60 to the minute.
Case 1. 2.

Fig. 5

Tracing taken 8.9.20. Showing 3:1 rhythm with a normal beat alternating.

Fig. 6

Tracing taken 8.9.20. Showing a 3:1 rhythm.

As. 88.2
Vs. 28.8
Case 3.

Case 3. age 10 years, female.
Admitted hospital 2nd March 1920, on 3rd day of illness.

Dirty sloughy throat, with scanty membrane on tonsils and uvula. The cervical glands were enlarged and tender on the left side.

She was given an injection of 2000 units of antidiiptheritic serum.

A swab was taken and report received 7. 3.20.
"Pure culture Diptheria."

Her condition improved and gave rise to no anxiety. She was very restless during the night of the 6th. Had frequent calls to micturate and defaecate with no result during the night.

I saw her on the morning of the 7th and noticed she was slightly cyanosed about the face. Hands were pale and cold and the pulse extremely small and slow, imperceptible at times at the wrists.

Examination of the precordia showed no visible sign of Apex beat. Cardiac dilness was just within the nipple line.

Judged by the sounds, the heart was beating at the rate of 30 to the minute and irregularly.

A reduplication of the second sound amounting to a third sound, was heard but was not constant.

No murmurs were detected.

A polygraph tracing was taken.
Patient vomited for the first time on the morning of the 8th. She complained of pain in the right shoulder and scapular region, and the neck on the right side was tender to touch. The pain often wakened her in the night.

The cyanosis was very marked. Vomiting was persistent, nothing, not even the smallest quantities of water were retained. Heart rate 20. A tracing was taken.

Had a very bad night, extremely restless, mentally very acute, described by the nurse as uncannily sensible to the last. Died at 8 a.m. 9th March.

Post-mortem examination shewed a few epicardial milk-spots, and marked flabbiness of the heart muscle generally. On microscopic examination, marked round-cell infiltration and fatty degeneration of the muscle was observed.

These changes were seen also in sections of the sino-auricular node and a-v bundle, but in lesser degree.
Tracing was imperceptible.

Pulse at the knee and over the wrists at the time of heart-block showed a varying and high degree of heart-block.

Copy of tracing taken 8.30. Shows a varying and high degree of heart-block.

Tracing taken on the 8th March 1920.

- Shows a varying degree of block.

Both ventricles and auricles are irregular - A.S. 75 to the minute.

Fig. 1

Fig. 2

Fig. 3

Fig. 4

Cases.
REMARKS.

The great majority of cases of heart-block reported have been associated with chronic lesions.

Sir James Mackenzie in 1905 reported the occurrence of heart-block in the course of acute rheumatism and influenza which was of a temporary character.

Magnus Alsleben (10) in 1910 published tracings showing the presence of complete heart-block which developed on the 4th day of disease. Heart-block persisted till death on the 9th day. Severe parenchymatous degeneration of the A-V bundle was found.

Fleming & Kennedy (11) reported a similar case in the same year 1910.

Rohmer (12) in 1911 published two fatal cases showing complete heart-block, and in 1912 five fatal cases.

Price & Ivy Mackenzie (13) published a case of heart-block with auricular fibrillation. Fibrillation in this case is open to doubt.

Hume (14) published an account of four cases of cardiac irregularities in diptheria. His second case which was fatal showed 2:1 heart-block which developed three days before death.
Parkinson (15) in 1915 published a case showing complete heart-block developing in the fourth week of illness in diptheria. Auricular fibrillation supervened the following week and six months later still persisted.
Case 4.

Case 4. age 65, male, a Paper-cutter, seen first on the 21st Nov. 1919, when he complained of having fits.

He had had four fits, one in June, one in Sept. and two in November a week before. On one occasion he had got up to make water at night when he fell unconscious on the floor. He was unconscious for two minutes.

On another occasion he was walking along a road when a brightly lit car passed, he fell unconscious.

