Case of rheumatic heart disease (mitral incompetence and aortic stenosis) with auricular fibrillation.

Wrightman Prize in Clinical Medicine 1936

Tom Hewittson Brown

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MARTIN BATTLE. act. 55. born in Edinburgh.

married. electrotyper.
admitted 6. XI. 1934.
examined 6. XI. 1934.

Complaint: breathlessness on exertion & lassitude since July 1934; extreme breathlessness on exertion since Oct.

History.

Present Illness. Until July 1934 the patient was in his usual health. Having travelled to London by motor omnibus he returned to work on July 27th, in spite of feeling nervous and out of sorts. On July 30th, on his way to work, he experienced a sensation as of something rising in his chest and choking him, and of trembling in the upper part of the thorax; there was no pain, no dyspnoea and no faintness. He went straight home and to bed, where the choking feeling left him, nor has it returned since. His doctor treated him at first for a chill, later for disordered action of the heart. After a fortnight the patient got up, and in the second week of September returned to work, still tired, overdone & breathless on exertion. About a fortnight ago, in the course of his work, he had to lift a 70 lb. weight, as he did so he felt a pain shooting through his chest to the back. Since then he has been more breathless than ever, and now cannot walk more than 20 yards without stopping. He remained at work until Oct. 21st. During his last fortnight at work he occasionally felt slightly dizzy, but recovered within half a minute if he could sit down. During the last three weeks his normally excellent appetite has left him, & he has been nauseated by the smell of the kitchen. His doctor sent him to the R.I.E. with the provisional diagnosis: "? partial heart-block."
Previous History. At the age of 13 the patient was in bed at home for several weeks with rheumatic fever. He made a perfect recovery, and was subsequently able to take part in such strenuous sports as cross-country running and playing. At 21 he was in bed at home for four months and in the R.I.E. for two months with sub-acute rheumatism; at that time he had very severe joint-pains. From that time onwards he has never taken any exercise apart from his work, which entails hard physical exercise, and walking. On an average he has been able to remain at his job about eleven months in a year, his commonest complaints being "colds" and "slight break-downs." The doctor has never mentioned heart disease to him, but in 1917 or 1918 he was placed in category C2 because his head was affected; he was never called up for service. He has continued to live a very careful life until his present illness.

For years he has suffered from palpitations, particularly when he was excited or had to work late, also if he attempted to lie on his right side. He is much troubled by wind, and only obtains relief by eructation; if he was unable to do so, that too brought on palpitations. He has never had any swelling of the ankles. He has had no urinary troubles, except that of late he has passed little water during the day and had to rise about twice nightly.

During the past few weeks he has suffered from cough, accompanied by pain in the right parasternal region; there has been little spitting, which was never brown or blood-stained.

His general surroundings at home and at work are good. He is a moderate smoker and drinker. Family. His wife is alive and well. Of their four children two died of pneumonia in infancy; the other two are alive and well.
State on Examination.
Intelligence average. Height 5 ft. 3½ in. height 11 st. 2½ lbs.
Well-built, muscular, fair hair, very well nourished.
Florid appearance. No apparent jaundice, no cyanosis, no dropy. Temperature 97.0° F.
Circulatory System.
No complaint of pain. Patient suffers from palpitation when worried, excited or over-tired, also when troubled with flatulence. Of late he has occasionally felt dizzy when at his work. He is very breathless on exertion. He has a slight cough, and some sputum. Pulse: arterial wall soft, palpable, not tortuous. Frequency 120. Totally irregular in time and force. Wave of fair strength and well-maintained. B.P. 150/90. Sphygmographie tracing:

Heart: precordium normally developed. Pulsations are readily visible in the epigastrium and over the left anterior part of the chest. The apex beat is particularly readily seen when the patient sits upright; it lies 5½" from the median plane, in the 5th intercostal space. There is no visible pulsation in the neck.
The apex beat is felt most easily at the point at which it is seen; it gives the impression of a gradual increase of pressure followed by a gradual decrease, but its whole duration is short compared with the cardiac cycle. Pulsations are also felt, more faintly, in the 6th intercostal space, below the apex beat. There is no palpable thrill.
Deep dulness to percussion extends 2" to the right and 5½" to the left of the median plane; the upper
border lies at the 3rd costal cartilage. In the initial area the first sound merges into a loud, blowing murmur which is terminated by a very faint, sometimes inaudible, second sound. The murmur is propagated slightly towards the axilla. In the auricular area both sounds are pure and closed. In the aortic area there is a variable systolic murmur; it is always rough, but sometimes faint and sometimes loud; it is readily audible at the 2nd right costal cartilage, loudest in the 2nd left intercostal space near the sternum, at the right side of the sternum about its middle; it is propagated into the root of the neck, and sometimes high up into the neck. The second sound is rough, but there is no true murmur. At a point midway between the apex and the 2nd right costal cartilage there is a loud pure first sound, followed by a very accentuated second sound. The frequency of the ventricular rate as determined with the stethoscope is greater than that of the radial pulse; the deficit is small when the rate at the apex is rapid, but increases very considerably when the apex rate slows, as, for example, 135 : 120; later 110 : 90. The veins in the neck are not obviously distended. There is no visible capillary pulsation.

The electrocardiogram was recorded on the date of admission.

Alimentary System.

Appetite good as a rule, poor during the past three weeks.
Flatusence is very troublesome at times, but is relieved by
exercise. There is no complaint of pain.

Toque covered with whitish fur; teeth all fair, dentures
fit satisfactorily; on the right tubis there is small white
pulvinate spot.

There has been no vomiting. The patient has been in the
habit of regulating his bowels with aperient medicines. He
has at times suffered from piles and passed traces of
fresh blood with his stool; he has been free for some
time.

Abdomen: Flaccid, adipose; movements free. There is no tender-
ness or undue resistance. The spleen is not enlarged to per-
cussion or palpation; the liver is not palpable; the lower
border of hepatic dulness coincides with the costal margin
in the nipple line.

Haemopoetic System.
No abnormal subjective phenomena. There is no enlargement
of the lymphatic glands or thyroid gland.

Blood Examination: R.B.C. 5,150,000. Hb. 108%. C.1. 1.0
W.B.C. 8,000.

Respiratory System.
No history of pain. There has been some cough during the
past three weeks, but no sputum or haemoptysis. Patient
complains of extreme breathlessness on exertion.

Respiration: Frequency 20, regular. Phonics healthy. Voice
clear.

Thorax: Ossosous thorax well-formed, symmetrical, well-
covered with fat. Vocal fremitus is normal & equal in
corresponding areas. The percussion note is resonant &
equal in corresponding areas. Auscultation: in front the
breath sounds are vesicular, without accompaniments
except for occasional phonchi. On the left axilla the breath
sounds are broncho-vesicular, without accompaniments.
Posteriorly there are fine respitations at the base & occasional
phonchi; breath sounds otherwise pure, vesicular.
Integumentary System.
No abnormal subjective or objective phenomena.

Urinary System.
Of late the patient has passed little urine by day, but has had to rise twice nightly for the purpose. There has been no difficulty in starting or stopping urination, nor weakness of the urinary flow.

Urine: clear, yellow, acid; no abnormal constituents. Specific gravity 1023.

Nervous System.
Intelligence normal. Patient is obviously a man who has always taken extreme care of his health. Memory for distant and recent events good. Sleep is very poor. Cranial nerves: normal. Acuity good; fields of vision normal; the fundus of the right eye is much more pigmented than that of the left. Ocular movements intact. Pupils normal; no mydriasis. Hearing is good; bone conduction is relatively poor on the right side. No other abnormality is noted. Motor functions: no abnormal movements, no paresis; muscle tone fair; co-ordination good.

Reflexes all normal. There is no sensory disturbance.

Locomotor System.
Nothing abnormal noted.

Provisional Diagnosis.
Rheumatic heart disease (mitral incompetence & aortic stenosis) with auricular fibrillation.

Treatment as administered.

1. Rest in bed.
2. Light diet.
3. Tincture of Strophanthus, 40 min t.i.d.
Progress notes.

Admitted.

Patient feels quite comfortable, and looks extremely well, unless his colour be considered too florid. Heart sounds unchanged.

Urine contains a trace of albumen.

Weight: 11 ft. 297 lbs.

P.P. 178/100.

To-day, for the first time, a systolic thrill was felt at the apex, and only for a brief period after the patient had exerted himself. The systolic murmur at the base is louder than before and more clearly propagated into the neck.

The systolic murmurs at apex and base were harsher in character than before, so long as the patient remained recumbent; when he sat up, the murmur at the apex disappeared. There was at no time any thrill to be felt.

Pulsus alternans readily detected by palpation.

Weight: 11 ft. 2 lbs. Wassermann reaction negative.

P.P. 164/108.

Weight: 11 ft. 1½ lbs.

In the morning the patient felt perfectly well, as usual. At mid-day he felt a sharp stabbing pain in the right axillary line; the pain was worse on coughing or on taking a deep breath. At this time there was no objective sign of change in the chest. Towards evening the pain became worse and the patient expectorated some blood-stained sputum. By
this time there were numerous medium & coarse crepitations at the site of the pain, but no friction sounds were heard. The temperature rose to 103° F and the radial pulse rate to 112; the ventricular rate was 142. B.P. 144/70.