On the other two occasions he was at his work as a cutter in the paper factory. He had slightly injured his scalp once when he fell. He had neither micturated nor defaecated during these fits. For a week he had been feeling dizzy and headachy. His radial pulse was 60 to the minute and regular. The vessel wall was thickened and tortuous.

The B.P. 140 systolic and 80 diastolic. Heart dulness extended to the nipple line. The heart sounds were closed but very faint.

A polygraph tracing showed slight widening of the a-c interval marked (x) in the tracing attached for Nov.21st 1919. The a-c interval here is .3 of a second.

Nothing abnormal was detected in any of the other systems. November 30th 1919 he had a syncopal attack in which he lost consciousness for 1½ minutes.

December 4th he had a similar attack, and had for the
first time involuntary micturition during the attack. He was also much cyanosed.

December 14th, he was giddy on rising and had a slight syncopal attack. His pulse was irregular.

Dec. 28th. He had a syncopal attack with spasms of the limbs. The fit lasted 5 minutes. He was troubled much with vomiting and flatulence afterwards.

His pulse rate was 22 to the minute and he had a pain in the epigastrium.

Dec. 30th. He had frequent syncopal attacks.
Pulse rate was 18 to the minute. During the attacks he had slight spasms. He had much nausea and epigastric discomfort.

Dec. 31st. Cheyne-Stokes breathing was noticed for the first time.

Jan. 1st. to 5th. 1920. His pulse rate was 24 to the minute. Giddiness was present but better. He was giddy now only on sitting up in bed.

Jan. 7th. Very slightly giddy on sitting up.

Jan. 8th. complained of feeling hot and thirsty.

Jan. 10th. Thirst less. No giddiness on sitting up.

Breathing Cheyne - Stokes in character. Pulse rate 39.

Jan. 11th. Expectoration was blood stained. He had no giddiness.

Jan. 12th. Felt much better.

Jan. 16th. " " "
3.

Jan. 20th. Had syncopal attacks with spasms and laboured breathing, occurring frequently from 4 a.m.

He died at 12 noon.

At no time had he any elevation of temperature. Even when he felt hot and thirsty his body temperature was normal or below normal.

Unfortunately no post-mortem examination could be obtained in this case.
Case 4.

Tracing taken November 21st. 1919.
The a - c interval which is .2 second elsewhere in this tracing is about .3 seconds where marked with (x).

Tracing taken 5 - 1 - 20. showing complete heart-block. The auricular rate is 75 to the minute. The ventricular rate is 27.2 to the minute.
Case 5.

Case 5. Male a joiner and cabinet maker, age 60.

COMPLAINT.- Feels weak on exertion.

Healthy till 18 years ago, then had Influenza and Pneumonia. Ill for three months. Often felt done up at the end of the day, even after a light days work. Has been in bed ill once in 17 years, 8 years ago when he complained of weakness and sweated profusely, felt much relieved with a glass of whiskey and water and completed his work. A week later he went to see his doctor as the weakness was progressing. He had no breathlessness at that time. Two or three years later he began to be breathless, but then, and now, walk in his own time on the level for about 3 miles. Cannot hurry without becoming breathless and fatigued, especially on an incline. He has no pain.

His legs began to swell about a year ago. He often did not remove his boots for a fortnight at a time, as he was afraid he could not get them on again. He slept as a rule in his chair. The swelling disappeared 6 months ago and there has been no recurrence.

Appetite is fairly good, no indigestion. Bowels regular. Temperate with alcohol. Sleeps not so well. Slow in falling asleep. Sleeps with head high. Rests better on left side. Rises to pass water generally about three times in the night.
With strain at any time he begins to shake in the limbs. There is no history of loss of consciousness. His colour is good and he is healthy looking for his years. There is no cyanosis and he is well nourished. He has 9 teeth. There is pyorrhea.
Pulsation apart from the carotid throb can be detected in the neck. The apex beat of the heart is in the 6th interspace in the nipple line. A soft systolic murmur is heard at the apex and is conducted into the axilla. The second sound is closed. A third sound is occasionally heard at the base, in addition to a systolic murmur.


----------------- Diastolic 100. " " 110.

The radial arteries are thickened and tortuous. The pulse rate is 38 to the minute.
March 19 1920.
As. 60 to the minute.
Vs. 27.2 to the minute.

Note the gradual lengthening of interval from left to right between the carotid beat and the auricular immediately preceding it.
Case 6.

Case 6. Seen first Aug. 5th 1920, a Ploughman, age 53.

COMPLAINT.- Breathlessness on exertion, which began four days before. He had previously been quite fit.

He had worked all summer forking hay and hoeing in the fields. For a day and a half now, he had been off work on account of breathlessness. Though he could not work as formerly, he could walk on the level and even up hill without resting.

For three weeks he has had pain in the epigastrium and along both flanks. The pain is dull and aching in character. He feels it most just after retiring to bed. It eases off in one hour. He does not feel it during the day time. He has no faintness, no giddiness and no palpitation.

No history of syphilis could be obtained. His previous health had been perfect except for sciatica 7 years ago.

He drank rather heavily at one time, but had been temperate for some years.

His heart showed considerable enlargement, the dulness extending into the 6th interspace 1½ inches beyond the mid-clavicular line. The apex beat showed a marked indrawing during systole of the heart, and a double movement during diastole. (See tracing No. 2)

A systolic and diastolic murmur could be heard at the
apex and more distinctly at the base of the heart and down the sternum.

The wall of the radial artery was somewhat thickened. The pulse rate 60 to the minute was not water-hammer in character, and showed no arrhythmia.

The systolic blood-pressure was 140 m.m.Hg.

I saw him again on the 12th August. He had been getting gradually worse. He had had no syncopal attacks. He had become orthopnoeic and for two days had been troubled with a hard dry cough whenever he lay down. His colour had become ashen and his ears cyanotic. He had a considerable amount of tenderness in the epigastric region, liver and splenic dulness were both increased. His pulse rate was 44 to the minute. He complained greatly of thirst. He told me he could drink the sea dry.

His temperature was normal. There was no oedema of the ankles and no dulness or crepitations at the lung bases. A polygraph tracing showed he had partial heart-block.

The next day he was much more breathless. The breathing was Cheyne-Stokes in character. He was having severe attacks of cardiac asthma. Crepitations could be heard over both lung bases.

I determined to observe the effect of 1/50 grain of atropine sulphate in his condition and took a continuous record of his radial pulse and jugulo-carotid for 10 minutes before and an hour after the injection. No alteration
occurred. The heart-block remained of the same partial degree. (See tracings attached)

A drachm of the tincture of digitalis was administered daily and the effect observed. (Tracings attached)

A gradual improvement of his symptoms occurred. Breathing became easier, the attacks of cardiac asthma less frequent and less severe. He slept better, cough was slight. Pain and tenderness in the epigastrium was less and the liver dulness decreased.

Digitalis was stopped after five drachms had been administered, - when one effect observed was a very definite coupling of the ventricular beats. (Fig.6.)

Twenty-four hours later all his symptoms became aggravated, Cheynes-Stokes respiration, tenderness in the epigastrium. (Fig.5.)

During an attack of dyspnoea which he had while I was present, his radial pulse for about 20 beats was at the rate of 100 to the minute, then continued at about 42 to the minute. (Fig.5.)

During a subsequent slighter attack of dyspnoea this pulse change did not occur.

For the next two days he seemed to drift from bad to worse, sleeping badly, very breathless, great pain in the hypogastrium on both sides, face puffy and hands swollen, a few crepitations at the base of the lungs and no oedema of the ankles. The pulse was 41 to the minute
with only an occasional irregular beat.

He died suddenly the next day.
Figure 1

August 5th, 1920. Radial Tracing. 60 to the minute. Regular.

Figure 2

August 5th, 1920. Apex beat showing indrawing during systole.

Figure 3


As. 110.
Vs. 40.

Marked widening of a-c interval (beats 1, 3, 4, 6, 7.)
Fig. 4

Action of Digitalis upon pulse, one Drachm daily for five days.