1. XII. Pain has disappeared; persistence of blood-stained sputum. Many crepitations still heard.

5. XII. No more crepitations to be heard. Weight: 11 st. 1½ lb.

12. XII. Pulse deficit at night 52. B.P. 160/100.

13. XII. Patient complains of feeling first shivered, then feeling very hot during the night. Return of the blood-stained sputum, the blood being in the form of small clots. Pain was also experienced in the same region as before; again no friction sounds were heard, but there were numerous crepitations.

15. XII. Pain & sputum have disappeared. The systolic murmur at the apex has at times a slightly musical character; at the base both sounds are fainter than before. No crepitations. Few rhonchi at right base.

16. XII. B.P. 120/80.

17. XII. Patient discharged at own request.

X-Rays of 12. XI. 1934
Commentary.

If, as his doctor suggested, this patient had been suffering from heart-block of diagnosable degree, then his very rapid and irregular pulse could only have been accounted for by assuming the occurrence of very numerous extra-systoles. The alternative and much more usual condition in which such a pulse is found is auricular fibrillation.

In the former condition the extra-systoles must be assumed to be very numerous and therefore an accompaniment of advanced myocardial degeneration, such as might be expected to lead to oedema of the legs, enlargement of the liver, and hydrothorax; none of these was observed. Moreover, in this condition the contractions of the auricles might be expected to produce more readily visible venous pulsation in the neck than was actually present.

On the other hand, his history of rheumatism and long-standing heart-trouble, the rapid, irregular pulse, and the examination of his heart (which suggests a combined mitral and aortic lesion) all agree well with a diagnosis of auricular fibrillation; in its favour is the increase in the pulse-rate which was noted when the patient walked to the electrocardiographic department. A true pre-systolic murmur would not be consistent with this diagnosis, but none was heard at any time. The patient's age does not help us to decide between the two conditions, neither does the sphygmographic tracing. But if we had to decide on clinical grounds alone, then the decision must rest with auricular fibrillation, and in this case the electrocardiogram settles all possible doubt.

This being the case, it is impossible to understand why the doctor should have written of heart-block, unless it be that the patient's pulse-rate has changed very materially; certainly auricular fibrillation lasted throughout the whole of the patient's treatment in hospital, that is, sufficiently
long to preclude the possibility of its being paroxysmal in nature.

The pulsus alternans, which was sometimes very readily recognized by the finger, is to be regarded as a serious sign; as Lewis says: "Whenever it occurs there is reason to believe that the heart, or a substantial part of it, is struggling to perform work of which it is scarcely capable."

As to the valvular lesion, the loud blowing murmur at the apex, slightly propagated towards the axilla; the marked accentuation of the 2nd pulmonary sound; and the appearance, on one occasion, of a systolic apical thrill, are conclusive evidence of mitral incompetence; the incidents of November 20th and December 12th - pain in the side, raised temperature, expectorations, blood-stained sputum - are interpreted as pulmonary infarcts, and being of common occurrence in mitral disease. Mitral incompetence is generally associated with stenosis, but the presence of fibrillation makes it impossible to detect stenosis by auscultation or from the echocardiogram.

Aortic stenosis is much less common, and is generally accompanied by a palpable thrill in the aortic area, by displacement of the pulse, hearing apex-beat downwards and to the left, by an aortic systolic murmur propagated into the neck, and by a low pulse pressure; the pulse is slow and is generally anacrotic or flat-topped. In this patient there is no palpable thrill at the base of the heart; the apex-beat is displaced downwards and to the left; the character of the apex-beat is hearing and slow, as is shown by the following tracing:

[Diagram of heart tracing]

Date: 9-11-34.

There is a systolic aortic murmur propagated into the neck; the blood pressure is given as 150/90, but this value is
completely discounted by the irregularity of the force of the
pulse wave, 15% being the systolic pressure of the strong wave,
30% the diastolic pressure of the weak one; and the systemo-
graphic tracing shows a slowly rising, flat-topped wave. The
evidence is therefore very strongly in favour of aortic steno-
sis, and in fact this group of signs could not be pro-
duced by any other condition, unless it be aortic aneurysm,
and that is excluded by the absence of other signs and
symptoms, by the negative Wassermann reaction, and by
the X-ray appearance of the heart.

(The nature of the apex-beat is particularly striking in
view of the associated mitral incompetence; for purposes of
comparison another apex-hanging, taken from a patient with
auricular fibrillation and pure mitral disease, chiefly
incompetence - is appended:-

| Janet Cunningham. 13.5.31. | Apex Hang. |

If confirmation of the presence of aortic disease be needed
it may be looked for and found in the X-ray picture.
In it the transverse diameter of the heart is so greatly
increased as altogether to overshadow the abnormalities
generally associated with mitral lesions. But the right
border of the heart is displaced to the right and markedly
convex, and there is also a suggestion of a prominence
on the left border, between the aortic and the left ventricular
shadow, which may represent a dilated cornu arteriosum;
that is to say, the X-ray picture is such as might be ex-
pected in a case of combined aortic stenosis and mitral in-
compentence.

From the patient’s history we conclude that the cardiac
lesion dates from the second pneumonic attack, when he was
23. For 32 years since then he has led a very careful,
well-regulated life and has been able to do his heavy daily
work. Now, for no very obvious reason, - possibly his journey
to London, possibly overstrain at work, probably unconscious, this heart has begun to fail and there is little that can be done to help him. At first there was reason to hope that rest in bed, coupled with administration of one of the digitals group of drugs (in this case strychnin), might affect a real improvement. Now illness these hopes were, is shown by a glance at the temperature chart. The pulse rate as counted at the wrist has remained fairly constant, between 70 and 100, while the ventricular rate has tended to rise almost throughout treatment. The pulse deficit was ascertained during five of the six weeks, and the average value for it was 16, 21, 26, 22.5 and 36; and these values must be taken to represent the degree of unproductive work which the heart is performing. The two pulmonary infarcts are more in the nature of unwelcome incidents than of essential features; they are almost certainly due to the passage of clots formed in the dilated right auricle into the pulmonary circulation, and while they embarrass the heart it render the patient's condition more serious. There is no obvious reason why they should recur in the near future. That is to say, the outstanding fact of prognostic significance in this case is, that while the one essential for the patient's heart is rest, the pulse deficit is very high & tending to rise rapidly. Such a condition cannot continue much longer, and the outlook is very gloomy; even under the optimum conditions of hospital treatment the patient was not even able to hold his own.
John Tulloch, act 37.

of "coronary occlusion of syphilitic aetiology."

Hechtman Prize 1935.

I. H. Brown

Contents: (i) Chart
(ii) History
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(vii) Notes on electrocardiograms.
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JOHN TULLOCH.  aged 37.  gardener. born in Ayrshire.

married.  10 Livingston Place Edinburgh.

admitted:  18. IX. 1934.

examined:  18. IX. 1934 circulatory system.

21. IX. 1934 examination completed.

Complaint: unconsciousness of 1/2 hours' duration.

History.

Present Illness. The patient felt perfectly well on the morning of 18th September; having breakfasted, he was hurrying along on his bicycle at 8 am, when "things went black in front of him." He remembers dimly looking and telling someone that he was ill; also he remembers that he was frightened by the severe palpitations that he was experiencing. He had no pain. The next thing he recollects is lying in S.O.P.D. where he received a good deal and vomited a little. From that time he began to feel a constant, dull pain in the precordial & epigastic regions. He was admitted to ward 23 at 10 am.

Previous History. Prior to the war, during which he served with the Cameron Highlanders, the patient had no illnesses of note. In 1916 he received a blow over the right eye which impaired its vision very considerably. In 1917 he suffered a compound fracture of the right leg, the result of a gunshot wound. For these two war injuries he is still receiving a 50% disability pension. In 1918 he was sentenced to two years' imprisonment for striking his superior officer, although he has never been able to recall the circumstances under which the offence occurred; on this account allegations were made that "he was mad." In 1920 he started work as a gardener & has been in regular work ever since. In 1925 he worked under a head-gardener who suffered from epilepsy and later was suffocated in a fit; this incident appears to have made a very deep impression upon.
In the patient's mind. At times since the war the patient "has not felt himself." On such occasions he gets unnecessarily excited and flies into a rage; subsequently he has to retire to bed for some days or even weeks. He believes that this has not happened for 6 years; on the last occasion the doctor was called in. I treated him for sciatica. From time to time he has suffered from giddiness and headache, which he attributed to his heavy work in the hot sun. He has never previously lost consciousness. His memory of late has become much worse; he is worried about his mental condition, but has hesitated to consult a doctor for fear of being certified insane.

His wife says that she has had an extremely difficult time with him during the past year; his character seems to have changed for the worse. The employer states that recently he gave notice because he wanted to better himself; he was going to work "where there were lots of gardeners," but could not say where.

General surroundings at home & at work are good. The patient is a non-smoker and a teetotaller.