Before administration shows 3:1, after administration shows 3:1 and 2:1.

August 13th, 1920. Shows Cheyne-Stokes breathing with a 12 second apnoic period.

Ventricular, auricular rate 42.8 to 50 per minute.

Fig. 5

Continuation of tracing upon previous page. (Fig. 3)

Heat-block auricular rate 40. Before administration shows 3:1 and 2:1.

August 17th. 1920.
Shows coupling of the ventricular beats, due to Digitalis. Patient had had 5 drachms of the Tincture by this date.

Auricular rate 107 to the minute.
Ventricular " 45 "  "  "

The jugulo-carotid tracing is poor, but there are two auricular beats which are distinct. Auricular waves can be noticed upon the down slope of the radial tracing.

August 18th. 1920.
Shows 3:1. and 2:1.

Heart-block.

Auricular rate 109.
Ventricular " 40.9."
Action of Atropine Sulphate 1/50 Grain Injected Hypodermically.

10 minutes after injection.

Before injection.

Auricular rate 100 to the minute.

Ventricular 37.5 to the minute.

15 minutes after injection.

As. 100. to the minute.

Vs. 37.5. " .

18 minutes after injection.

As. 100. to the minute.

Vs. 37.5. " .
20 minutes after injection.

As. 100. to the minute.

Vs. 37.5.

30 minutes after injection.

As. 100. to the minute.

Vs. 37.5.

35 minutes xx after injection.

As. 100. to the minute.

Vs. 38.
45 minutes after injection.

As. 100. to the minute.

Vs. 36. 

60 minutes after injection.

As. 100. to the minute.

Vs. 37.5. 
I am indebted to Dr. Ivy Mackenzie of Glasgow, for the following report on the post-mortem examination of the heart.

POSITION IN BODY,-

Large and flabby, the right ventricle was lying to the front.

There were 4 oz of blood stained fluid in the pericardial sac. Before opening the organ it was noted that there was an aneurysmal dilatation of the right posterior wall of the lower part of the aorta, large enough to occupy half a tangerine.

Palpation revealed a hard thickening, occupying the base of the heart in front of and to the right side of the region of exit of the pulmonary artery and aorta.

The four chambers of the heart were markedly dilated and were found on being opened to contain a considerable amount of ante-mortem clot.

Two large areas of gummatous infiltration lie in the musculature.

1) One moderately circumscribed and about the size of a walnut lay in the anterior wall of the infundibulum and extended from the region of the moderator band in front to the membranous septum behind.
2.

2) The other, more diffuse, infiltrated the wall of the inter-ventricular septum and could be seen beneath the endocardium extending in the infundibulum from the insertion of the pulmonary valves right through the moderator band to its insertion in the anterior wall of the right ventricle.

In the left ventricle this nodular infiltration could be seen and palpated throughout the region of initial spread of the left limb of the atrio-ventricular bundle in the upper third of the inter-ventricular septum.

VALVES,-

Associated with the flabbliness and general dilatation of the organ, the bicuspid and tricuspid apertures are so wide as to render the valves incompetent.

The bicuspid admits 3 fingers, and the tricuspid " 4 "

The valves themselves are thin and transparent. The muscle substance is soft and flabby, and apart from this shows no change to the naked eye except in so far as it is invaded by the infiltrating masses referred to above.

AORTA,-

The thoracic aorta is diseased throughout its whole extent. There is extensive pale blue cicatrical scarring interspersed with areas of calcareous and fatty
infiltration, only in few and isolated patches is there evidence of the existence of that normal yellow appearance due to elastic tissue.

The right posterior wall at the commencement of the aorta shows the hemispherical dilatation described above.

On holding up to the light, the patchy thinning of the aortic wall is well marked, and the post-coronary artery lies in the lower aspect of this dilatation and there is considerable narrowing of its opening through occlusion by the cicatrization in the wall of the aneurysm.

The pericardial surfaces are smooth.

Liver and kidneys show venous congestion.
There are features in this case that deserve discussion.

PAIN

This was the first and only symptom present over a fortnight before the breathlessness began and compelled the patient to rest from his work and seek medical aid.

The site of the pain is interesting and instructive. It is not in the usual site for pain of cardiac origin, which is precordial as a rule with a tendency to radiate into the neck and down the left arm. In this case the pain was as indicated in the diagram on the right side extending from the lower margin of the liver to about 2 inches below the navel. The skin was not hyperalgesic but the muscle was tender to touch. A separate and smaller area of muscular tenderness could be elicited in the left hychondrium. With the improvement of the patient's symptoms between the 13th & 18th of the month, these areas of tenderness disappeared to return as definitely as before on the latter date.
These areas of tenderness in this case would seem to be associated with the engorgement of liver and spleen, being most acute when these organs showed evidence of greatest enlargement. His freedom from precordial pain or pain of an anginous nature is worthy of note in view of the narrowing of the orifice of the posterior coronary artery in the wall of the aneurysmal dilatation.

BREATHELESSNESS,- was his most prominent symptom and the symptom which compelled him to desist from work and seek medical aid.

THE APEX BEAT,- of the heart in this case is worthy of note. There was a marked indrawing during systole and a double movement of the chest wall during diastole when he was first seen. $\left(\pm q.2\right)$.

It is likely that this was due to embarrassment of the right ventricle which was considerably dilated and post-mortem was found lying well to the front.

The gummata so encroached upon the cavity of the infundibulum that it must have been with great difficulty that the right ventricle pumped its contents into the pulmonary artery.

The systolic and diastolic murmurs heard over the precordia may in part if not altogether have been produced in this constriction of the infundibulum, for I am not satisfied that they could be produced at the aortic orifice.
3.

the valves of which post-mortem in spite of the aneurysmal sac were competent to support a short column of blood in the aorta. Again, the absence of a water-hammer pulse and the distribution of the murmurs heard most distinctly at the base and down the sternum may be supporting evidence in favour of the murmurs being caused at the infundibulum.

Against this the difficulty of accounting for the diastolic part of the murmur. On the other hand the blood pinched between the constriction and the pulmonary valves regurgitating into the main cavity of the right ventricle, in early diastole and the inrush the other way through this constriction during active diastole, might be urged as possible factors in the causation of this diastolic murmur.

Since writing the above, Case 7 has come under my notice. Here the systolic and diastolic murmurs were heard best to the left of the sternum at the base of the heart, and the snap of valves closing could be heard to the right of the sternum.

The systolic and diastolic murmurs in this case were no doubt produced by the distortion of the cusps of the pulmonary valve, two of which were rendered incompetent.
Case 7.

Heart-block due to Gumma.

Patient "B", age 30, a clerk, awoke in the middle of the night, four days ago, feeling faint, found himself struggling with the bed-clothes, and fell out on the floor. He rose and turned on the light. He felt nauseated took a drink of water, and began retching.

Attacks of retching accompanied by a dizzy feeling in the head, a dull feeling of discomfort in the pit of the abdomen and a sinking-away feeling came on at half hour intervals throughout the night until 10 a.m. next day.

In the afternoon he felt much better but remained in bed. Next evening at 11 o'clock he had been asleep three hours when he was awakened with a repetition of the attacks of the previous evening. This time he also sweated profusely.

On the day he was examined he had been having frequent faint turns lying flat in bed, unconsciousness would last about 20 seconds. He awoke to find himself in a sweat, very thirsty, and his feet feeling cold. No convulsions had been noticed.

The patient had been sleeping badly and was extremely restless. He was thin, sallow, rather ashen complexioned, short of breath with the exertion of talking, but mentally acute.
He was having frequent turns, and these coincided with an absence of pulse at the wrist, which otherwise was regular at 30 to the minute, and absence of heart sounds over the precordia.

These pauses lasted three seconds, and the patient did not altogether lose consciousness. He complained of his feet feeling cold and of a thirst.

His hands and feet felt cold to the touch. The body temperature was normal.