Family. Father and mother are dead of old age. Wife is alive and well; she has had no miscarriages, but has borne 3 healthy children, the eldest being 8 or 9, the youngest 3 yrs. old.

State on Examination.
Intelligence average. Weight & height not determined on admission. A well-built, very muscular man. Face drawn and anxious, of leaden-blue color, covered with sweat; veins of face and neck dilated. Pulse is not restless. There is no oedema or jaundice. Right pupil dilated, slight ptosis of right eyelid. Temperature 97.2° F.

Circulatory System.
Patient complains of a dull constant "soreness" in the precordial...
and epigastric regions, which he attributes to the repeated pitching. He also complains of a rapid, fluttering feeling in the chest, which frightens him. He is dyspnoeic (rate on admission 52) and faint.

Pulse: artery soft-balanced, palpable, not thready. Pulse is only detected with great difficulty; it is exceedingly weak, apparently regular, and of great frequency.

Heart: The precordial area shows no abnormality. There are no visible pulsations in any part. On palpation quivering beats are just perceptible in the apical region, 2½" from the mid-line, in the 5th intercostal space. Cardiac dullness extends 1½" to the right, 4" to the left, of the mid-line, and upwards to the 3rd intercostal space.

Auscultation reveals nothing except a succession of apparently regular, excessively rapid, faint sounds, whose nature cannot be compared with normal heart sounds; they bear a resemblance to very distant first sounds at the apex. These beats are heard in all areas corresponding to the cardiac dullness.

The superficial systemic veins are dilated.

Respiratory System 21. 5. 1934.

No cough, no haemoptysis. Dyspnoea has disappeared. No pain, Inspiration rate 20 per minute. Voice clear, rattle weak.

Thorax: Thorax well-formed, very muscular, symmetrical. Respiratory movements normal & free, equal on the two sides. Vocal fremitus normal. Percussion reveals a resonant note, equal in corresponding areas; no dulness of the bases. On auscultation, vesicular breathing is heard, free from accompaniments in all areas except for an occasional coarse reiteration at the bases of the lungs.

Alimentary System 21. 5. 1934.

Appetite good. No abnormal subjective phenomena. Some teeth extracted, otherwise mouth, tongue and tonsils are healthy. Bowels tend to be constipated, but patient is in the habit of regularising them with laxative medicines.

Abdomen is well-formed, muscular. Movements normal, free.
Palpation reveals no undue resistance or tenderness. The liver is not enlarged to palpation or percussion; (on 18. IX. 1934 hepatic dulness had extended 1 1/2" below the costal margin in the nipple line); the spleen is not enlarged to palpation or percussion.

Nervous System. 21. IX. 1934.

Intelligence at first seems normal, but subsequent examination shows that the patient (as he says, contrary to his wont) has great difficulty in concentrating and in performing simple mental calculations, e.g. 100 - 7... long pause... = 93, 93 - 7... long pause... = 87. His memory appears normal. He is evidently of a brooding, introspective nature; enquiring elicited the fact that he has been afraid that the present attack was an epileptic fit. His speech is clear.

Craniocerebral Nerves: vision of right eye much impaired by injury; vision of left eye good. Examination of the right fundus is impossible owing to opacity of the refractive media. The left fundus is normal. There is slight paresis of the right eyelid. On looking to the left there is occasionally slight nystagmus. The right pupil is larger than the left, is not quite round, it reacts sluggishly to light. The left pupil is round & reacts promptly to light & accommodation. All other cranial nerves are intact.

Motor Functions: there are no abnormal movements of any kind, and no paralysis. Muscle tone good. Coordination good. There is no hemi of the tongue or face.

Reflexes: abdominal and cremasteric reflexes are absent. Elbow jerk is very brisk, right side stronger than left; wrist, knee & ankle jerks present. No knee or ankle clonus. No incontinence. no difficulties in urination.

Sensory Functions: no abnormal subjective phenomena. No loss or abnormality of sensation demonstrable.

Hemopoietic System:

No demonstrable lymphadenitis. No splenic enlargement.
Blood examination: R.B.C. 4,850,000; Hb. 106; c.r. 1.1; W.B.C.'s (on admission) 10,800. Blood Wassermann strongly positive.

Urinary System.

No abnormal subjective phenomena. Urine (on admission):

- Prick colour, acid, Sp. gr. 1.020, albumen +ve, no blood, no sugar, no acetone blood bodies, no bile, no pus; loaded with dumb-bell oxalate; granular & hyaline casts; renal and squamous epithelium.

Locomotor System.

The right leg bears the scar of a compound fracture (gun-shot wound). Otherwise no abnormality of the bones. All joints freely mobile, no pain on movement.

Integumentary System.

No abnormal subjective or objective phenomena.

Provisional Diagnosis:

Coronary occlusion of syphilitic aetiology.

Treatment as administered:

1. Absolute rest.
2. Light diet.
3. Quinidine sulphate 18. 8 0.3 gm. 4 hourly
   21. 8 0.3 gm. 6 hourly
   24. 8 0.3 gm. 12 hourly
   19. 8 Stop quinidine.

Potassium iodide: 25. 8 gr. f.i.d.

Aqu. Hydriarg. Perchlor. 19. 8 M. f.i.d.

Laboratory Test:

Blood N.P.N. 46 mgm. %

Creatinine 1.8 mgm. %.
Progress notes.

18. 2 p.m. Condition of patient quite unchanged. After the electrocardiogram contacts had been applied to the limbs, pressure was exerted on the vagus nerves in turn, without any change occurring in the cardiac rhythm. Then 0.5 gm. quinidine sulphate was injected intravenously: abruptly within half a minute, the rhythm changed. (It had been intended to record the change on an electrographic film, but unfortunately the machine failed to work, and the exact interval between the injection and the effect cannot be stated.) The pulse now became almost regular, the quality very soft and poorly propagated, rate 78. The condition of the patient improved immediately. Even before the injection was given, the epigastric & precordial pain had passed off; he now became much quieter and his colour less leaden-blue. The area of cardiac dulness was unchanged. The heart sounds were feeble, and as far as could be ascertained, pure. There was no friction to be heard. At 9 p.m. the white cell count was 10,800.

19. 12. Patient looks and feels much better. Pulse soft, regular, easily compressible, rate 74. Cardiac dulness unchanged. Heart sounds soft, pure; no friction to be heard.

W.B.C. 10 a.m. 10,800.

20. 8 Clinical condition continues to improve. W.B.C. 9,400.

21. 8. Patient states that he feels quite well & fit for work. His appearance is good. Cardiac sounds slightly stronger, pure; no friction.

22. 8. W.B.C. 8,000.


3. 8. Heart sounds much stronger, closed, quite regular. Cardiac dulness unchanged, apex beat his 4" from the mid-line.

10. 8. Condition remains unchanged, very satisfactory. W.B.C. 7,000.

14. 8. W.B.C. 8,000.
23.8. Condition apart from low blood-pressure satisfactory. Apex beat 3½" from mid-line. The 2nd aortic sound is slightly accentuated.


5.5. Patient up for first time.

7.51. Cerebro-spinal fluid examined. No excess of cells or globulin; Wassermann Reaction negative; colloidal gold: 00000 00000.

19.51. Patient is pale from long confinement to bed, and he has a rather sallow appearance. Not feeling fit for much exercise.

Radial artery easily felt, pulse wiry; artery thrumorous. Pulse very soft, easily compressed. Apex beat 4" from mid-line in 5th intercostal space. The 2nd aortic sound is definitely accentuated.

20.8. Patient discharged to convalescent home.
Notes on the Electrocardiograms.

The record taken before treatment shows an absolutely regular pulse of 77.9 beats per minute, the excursions being of very great size and their shape such as is associated with ventricular extra-systoles. There is an S or Q wave periodicity in the excursions (most clearly seen in lead I) which is clearly associated with respiration.

In the records that follow there are certain features of diagnostic value, concerned chiefly with the form of the ventricular complexes. At the same time there is a remarkable change in their duration; these changes are here set out in graphical form & attention is drawn to their relationship to quinidine medication.

Ventricular Systole and Quinidine

The second record was taken immediately after the injection of quinidine sulphate. In it the rate is 69 per minute (lead II), not quite regular; the duration of the PR interval and of the QRS complex (both of which remain constant throughout the whole series of records) are 0.16" and 0.08" respectively. In leads II & III the S-T interval...
shows a plateau effect (failure to return to the iso-electric line) in the T-wave is inverted, sharply pointed and, particularly in lead III, of large size. In this record, too, as in all subsequent ones, lead III shows a bifid R-wave.

The record taken on the following day, 19. 15. 24, shows a large R-wave in lead I and a large S-wave in lead III. In other respects it is different in degree rather than in quality from the former record. In leads II & III the 3-T interval still shows the plateau effect and the T-wave remains inverted; the latter is very large and prolonged, though now it is rounded instead of pointed; also in lead I it is enormously prolonged.

On 20. 15. there was no material change, except that the T-wave tends to be even larger; on 21. 15. there is a tendency to return to the sharply pointed form of T-wave noticed on 13. 15.; this is particularly the case in leads II & III.

By 24. 15. the excursion of the T-wave is much reduced & more rounded in leads II & III; on the following day the pointed form is once more prominent, but both the magnitude of the excursion and the extent of the plateau effect are obviously reduced. These tendencies persist through the succeeding records until on Oct. 1 at the T-wave in lead II is ambiguous and on Oct. 15 becomes upright, though it is still inverted in lead III. The record of B. 15. is almost identical with the previous one.

On 9. 15. amyl nitrite inhalation was tried, with the result that the pulse rate was temporarily raised to 120 and the T-wave in lead II became upright; a minute later the rate was reduced to 69 and the T-wave absent, evidently a transitional stage between upright and inverted form.

A fortnight later the T-waves in leads I and III have become much more pronounced and are more pointed, that in lead III still being inverted; in lead II the T-wave is smaller; that is, to say, all three leads show a tendency on the part of the T-wave to revert towards an earlier form. On 27. 15. these changes are once more less in evidence; the
Amyl nitrite experiment which was carried out shows no change of interest (lead III). The records taken before and after the inhalation are almost identical.

The last record, taken on 10. XII. 34, shows two chief deviations from the normal; the inverted T-wave in lead III, and the large R-wave in lead I and the large S-wave in lead III.

After his discharge to a convalescent home, the patient returned on 4. XII. 34., to have an E.C.G. tracing taken. It shows a T-wave of similar kind, but much more sharply pointed than that of the previous record. The duration of ventricular systole is 0.32 sec.
Commentary.

There is little in his history prior to the date of admission to suggest that this patient was the victim of serious disease. Feeling well, and performing his strenuous daily duties as a gardener, he was in a moment struck down and brought very near to death. The fact that his first symptoms were palpitation and faintness, succeeded by syncope, is sufficient to make us look in the first place to the heart for the source of the trouble; and that we are correct in doing so is proved by the pulse rate of 232, a rate which never occurs except as the result of a cardiac lesion, which is sufficient to account for the first symptoms. The suddenness of onset, the palpitation, the regularity of the rapid pulse, are characteristic of paroxysmal tachycardia, an abnormal cardiac rhythm which may originate in the atria, the auriculo-ventricular junctional tissue, or the ventricles; to decide between the three from clinical observation alone would be very difficult if not impossible. But by the time the patient reached hospital he had not only his initial symptoms of collapse and palpitation, but also nausea and vomiting, precordial and epigastric pain, dyspnoea, restlessness, which, taken together, suggested very strongly that the abnormal stimulus would be found to be due to thrombosis of a branch of one of the coronary arteries. Experience tells us that generally a part of the left ventricular wall is affected.

To establish a diagnosis of coronary thrombosis it is generally necessary to exclude a number of other conditions, mostly extracardiac, and these require no further discussion here. There remains one important alternative, angina pectoris. This is more likely to occur during hard physical labour than coronary thrombosis is; against it are the delay in the onset and the duration of the pain; the rate and the quality of the pulse; the blood pressure; the sweating; the vomiting; the state of collapse; the dyspnoea and the restlessness.
On clinical grounds alone, therefore, the decision rested with coro-
nary thrombosis.

The first attempt at restoring a normal rhythm — by
oral administration — was entirely ineffectual, as might be ex-
pected when the lesion lies in the ventricle. Next, 0.3 gm.
of quinidine sulphate was injected intravenously, and the
result was an abrupt transition to an almost regular, though
naturally very weak pulse, of 70 beats per
minute. The quinidine, by virtue of its power to render
cardiac tissue less sensitive, had inhibited the spread of
the presumably persistent abnormal stimulus, without
hindering the propagation of the normal one. The new
rhythm persisted, the blood pressure gradually rose, & the
patient's condition was enormously improved.

In the further course of coronary thrombosis there are
certain clinical signs to be looked for: — pericarditis, which
was not found in this case; pyrexia, — here the temperature
was raised to 100.4° F. on the evening of admission &
returned to normal within three days; embolism, which
happily did not occur; leukocytosis, of which we can
judge in this case by the following chart:

![Graph showing leukocyte count over time](image)

It will be seen that from the time of the first count, 12
hours after the catastrophe, there was a steady full
amounting to 25%. The maximum rise generally occurs
some days after the thrombosis, but Libman & Sachs
believe that the leukocytosis may commence within 1% hours,
so that the above curve may have some significance.
The electrocardiographic records are of great interest. It has been mentioned that the commonest seat of coronary infarction is the wall of the left ventricle, and it would be interesting to be able to deduce from the first record whether or not the form of the excursions proved the extra systoles to arise there; for it is very probable that the abnormal stimulus arises at the seat of the lesion. Unfortunately the analogy of their form with that of other known extra systoles is too slender a basis for decision.

The subsequent records show all the classical signs associated with coronary thrombosis, namely, the plateau effect of the S-T interval and the inverted T-waves in lead II and III, the T-waves being also sharply pointed. The modification of these abnormalities from day to day indicates a gradual return towards more normal conditions. The accepted interpretation of the amyl nitrite test as carried out on Sept. 28th is that the degree of normality attained is a measure of the potential recovery of the infarcted area; the fact that the T-wave becomes upright in lead III (being already upright in lead II) is at once a promising sign and an indication that the area of infarction was relatively small.

As has been pointed out, the duration of the P-R interval, that is, of the auriculo-ventricular conduction period, remained normal and constant throughout (0.16"), whereas the ventricular complex varied in duration between a maximum of 0.67" and a minimum of 0.32." Parallel with these changes there was a partially illusory change between the P-R and the R-T more pointed forms of T-wave; the more pointed forms accompany a sudden change from the longer to the shorter duration of the deflection. These changes in duration are set out in a graph given above and associated with the changes in the administered doses of quinidine. This isolated case proves nothing, but it does suggest that quinidine has little or no effect on the functional tachycardia, but depresses the myocardial digitalis on the other hand is known to depress the finishing
times and to stimulate the myocardium.

Up to now we have considered the clinical diagnosis and its confirmation by the results of treatment and by the electrocardiograms. It remains to find out why an apparently healthy young man should suffer from coronary occlusion.

It is known that this condition is rare before the age of 40, and that the commonest predisposing cause is atherosclerotic degeneration of the coronary arteries. This man is 37 years old, and, for what it is worth, the state of his peripheral arteries affords no evidence of arterial disease elsewhere. Embolism is a rare cause, and this man presents no signs or symptoms which would justify the assumption that an embolus had been responsible for his trouble. He has a strongly positive Wassermann reaction; Parkinson & Bedford state that less than 10% of all cases of coronary occlusion are due to syphilis; actually in their first 51 cases that came to post-mortem examination 7 were due to syphilis, and an area of thrombosis was demonstrable in only two out of these seven. Should this man's condition be due to syphilis, then we may explain the fact of his accident occurring during physical exertion instead of as usual during rest on the assumption that the months of his coronary arteries are constricted by the lesions of syphilitic aortitis and therefore unable to meet the demand of the myocardium for a maximum blood supply. Dietrich of Tübingen states that the commonest cause of death in the street of a man under 50 is coronary occlusion due to syphilitic aortitis, and it is a justifiable conclusion that this man came within an ace of swelling Dietrich's majority.

The prognosis depends on three main factors: the degree of damage done to the heart by the present accident; the course of the postulated syphilitic aortitis;
and the possibility of other syphilitic manifestations arising elsewhere.

We have already seen that the result of the first amyl nitrite experiment indicated a small area of organic damage and a high degree of potential recovery; often satisfactory features are the rapid defervescence & recovery of the patient; the absence of any very marked leucocytosis, and the lack of evidence suggesting that the damaged heart wall was seriously yielding freely to the pressure within. The apex beat was never more than 4" from the mid-line and scarcely varied in position. On the other hand the x-ray shows a heart which is decidedly enlarged in the transverse diameter, which cannot be accounted for by ventricular hypertrophy or as due to raised blood-pressure. In the whole, it seems not improbable that the necrotic area of the heart will be transformed into a healthy scar & that by now there is little danger of aneurysmal dilatation and rupture of the ventricle; that is to say, as regards the present change in the heart, the prognosis is as favorable as may be after such a serious accident.

As to the second factor in the prognosis, the further course of the syphilitic aortitis, the three chief dangers are aneurysmal dilatation of the aorta, further occlusion of the mouths of the coronary arteries, and the development of aortic incompetence. The last is the commonest of these and the most frequent lethal consequence of syphilis; and as in this case we have reason to believe that the syphilitic process already implicates the coronary arteries, it may well spread to the aortic valves. Further occlusion of the arteries and sudden death have not infrequently resulted from the injudicious use of anti-syphilitic remedies, the first effect of which may be to produce an increase of size in all specific lesions. The screening under x-rays proves that at present there is no aneurysm of the aorta. It is to be noted that the electro-
cardiograms show the changes (large R-wave in lead I, large S-wave in lead III) commonly interpreted as indicating left ventricular preponderance, which is not easily explained on the basis of the clinical findings. It seems not unlikely that there is a certain degree of chronic interstitial myocarditis, arising either as a result of the gradual occlusion of the coronary arteries, or because of active syphilis of the myocardium; for it is known that that condition may give rise to similar changes in the electrocardiogram.