The apex beat of the heart was visible in the sixth inter space three quarters of an inch outside the nipple line. The deep dullness of the heart extended also to this point. A rough systolic murmur could be heard at the apex and louder in the pulmonary area.

In the aortic area the second sound was closed. A faint diastolic murmur could be heard to the left of the sternum at the base of the heart. A third softer muffled sound could be heard at times, and periodically the first sound was much increased in intensity and roughened.

The systolic blood pressure was 103, the diastolic 45 m.m. Hg. The pulse rate 30 to the minute.

Moist crepitations could be heard at the lung bases. The liver and spleen showed no obvious enlargement. There was no oedema of the subcutaneous tissues anywhere. The urine contained nothing abnormal.

Sixteen months ago he had suffered from a "nervous
breakdown. " On detailed inquiry it seemed that he had peculiar turns when walking. He would stop for a few seconds and require the support of his wife.

He was suffering at this time with an ulcer upon his tongue which took six months to disappear. There was a recurrence two months later of an ulcer upon the tongue which took three months to disappear. He began complaining at this time of a dull ache under the left breast, and off and on this had worried him from that time.

He had had influenza severely in 1918, and pleurisy eight years ago on both sides. There was no history of rheumatic fever, chorea or tonsilitis, and no history of venereal disease could be obtained.

He was a married man with one child five years of age. His wife had had no miscarriages. His family history was very good. A month previous to his illness four days ago, he had played thirty-six holes at golf, and up to a week ago he had played 18 holes three times a week.

For a day or two the patient seemed to improve. The faint turns disappeared. The pulse became fairly regular varying between 28 and 44 to the minute.

To exclude vagal influence on the heart's rate, an injection of 1/100 gr. of Stropine Sulphate was given and the effect observed on the pulse tracing as taken with the polygraph, before, during, and for an hour after the injection.
intervals. Pauses of three seconds became frequent disturbing the previously regular action of the ventricle.

The disturbed action which was present 45 minutes after the injection disappeared 15 minutes later, as shown in the tracings. Apparently under the influence of atropine a temporary complete dissociation of auricles and ventricles was rendered partial for about an hour.

Thus a small portion of the block may have been due to the influence of the vagus, probably upon a few surviving fibres of the A.V. bundle. The greater part of the bundle was involved in some structural defect.

A Wasserman reaction taken now proved very strongly positive.

A provisional diagnosis of gumma of the heart involving the A-V. bundle was arrived at, and the patient put upon iodides and innunctions of mercury, but without benefit. The signs of cardiac failure increased, fluid accumulated at the lung bases, the liver and spleen became enlarged, breathlessness increased, cough became troublesome, accompanied by blood-stained frothy sputum.

The blood-pressure fell to 97 systolic, 44 diastolic. The pulse rate varied between 24 and 32. The patient complained of great weariness and thirst, and death occurred 18 days from the sudden onset of symptoms.
Photograph of heart viewed anteriorly.
A - Aorta.  P.A. - Pulmonary artery.
L.V. - Left ventricle.
G.G. - The upper and lower limits of the gummatous growth.

The anterior wall of the right ventricle has been turned back from an incision parallel to the anterior inter-ventricular septum and the growth on the posterior wall of the infundibulum is disclosed.

Note its close relationship to the pulmonary valve, the right anterior and posterior cusps of which are distorted.
The dissociation is probably complete, at any rate while the tracing was being taken.

The auricles and ventricles are acting with great regularity. Both auricles and ventricles are acting at a rate of 85.7 to the minute. The ventricle at 46.15 to the minute.
This was taken 25 minutes after an injection of 1/100 gr. of Atropine Sulphate. The auricles are beating at the rate of 84 to the minute. The ventricle, except for the one pause of 3 seconds duration, is beating at the rate of 42 to the minute. Probably a 2:1 partial block. After the pause the brachial tracing shows the pulsus alternans, a condition to which Wardrop Griffith* (9) has drawn attention. He found it only in a 3:2 ratio of As. to Vs. and only when there was a coincidence of every third atrial systole and every second ventricular systole. Here the ratio is 2:1. There is no nearer coincidence of As. and Vs. after the pause than before. The slight delay in the appearance of the smaller beat is also shown here. (9).
Fig. 3

A tracing taken 45 minutes after an injection of 1/100 gr. of Atropine Sulphate.