The third factor is the course of the syphilitic disease elsewhere. Various points were noted - the history of headaches, the change of temperament, the fits of temper, his optimism as to finding a job "where there are lots of gardeners," the recently developed inability to perform even the simplest mathematical calculations - which gave rise to the fear that even now he might be suffering from the earliest stages of general paralysis; fortunately this suspicion was not confirmed by the examination of the cerebrospinal fluid, which showed no deviation from the normal. It would have been particularly serious in his case, as his cardiac condition would preclude the possibility of employing the most potent therapeutic measure against neurosyphilitic malarial inoculation.

With constant, cautious, anti-syphilitic treatment I may well be possible to arrest the progress of the evil, but sufficient damage has already occurred to jeopardize his patient's life, for physical exertion which has hitherto been his daily habit might at any time be fatal.
Lead II. At conclusion of inhalation.

End of Amyl Nitrites

About one minute later.
Before Inhalation.

At conclusion of inhalation. (Lead II)

After inhalation.
Case of acute pericarditis of rheumatic origin.

Wightman Prize 1935

T. H. Brown

Contents:
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(ii) History
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Complaint: precordial pain of 3 weeks' duration.

History.

Present Illness. Three weeks ago the patient began to experience a sharp, stabbing pain in the precordial area. It was severe, constantly present by day and by night, worse on exertion and on stooping. Three days before admission the pain became much worse; while at his sister's house he fell asleep in a chair, and it was noticed that during sleep his left arm and leg and the left side of his face were twitching. This day he almost collapsed because of the pain and weakness, and returning home he went to bed, where he has remained ever since. On the day before admission he was seen by a doctor, who diagnosed acute pericarditis, and sent him to R.I.E. for admission. On the day of admission the pain was no longer confined to the precordium but radiated towards the left clavicle.

During the three weeks of illness the patient has felt very tired, has had no appetite, slept badly and sweated very freely; he has had no rigors. He has also become noticeably hoarse. His legs have not been swollen. He has had no difficulty in swallowing. He has been very breathless.

Previous Illnesses. The boy states that he has always been a healthy but not very strong. He has always taken his full share in games. He has never been confined to his bed, so far as he knows, except once nine months ago, on account of a cold. He has sometimes suffered from sore throat, but they were not sufficient to make him lie up. He has never had chorea or rheumatic fever or growing pains.

At home he is obviously neglected; his condition on admission proves that and it has not been possible to obtain any useful information.
information from his relatives.
This mother died some years ago as the result of a haemorrhage; his father has married again. He is the youngest of his mother's children; he has no brothers and all his sisters are married.

Statement on Examination.
Intelligence below normal. Patient states that he never reads, but is often led into mischief. On account of his condition he was neither weighed nor measured on admission; he is very thin & very dirty. He is fairly well developed, but has very little muscle. His face wears a fixed smile which appears to be due to pain. He is pale & cyanotic, and his forehead is covered with sweat; he seems very weak and ill. His face is grayish and his lips blue. Temperature 103°: pulse 114: respirations 30.
Circulatory System.
There is severe sharp pain in the precordium radiating towards the left clavicle; it is constantly present, and worse when the patient attempts to lie on his side. There is no palpitation and no faintness. He is breathless, but has no cough.

The artery wall is not palpable. Pulse rate 114, regular in time and force; wave very weak, scarcely perceptible, thready. It was impossible to get any tracing with the sphygmograph. Blood pressure 80/60.
Heart: the precordium is normally developed. There are no visible pulsations at the apex; there is feeble pulsation visible in the root of the neck. No true apex beat is palpable; there is a feeble diffuse flicker palpable in the 5th intercostal space, 3" from the mid-line. The area of dulness to percussion extends 2" to the right and 5" to the left of the mid-line; the upper border lies at the 3rd rib. Auscultation reveals very feeble heart sounds, at times scarcely audible; regular in rhythm; there are no murmurs audible.
at any point. Louder than the heart sounds, and to a large extent obscuring them, is a loud to-and-fro friction sound, audible over an area lying to the left of the sternum, from the 3rd intercostal space down to the 5th space, and extending 2" to the left of the mid-line. The sounds become slightly louder when pressure is applied to the stethoscope, and they give the impression of being superficial.

The veins of the neck are distended, even when the patient sits up; they pulsate feebly.

Respiratory System.

Patient complains of breathlessness, not of cough or expectorin, nor of either than the precordial pain. Breathing shallow, rapid, regular, rate 30 per min. Voice is full and hoarse. Laryngoscopic examination was omitted in the patient's interest.

Thorax is normally developed, symmetrical, extremely poorly covered with muscle or fat. Movements are slight but free. 

Vocal fremitus is found to be normal and equal on the two sides. On percussion the note is found to be impaired.

J. MEG. III
on the left side posteriorly, from the lower border of the lung to the level of the 5th rib, also on the lateral surface of the chest from the lower border of the lung to the axilla. The degree of dullness decreases from the lower border upwards, being very marked below. Over the same area there is a slightly increased sense of resistance on percussion. On the right side there is no impairment of the percussion note. On auscultation the breath sounds are found to be harsh vesicular without crepitations or rhonchi over the whole right lung and the anterior surface of the left lung; on the lateral & posterior surfaces of the left lung there is medium or high pitched bronchial breathing with occasional fine and more numerous medium crepitations.

Over the lower part of the left lung there is very marked whirring pectoriloquy.

Alimentary System.

Appetite is poor at present, otherwise good. Lips, gums, teeth are healthy. Tongue is heavily coated. Tonsils are not enlarged and apparently healthy. There has been no vomiting. Patient is at present constipated, bowels are normally regular. Abdomen thin; movements free; there is no resistance or tenderness. Liver is not enlarged to palpation or percussion, neither is the spleen. There is no free fluid in the abdomen.

Haematopoietic System.

Tonsillar glands slightly swollen, quite firm. No other glands palpable. Blood examination: R.B.C. 3,590,000; Ht. 65%; C.1. 0.9; W.B.C. 14,000.

Urinary System.

No abnormal subjective phenomena. Patient has not noted that he has been passing less urine than usual. Urine: pale amber, acid, sp. gr. 1027, no albumen, no sugar, no acetone bodies, no bile, no pus, no R.B.C.'s.

Locomotory System.

No osseous deformities. Joints are all freely mobile, both passively and actively. No swelling of the joints, no pain.
Integumentary System.

Patient complains of much itching at all times. Skin at time of examination was dry, scaly, of atrophic appearance. There are numerous scratch marks distributed generally over the body, many of them being pulsatile. There is no oedema. There are no rheumatic subcutaneous nodules or other abnormalities.

Nervous System.

Intelligence rating below average. Patient is inclined to be unoccupied and dreamy in his bed, "thinking." He says he was not good at school work. Sleep has been bad of late, otherwise it is good.

Fields of vision normal. Fundus oculi normal on both sides. Eye movements intact. No hypertonus. Pupils equal, round; react promptly to light and accommodation.

Patient's face is inclined to twitch, but observation seems to indicate that this is a manifestation of pain, hearing normal.

Motor functions intact. There are no abnormal movements of any kind. There is no paralysis.

All superficial & deep reflexes equal on the two sides, normal. There are no abnormal subjective sensory phenomena, apart from headache, which has been intermittent since the beginning of the illness.

Provisional Diagnosis.

Acute pericarditis of rheumatic origin.
Treatment as administered.

(1) Complete rest.
(2) Diet. 12. milk food, soup, jellies, 2-hourly.
       16. small light diet.
(3) Medication. 11. to 16. Torict. Digit. M 15 t.i.d.
       11. Sp. Bismuth 3 g as needed.
       23. - 16. Parrish's syrup 3 g t.i.d.
       23. - 16. Cod liver oil & malt 3 g t.i.d.

Laboratory tests.

Examination of the pericardial fluid showed many R.B.C.'s.
with lymphocytes and large mononuclear cells, a few polymor-
phs; no organisms were found. On staining, and no
growth obtained on culture.

Further blood examinations:
19. W.B.C.'p 14,000 polymorphs 58% lymphocytes 23% monocytes 15% eosinophils 3% basophils 0.5%.
24. W.B.C.'p 9,400
4. W.B.C.'p 6,400

Urine: specific gravity was always about 1020, reaction
sometimes acid, sometimes alkaline; no abnormal con-
stituents were found.

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Progress Notes.

12.45. Patient appears very ill. Heart & friction sounds unchanged. The pericardium was tapped and a few cc. of blood stained fluid withdrawn.

13.45. Cardiac dulness extends 2" further to right and 1" further to the left than on admission. Electrocardiogram shows prolongation of the P-R interval to 0.20" and an R-deflection of broad base and low voltage. The T-wave is inverted in all three leads. On percussion there is a tympanitic note over the lower left ribs anteriorly. X-Ray examination shows this to be due to a sub-diaphragmatic collection of gas.

(On reproduction
The x-ray photograph was reversed)

14.45. Patient appears no less ill than before but complains less of pain. Cardiac dulness has extended still further towards the axilla. Friction sounds are most readily audible in the neighbourhood of the punctured wound made when the pericardium was tapped. It was noticed today for the first time that the patient gave off a
18. The patient appears somewhat better. Cardiac dulness is of smaller extent, 1" less to the left. The electrocardiogram shows changes similar to the earlier ones, except that the PR interval is further increased to 0.24".