The auricles are beating at 96 to the minute, the ventricles irregularly varying from 27.2 to 35 to the minute.

The irregularity may be due to ventricular extra systoles, but in view of Fig.2 it may be that the auricle is occasionally awakening a ventricular response in the earlier part of the tracing.

Fig. 4

Tracing taken 60 minutes after Atropine Sulphate injection of 1/100 gr.

As. 102.
Vs. 27.2

Vs. regular, As. irregular.

In the tracing taken immediately before the injection the As. were 90 and the Vs. 40, both regular.
Case 7.

I am indebted to Dr. J. H. Harvey Pirie, of the S.A. Research Institute, for the following description of the heart post-mortem.

POST-MORTEM EXAMINATION OF "B."

PERICARDIUM._ The pericardial sac contained 6 oz. of a clear greenish-coloured fluid. The visceral pericardium over the right ventricle and auricle showed a diffuse fibrous thickening with some fibrous tags.

There were no adhesions to the parietal layer. This thickening extended upwards as a diffuse thickening of the adventitial tissue around the intra-pericardial portion of the pulmonary artery and aorta.

THE HEART" was enlarged particularly in a transverse direction, the enlargement being chiefly of the right side.

There was an obvious nodular bulging the size of a split walnut in the anterior wall of the infundibulum of the right ventricle, one inch below and to the right of the root of the pulmonary artery. On palpation a hard mass could be felt extending from this position upwards to the level of the pulmonary valves and downwards, and to the right, almost to the right border of the heart, a total distance of three inches vertically and 1½ inches wide at its widest portion.
THE AORTIC CUSPS were competent by the water test. The pulmonary cusps were incompetent by the same test. The heart was fixed in formalin before being opened up for further examination.

Looking into the pulmonary artery towards the cusps one could see immediately under the cusps a mass projecting from under the right anterior cusp across the lumen to the opposite side, blocking the passage-way under the right anterior and posterior valves, leaving merely a small slit-like opening beneath the left anterior cusp.

On opening up the right ventricle with a cut parallel to and close to the inter-ventricular septum, the projecting mass referred to above was seen to be merely the upper pole of a large hard nodular yellowish growth on the posterior wall of the infundibulum, involving below the papillary muscles almost to their attachment to the inter-ventricular wall.

The cavity of the infundibulum is almost completely occupied by the growth.

The right auricle is dilated and the muscle of the wall is considerably hypertrophied.

Between the coronary sinus and the superior vena cava and one inch anterior to the fossa ovalis, a yellow hard mass projects into the lumen of the right auricle.

This mass was felt to be continuous with that described in the infundibulum and it extends to the right margin of
the heart in the auriculo-ventricular septum, narrowing the lumen of the tricuspid orifice, the anterior and medial cusps of which are stretched over the growth and rendered incompetent. The cusps are more opaque and thickened than normal.

THE LEFT VENTRICLE is of normal size. There is no hypertrophy or dilatation obvious and the muscle appears healthy. A single smooth yellowish nodule is seen projecting into the left ventricle from the inter-ventricular septum in the region of the membranous portion opposite the base of the anterior cusp of the bi-cuspid valve.

THE LEFT AURICLE, shows no dilatation or hypertrophy, the bicuspid valve is not dilated, and the cusps appear healthy.

THE AORTA. The aortic cusps show a slight diffuse thickening, but are not distorted and appear to be competent. There is a slight bulging into the sinus of Valsalva of the anterior cusp.

The orifices of the coronaries are not obstructed. The aorta is small in diameter. The intima shows small yellow fatty patches and streaks throughout its whole length, this condition being at least as marked in the abdominal portion as in the thoracic. At the root of the aorta and again between the coeliac axis and the renals are patches of more advanced arterio-sclerotic changes.
There were about two pints of clear fluid in the right pleural cavity.