19. General appearance much improved. No complaint of pain except when pressure is applied with the stethoscope. The dulness over the lungs has altered, so that the maximum changes (also in breath sounds) extend 2"-4" from the spine from the level of the 5th to the 7th ribs. Whispering pectoriloquy is also most marked in this area.

20. Cardiac dulness extends 1/2" to the right and 1/2" to the left of the median plane. No rub is any longer to be heard. Blood pressure has fallen to 78/44.

21. Cardiac dulness slightly increased in area. Friction sound audible faintly. Heart sounds pure, closed, very feeble.

22. X-ray photograph shows marked reduction of the cardiac shadow.
25. E.C.G. similar to that of 18. E.
27. E. In initial area a soft systolic murmur is audible, not propagated.

2.E. Lung condition improved. Whispering pectoriloquy no longer marked, no accompaniments.
Scales diagnosed and treatment commenced.
11. E. E.C.G. shows large excursions in all leads. Cardiac dulness 1" to right, 3" to left of mid-line. P-R interval reduced to 0.20". T-wave is upright in lead III but remains inverted in leads I and II. X-Ray shows very marked reduction of the cardiac shadow.

15. E. Apex beat is visible 3" from mid-line in 5th intercostal space. With each systole there is a rippling pulsation of the parasternal portions of the 2nd & 4th spaces, and the apex is an outward thrust. Its position changes with change of position of patient. In the initial area the weak, short first sound is very slightly roughened. Dulness over the upper part of the lower left lobe has disappeared, and the bronchial breathing & whispering pectoriloquy are very much reduced.
Commentary.

The history of this patient, unsatisfactory as it is; his symptoms and the clinical findings, all point to the existence at the time of admission of pericarditis with effusion, and the supposition is finally substantiated by x-ray examination and by the withdrawal of fluid from the pericardial sac. If the condition had been of long standing it would not have cleared up so quickly and satisfactorily as it did, and we are justified in assuming that it commenced three weeks before admission with the onset of symptoms, that is to say, the pericarditis may be termed acute or sub-acute.

By far the commonest cause of acute pericarditis is acute or sub-acute rheumatism; but before concluding that rheumatism is responsible in this case it is necessary to exclude all other possible causes and to find positive evidence in its favour.

Pericarditis may arise as a sequel to acute infections such as pneumonia and scarlet fever, or in conjunction with sepsis. In any of these three conditions the pericardial fluid would probably have contained a great number of polymorphonuclear cells, and might have yielded growth on culture. There is nothing in the history sufficiently dramatic to suggest pneumonia, and scarlet fever would have been ushered in by vomiting and sore throat, both of which were absent here. The skin sepsis which this boy had as a result of his scabies might have given rise to pericarditis, but the temperature chart is certainly not suggestive of severe pyogenic infection.

The character of the effusion was consistent with the diagnosis of tuberculous pericarditis, and, as was virtually certain to be the case, the skin tuberculin test (carried out in the 1st by Alice Institute) was
positive, proving that the boy had been infected with the tubercle bacillus; but the rapid resorption of the fluid and the very satisfactory progress of the patient definitely negative the possibility of this having been a tuberculous effusion.

Obviously the boy's condition cannot be ascribed to a penetrating wound of the pericardium; nor did it arise as the result of the direct spread of infection, as in empyema, ulceration of the oesophagus, subphrenic abscess, or similar condition. It is equally clear that haemorrhage, chronic nephritis and diabetes may be excluded.

Embolism of a branch of a coronary artery may cause pericarditic friction, and the source of an embolus in this case might well have been the vegetations so commonly found on the mitral & aortic valves in acute rheumatism. The electrocardiograms in this case might be regarded as supporting such a hypothesis, for in leads I and II the T-wave is inverted throughout, and in lead III it is at first ambiguous, later upright; these facts however are of no value because similar changes are frequently found in pericarditis of known rheumatic origin when complicated by effusion (Scott, Neil, R.J., Am. Heart J. V; Porter & Pardee, Am. Heart J. XIV) Very strongly against embolism in this case are the gradual onset of symptoms, the lack of evidence of valvular involvement, and the absence of suggestion of embolism occurring elsewhere. (The twitching of the left side of the body, noticed during sleep, before admission to hospital, cannot have been due to embolism; there was no suggestion of any organic lesion of the brain.)

Other causes therefore being considered improbable or impossible, what positive evidence is there of rheumatic infection?
Apart from carditis the organic changes most commonly demonstrable in the course of rheumatic infection are:
tonsillitis, arthritis and "growing pains," chorea, various erythematous and subcutaneous nodules; and in this case none of these was found, even when searched for, so that it must be conceded that the course of the illness, if it was rheumatic, was an unusual one. Furthermore, as regards carditis, endocarditis is more commonly demonstrable than myocarditis, myocarditis than pericarditis, yet in this instance pericarditis was by far the most striking fact, and the presence of endocarditis was at no time even suspected. Not even the nature of the pericardial fluid was typical of rheumatism, for it was haemorrhagic in stead of clear, though it may be noted that similar haemorrhagic fluid was found in one of Porte & Parade's cases that came to post mortem examination.

It is in such an obscure case as this that the electrocardiogram may be invaluable. Lüdowski (Beut. Archiv für klin. Med. LXXIV 1932-33) analyses the electrocardiograms of 100 patients suffering from acute rheumatism, and comes up with observations under 10 headings.

1) Alteration in pulse rate. He found that tachycardia was relatively uncommon; 52 of his cases showed a pulse rate constantly below 90 and only 3 reached 120. One patient's pulse rate reached 120 on the first two days only, and this rate certainly cannot be considered excessive in view of his temperature, his toxic state, and the extent of his pericardial effusion.

2) Sinus arrhythmia was observed in 20 cases, in 14 of them during the acute stage when the temperature was still raised (over 38.0° C. in three cases, under 38.0° C. in one case); and Lüdowski put forward the hypothesis that there may be organic changes in the vicinity of the sino-auricular node in such cases, making it peculiarly susceptible to
the influence of the extra-cardiac nervous apparatus. Be
ting as it may, our patient's second electrocardiogram,
taken when his temperature was still raised and his
pulse rate 82, and when the pericardial effusion was
only just past its maximum, shows the irregularity char-
acteristic of sinus arrhythmia.

(2) Dissociation with interference, which Lukomski found in
7 of his cases, was not observed in our patient.

(4) Noval rhythm was not observed.

(5) Lukomski found partial heart block in 58% of his
cases, and he lays great stress on this as a diagnostic
point. He believes that it may be the first recognisable
sign of acute rheumatism, and he had the good fortune
to have two patients under observation before polyarthritis
had appeared or acute rheumatism was suspected, in both
of whom the P-R interval was found to be 0.32” on the
day that joint symptoms first appeared. He says that
partial heart block should be of great value in estab-
lishing the diagnosis of acute rheumatism in the absence
of arthritis, - "a condition which is often misinterpreted
and which should be just as readily recognised by
clinicians as those forms in which the joints are im-
plicated." Our patient showed a P-R interval on the
occasions of his four electrocardiograms of 0.20", 0.24", 0.24",
and 0.20", all of these values being abnormally long,
and only the first occurring during digitalis medication.
As in the cases of sinus arrhythmia, Lukomski
believes that these abnormalities are due to organic
changes in the conducting tissues, rendering them ab-
normally susceptible to nervous control; the condition
cannot have been entirely functional, for it was some-
times reduced, but never abolished, by the administra-
tion of atropine.

(6) Extrasytoles were observed in only 16 cases; in 8 they
were of auricular type, and sometimes aberrant, in 8 of

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ventricular type. In our case there is no record of extra
syptoms, but the first ventricular contraction in lead I
of the first electrocardiogram is of aberrant type.

(7) Auricular fibrillation or flutter did not occur.

Abnormalities

(8) Changes in the initial ventricular complex were fre-
quently observed, but Duboski considers that no such
changes can be ascribed to acute rheumatisation, if they
are not observed to arise in the course of the disease.
In our case, the form of the initial ventricular de-
flexion varies constantly from one record to the next.

(9) Changes in the T-wave and the S-T interval were re-
peatedly observed, and Duboski believes that they are
due to the presence of Koch's nodules in the myo-
cardium, but mentions Porte & Pardee's description of
inverted T-waves in three cases of acute rheumatisation
with pericarditis. The cases in which Duboski observed
these changes showed no evidence of pericarditis, and
conversely, those of his patients who had pericarditis
did not show the change. The electrocardiograms of
our patient shows inverted T-waves, constant in lead
I and II, inconsistent in lead III.

(10) Changes in the P-waves were seen in various cases;
in ten cases without nodal rhythm the P-wave was
inverted in lead III. In our case the P-wave is inverted
in lead III in the first, third and fourth electrocardiograms,
ambiguous in the second.

In attempting to assess the value of these observa-
tions, it is to be noted that whereas Duboski is
trying to establish electrocardiographic laws in the
light of an established diagnosis, we are attempting
to establish a diagnosis from the electrocardiograms.
Until very complete analyses of the changes in the
electrocardiograms in all forms of heart disease
are available, in particular until we know what
electrocardiographic changes to expect in various
form of pericarditis with effusion, including "idiopathic
pericarditis," if such a thing exists, there must remain
a certain element of doubt about the diagnosis. Never-
theless this fact cannot be devoid of significance, that
pains, arrhythmia during fever, partial heart block,
and inversion of the P and T waves were present in
the case of our patient, and that these were among
the commonest abnormalities which Bukowsky noted,
although only two of his 100 patients showed evidence
of pericarditis. It is certain that Bukowsky at least
would be compelled to agree with the diagnosis of
"acute pericarditis of rheumatic origin."

A final point of interest in connexion with the diagnosis
is that this boy was noticed, not on admission, when his
symptoms might have been responsible, but four days
later, to give off the monosy on which some authorities
have held to be indicative of rheumatic infection.

Summing up the course of the illness we may say
that during the first week there was a rapid accumulation
of fluid while the temperature fell; subsequently the fluid
was very largely absorbed and the condition of the patient
improved greatly. The signs of pulmonary compression were
noted to disappear much more rapidly at the base than at
the upper part of the lower left lobe, but the final result
was satisfactory. One most unexpected feature was that
during the period of maximal effusion the blood pressure was
higher than at any time before or after, and it is impossible
to be certain why this should have been so. One possible
explanation is that the rise was due to a reflex mechanism,
originating in the myocardium; the mechanical disadvantage
under which the heart was labouring made a satisfactory
coronary circulation more difficult of attainment and more
necessary than ever, the only way of meeting requirements
being by raising the arterial pressure. This would imply
that, had the pressure not risen, the patient would have

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died of myocardial failure. This explanation might
similarly apply to the rise of blood pressure which occurs
in angina pectoris.

The prognosis depends partly on the fact that an
attack of acute rheumatism predisposes to another; it
depends also on the degree of permanent damage in
the myocardium; and it depends on the change in the
pericardium. Lewis states that simple obliteration of the
sac (which may well be present here) is of no signi-
ficance; on the other hand he says that constrictive
pericarditis and mediastinal pericarditis do burden the
heart and may be very serious. He believes that they
arise when the inflammation has extended beyond
the pericardium to the surrounding tissues; but as he
also says that the same extension is responsible for
the pain in pericarditis, it follows that one or other
of these two sequels may be found in our case. At
the time of the patient’s discharge the various signs
which would indicate their presence — systolic retraction;
immobile impulse, venous distention, pulsed paradoxus,
— were not detected, but it is too early to be certain
that they may not appear later. Bearing all these
points in mind, the immediate prognosis must be
considered to be fair, the ultimate prognosis uncertain.
The immediate need is good nursing and prolonged
convalescent treatment.
Appendix (11th. xii. 1931).

The patient was seen today at the Ashby Leysley Institute, just two months after his discharge. His general appearance is such that it was difficult to recognize him. He is getting up every day and enjoying his "occupational therapy" (painting pictures).

The doctor in charge is entirely satisfied with his progress. His colour is excellent and his body well covered with fat. His temperature is normal and his pulse rate about 80 and tending to fall. His weight is 6st. 1½ lbs. - a gain of 7½ lbs. during the past four weeks. His voice is no longer hoarse. His good spirits are such that he is sometimes difficult to restrain.

The apex beat is in the 5th intercostal space, 4" from the mid-line; the right border (deep dulness) extends 1½" to the right of the mid-line. The heart sounds are pure and closed; there is no friction to be detected. The lungs show no abnormality.

An X-ray picture (taken recently) shows that the heart is still slightly increased in its transverse diameter, but the angles at which it meets the diaphragm are now acute, not obtuse as hitherto.
aortic incompetence due to syphilitic aortitis

Wrightman Prize 1935

T. H. Brown

Contents: (i) chart
          (ii) history
          (iii) state on examination
          (iv) treatment
          (v) progress notes
          (vi) x-ray and electrocardiogram
          (vii) commentary
JOHN GEAR. aged 55. Coal miner, born in Lancashire.
moved. admitted 9. 8. 1934
examined 9. 8. 1934.

Complaint. Gradual onset of breathlessness during the past year; extreme breathlessness on exertion, weakness and palpitation since 16. 8. 1934.

History.

Present Illness. Ever since the winter of 1933-34 the patient has noticed a tendency to excessive breathlessness on exertion. From the middle of July he really felt ill, but remained at work until 16. 8. 1934 when, having dressed in the morning he found himself too breathless and feeble to go to his job. He retired to bed and stayed there for three weeks, suffering from these complaints and from palpitations; when he got up he was not able to walk more than two or three hundred yards slowly on level ground without stopping to regain his breath. Each afternoon since then he has spent in bed. During the past two months he has suffered from dizziness 'like a black curtain in front of his eyes' on exertion; it never comes when he is lying down. During the past three weeks he has been troubled with persistent cough which is generally worse at night; an attack of coughing is accompanied by pain in the epigastrium and a choking sensation in the throat. The sputum he has is clear and frothy. He has become hoarse of late. There has been no dysphagia. He still suffers from palpitations, and when he lies down his whole head seems to throb; there has been no headache. Of late he has been unable to think so clearly as when in health. About two weeks ago, while seated, he noticed for the first time pain of a constant sore character between the shoulders; it did not radiate
and soon passed off, but has returned since then from time to time. There has been no pain in the thoracic or precordial region. He generally feels better when up than in bed, and in the morning than in the evening. He has been medically examined by an official of the Glasgow State Insurance Board, who informed him that he would never be fit for work again.

Previous Illnesses. At the age of 7 years patient had scarlet fever. He served in France and Gallipoli with the K.O.S.B. ; in Gallipoli he suffered from dysentery and was finally sent home from France in 1916 suffering from shell-shock. He has never suffered from any other serious illnesses, but since the age of 50 has begun to feel old. He feels overworked, and believes that his work underground has been too much for him.

He has been in regular work ever since the war. The house in which he lives is roomy and good. He used to take too much alcohol on occasion, but never during the past few years. He is a non-smoker.

His wife is well; of their eleven children one died in childhood, another has had valvular disease of the heart for 7 years; all the others, of whom the youngest is 19 years old, are alive and well.

State on Examination.

Intelligence average. Height 5'11½". Weight 104½. 129½ lbs.


Circulatory System.

No pain in the cardiac region. Palpitation and dyspnoea are experienced on slight exertion; breathlessness persists when the patient is in bed. There is a persistent loose cough.

Pulse: artery not hypertrophied or tortuous. Rate 98.

J.G. II
regular in time and force, wave strong, very poorly sustained (i.e., of water hammer type). Pulsations are felt wherever the hand is laid on the body, as for instance on the vertex, and at the cubital fossa it is easy to hear each pulse-wave, when the stethoscope is lightly applied. With every heart beat the whole arm throbbs, and the entire length of the brachial and radial arteries stands out under the skin. The radial pulse is synchronous and equal in force. The sphygmonanometric reading is 136/76, but the finger receives the impression of a considerably higher systolic pressure. Tracing from the radial pulse:

Heart
The lower part of the sternum is slightly depressed, precordium is otherwise well formed. Pulsations are readily visible in the precordial area but not more than 3” to the left of the mid-line. The whole epigastrium and left hypochondrium quivers with each systole, and pulsations are easily visible, still more easily palpable at the root of the neck. The apex beat is felt in the 6th intercostal space, 6” to the left of the mid-line; there is no palpable thrill in any region. The area of deep cardiac dulness extends 1½” to the right and 6” to the left of the mid-line, and upwards to the 3rd intercostal space. Auscultation is rendered difficult by the rapid breathing and loud phonchi audible in all parts. In the mitral area the first sound is a brief soft murmur, followed a long-drawn, harsh, blowing, systolic murmur. In the aortic area there is a loud, blowing, harsh to-and-fro sound, the diastolic part of which is propagated over the whole cardiac area, most strongly towards the lower left part of the sternum, the systolic part being slightly propagated.
into the neck. In the tricuspid area the first sound is
closed, the 2nd sound is obscured by the propagation of the
aortic diastolic murmur. The 2nd pulmonary sound is
not accentuated. There is no venous congestion. Capillary
pulsation is demonstrable with difficulty.
Respiratory System.
There is persistent and troublesome loose cough; no
haemoptysis, no pain on respiration. There is evident
dyspnoea (frequency 30). Voice a little lisping. No spittle
is available for examination.
Thorax: well-formed except for a slight depression of the
lower end of the sternum. Inspiratory movements are
short and catchings, expiratory slow and wheezy. There
is no tracheal tug. With each inspiration the auxiliary
muscles of respiration are brought into play. Respiratory
excursions are free over the right apex than over the
left. Vocal fremitus is readily felt on the right side, less
readily on the left. Phrenicobrachial fremitus is readily felt
in all areas. Anteriorly the percussion note is even
where resonant and is equal in corresponding areas;
posteriorly the upper half of the right lung gives a
relatively dull note. On auscultation it is found that
the normal breath sounds are entirely obscured by
loud, musical rhonchi; inspiration is short, expiration
very prolonged. There are also some medium and coarse
crepitations to be heard in all parts, the loudest and
coarsest being in the region of the right hilum anteriorly,
the finer and most numerous crepitations at the base of the
left lung posteriorly. There is no silent period between the
end of expiration and the beginning
of inspiration.
Alimentary System.
Appetite fair. There are no abnormal subjective phenomena.
Teeth are all extracted. Tongue moist, slightly coated.
Pharynx congested, purplish colour. Tonsils healthy. Patient
J. G. IV
is in the habit of regulating his bowel with laxative medicines. Abdomen is muscular, with considerable adipose layer. There is some tenderness in the region of the gall-bladder. There is no undue resistance nor any fluctuation. The liver and spleen are not enlarged to palpation or percussion.