The lungs, spleen, liver, and kidneys showed nothing abnormal.

Sections were made of a portion of the nodular growth projecting into the lumen of the right ventricle.

Dr. Pirie reports as follows: "The structure exhibited is that of a granuloma with extensive coagulation necrosis. Giant cells are not in evidence. Sections were stained for both tubercle bacilli and spirochaetes (the latter by a modified Levaditi method) but in each case with negative findings.

Despite the failure to demonstrate the actual presence of the treponema pallidum, I have no hesitation in diagnosing the nodule as a syphilitic gumma."
Case 8.

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Complained of a sense of oppression in the chest in the precordial area, felt somewhat vaguely. Occasional attacks of palpitation associated with dizziness and shortness of breath especially on exertion, there is no pain.

Found himself sighing frequently, with an inclination to take a deeper breath at times. He was subject to attacks of diarrhoea, after which these symptoms were readily induced. Worry, excitement, smoking, and hard work also brought on the attacks.

There is no history of syphilis or rheumatic fever, but the patient had dysentry in 1914 and trench fever in 1916 in France. The symptoms of which he complains date from that time.

On examination the heart showed nothing abnormal except occasional extra systoles. A polygraphic tracing taken at this time shows normal a-c-v waves.

For five months he was very much better in health, having had very few and slight attacks. He dropped smoking altogether.

On the 8th June 1921, he had five short attacks of shortness of breath, with a sensation of weight in the precordia. He feels "as though there were a lump or a weight behind the heart." There is no pain.
Exertion brings them on. The fifth attack during which I saw him and obtained the polygraph tracing Fig. 2. came on while he was standing watching an operation in a surgical theatre. The auricles are acting at 300 to the minute, while the ventricle is acting irregularly at about 100 to the minute. The attack lasted about half an hour. The tracing shown in Fig. 3 was taken within a minute of the cessation of the attack and shows the return of the normal rhythm.

There is no prolongation of the a.c. interval in this tracing or the one taken five months before.
Case 8.

**Fig. 1.** Shows a normal tracing.

**Fig. 2.** Shows Flutter.

As. 300.
Vs. 100. and irregular.

**Fig. 3.** Taken within a minute of the above (Fig. 2). Shows return to normal. a.c.v. rhythm.
III. SUMMARY.

Clinical records of eight cases of heart-block are given. Two cases, 1 and 5, are examples of chronic heart-block. Both are still alive.

In case I. the question arises as to whether the block had existed from infancy. It had then been observed that he had only half a normal pulse rate.

Still there is a possibility of this being due to a regular extra-systole occurring after each normal beat, the wave being too small in volume to be felt at the wrist. (4)

If the condition of block existed at that time the heart muscle must have been in a wonderfully healthy state and the lesion be confined to the conducting mechanism.

Case 5. was known to have heart-block for eight years. Here too the heart muscle must be fairly healthy as shown by his response to effort.

In neither I. nor 5. was there a history of convulsions.
2). Case 2. had his first attack with unconsciousness, eight months before he was seen. Atropine relieved the partial block completely. The normal a - c interval is noteworthy.

3). Case 3. is an example of very acute heart-block occurring in the course of an acute illness, - Diptheria. Death ensued in three days from the onset of block or upon the tenth day of illness. The prognosis when this condition has developed is almost hopeless. One case of recovery is described by Hume. (14) This child received only 2000 units of anti-diptheritic serum, and on the third day of illness.

4). Case 4. was rapidly progressive. He died two months from when he was first seen. Unfortunately no evidence of syphilis was obtained in his case, and the question of gumma was not excluded.

5). Cases 6. and 7. are examples of heart-block due to syphilitic gumma involving the A-V bundle. With the presence of systolic and diastolic murmurs at the base, the differential diagnosis from aortic disease arose.
Case 6. is primarily a case of flutter of the auricles, in which a degree of heart-block rather higher than usual existed during the attack.
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