Haemopoietic System.


Integumentary System.

Skin is moist. About the knees there are extensive brown areas as though freckled, which the patient states to have been present since birth. There is no oedema. The fact on the abdomen gives the impression of being abnormally lobulated.

Urinary System.

No abnormal subjective phenomena. Urine: Sp. gr. 1.020; acid; no blood, no pus, no bile, no sugar, no acetone bodies; no albumen. Microscopic examination reveals a deposit of amorphousurate.

Nervous System.

Intelligence normal. Sleep fair, Speech clear. Visual acuity good. Ophthalmoscopic examination reveals no abnormality. There is no myopia. The pupils are of equal size, not quite round; they react sluggishly to light, more rapidly to accommodation. Patient is not deaf. All cranial nerves are otherwise intact. Motor functions: there is a tendency to tremor of the hands when the patient is excited, as on the occasion of his first examination.

Reflexes: Abdominal and cremasteric reflexes present. Plantar reflexes flexor. Wrist, elbow, knee, tarsal, jerks are all brisk. There is no clonus. There is no disorder of miction and defecation.

J. G. V.
Sensory functions: Examination reveals no abnormalities.

Locomotor System:
Bone well-developed, normal. Joints show no swelling, no tenderness, no limitation of mobility. Muscle tone fair.

Reproductive System:
Patient has been impotent "for some time."

Provisional Diagnosis: Aortic incompetence due to syphilitic aortitis.

Treatment as administered:
10. X. 1934  Complete rest. Light diet.
  Mist. Ammon. Carb. 3 s.t.d.
13. X  Stop Mist Ammon Carb.
  Three Spirits 3 1 t.i.d.
16. X  Stop Three Spirits
  Mist Ammon Carb. 3 s.t.d.
  Pot. iod.  5a. 3 t.i.d.
Progress Notes.

10. X. Much less wheezing and fewer crepitations in chest. To-and-fro murmur in the aortic region more easily heard than before.

12. X. Few rhonchi now to be heard. Patient feels better & looks better.

19. X. Patient complains of occasional pain of an "irritating" nature, starting in the precordial area and working round to the back between the shoulders. It has come on generally during the morning of the past week, & lasted on each occasion about 2-3 hours, during which it is quite constant. There are now no rhonchi in the chest; the only abnormalities are the rather prolonged expiration and the coarse crepitations anteriorly in the region of the right humerus. Heart condition unchanged.

23. X. Few rhonchi in right lung.

30. X. Patient looks well, feels much better.

6. XI. Condition unchanged.

6. XI. Patient discharged.

J.G. W.A.
The photograph affords evidence of enlargement of the heart in the transverse diameter, consistent with an apex beat in the 6th intercostal space, 6" from the mid-line. There is no evidence of aortic dilatation. The lack of translucency in the lower right lobe suggests congestion.

Electrocardiogram. 11. 10. 1934.

The large size of the R waves in lead I and of the S waves in lead III are indicative of left ventricular preponderance. The R wave in lead II is bifid. The T wave in lead I is inverted.
Commentary.

The first pathological sign we encounter in the examination of this patient is the well-developed "water hammer" pulse. Such a pulse indicates that the arteries are able to discharge the blood they receive abnormally quickly, either in a central or in a peripheral direction; peripheral, as in toxic goitre, owing to the poor tone of the arterioles; or as in patent ductus arteriosus, with consequent passage of the blood to the pulmonary artery; or as in arterio-venous anastomosis, central, when the aortic valve is incompetent.

In this man the heart sounds - the brachio-femoral murmur in the aortic area, the systolic component of which is conducted into the neck, the diastolic along the sternum and towards the apex - point unequivocally to the last alternative, that is, aortic incompetence. The majority of his symptoms, his breathlessness, dizziness, palpitation, throbbing in the head, the very high pulse pressure, as well as the signs of cardiac hypertrophy, and the excessive pulsations which are felt at the occiput, etc., accord well with this diagnosis. Pallor, which many people hold to be generally associated with aortic incompetence, is paid by bicuspid to be present chiefly in cases of subacute bacterial endocarditis; in this case however the appearance is of little value, for a man whose work is underground, and who since he ceased work has been almost confined to his bed for three months, is not likely to be other than pale; he has only a slight degree of anemia.

Subacute bacterial endocarditis is however one of the common causes of aortic incompetence, though it is relatively uncommon after the age of 50; it is characterized by the signs and symptoms of multiple embolism, toxemia, and progressive cardiac disease; in this case only the cardiac disease is demonstrable, and it is not seen to be progressive during the course of the stay in hospital. We also miss the clubbing of the fingers and the cafe-au-lait.
Another common cause of aortic incompetence is rheumatic disease, the age incidence being on an average still earlier than that of acute bacterial endocarditis, which indeed generally attacks valves that are already abnormal, whether as the result of rheumatism or of congenital malformation. But there is nothing in the history or condition of this patient to suggest that his complaints are due to rheumatism.

There remains the third common cause—responsible for 70% of all cases of aortic incompetence between the ages of 51 and 55—namely syphilitic aortitis, which may affect the valves by direct spread of the disease process, or by dilating the aorta and aortic rings, or by both. The pain or discomfort in the left side of the chest, of which the patient complains, may be causally connected with some degree of aortic dilatation, not sufficient to show on the x-ray photograph. The final link in the chain of evidence, proving almost beyond possible doubt that syphilitic aortitis is the origin of the patient's trouble, is provided by the strongly positive Wassermann reaction.

Both the x-ray photograph and the electrocardiogram are consonant with a diagnosis of aortic incompetence, for they both suggest left ventricular hypertrophy, the two conditions being cause and effect.

In addition to the facts already considered, it is to be noted that the patient complained at the time of admission of cough, opisthon, choking sensation in the throat, wheezing in the chest, hoarseness; that he had a slight rise of temperature on the third day after admission; and that examination revealed an inflamed condition of the pharynx and, in the chest, much lord I numerous rhonchi that they sufficed to render the heart sounds almost inaudible. None of these except the hoarseness would appear to have any direct bearing on syphilitic aortitis or aortic incompetence; nor indeed does the hoarseness, for if it were...
due to the stretching of the left recurrent nerve by a dilated aorta it would have been permanent. All of them in fact go to prove the existence of acute bronchitis; which, judging by the manner in which he described his symptoms, must hitherto have been a stranger to the patient; its occurrence now is probably an indirect result of the cardiac condition which, in addition to lowering his resistance, causes dyspnoea and mouth breathing.

His progress during treatment was the best that could be hoped for; his general condition improved and the vascular disease made no obvious progress. The left ventricle is standing up well to the demands made upon it, and there are no suggestions of relative mitral incompetence.

For the future the outlook is not good. In some such cases subacute bacterial endocarditis develops. Some die of syphilitic meningitis, cerebral haemorrhage, or as the consequence of aneurysmal dilatation in pressure on important structures. The most likely sequence is, that at some not far distant date the coronary arteries, even now narrowed at their orifices, will be unable to supply the great demands made by the overworked, hypertrophied left ventricle; this will lead to marked chronic ischaemic myocarditis, with further dilatation of the ventricle, relative mitral incompetence and failing systemic circulation. Whatever the outcome, it may be delayed for many months; the patient will have to lead the life of an invalid, avoiding above all intercurrent infections. Fortunately his disability comes after a long life, and in any case he would not have been fit for many more years to work at the coal face.

A final point of interest is that his numerous children have escaped the burden of congenital disease. It would have been of great interest to know at what date the infection was acquired, but on this subject his history is silent.

J. G. X
Janet Cunningham, act. II.

"Acute rheumatism with mitral stenosis and auricular fibrillation."

Wightman Prize 1935-

T. H. Brown

Contents:
(i) Chart
(ii) History
(iii) State on Examination.
(iv) Treatment.
(v) Progress notes & x-rays.
(vi) Electrocardiograms with comment.
(vii) Commentary.
(viii) Drawing of subcutaneous nodules.
(ix) Extract from post-mortem report.
(x) Correlation of clinical findings with post-mortem report.
Complaint. Listlessness, weakness, breathlessness on exertion. Duration about 1 year.

History.

Present Illness. The child was active and apparently healthy until last autumn, when she gradually became more listless and easily fatigued. At this time she was kept in bed by doctor's orders for five weeks, after which she was allowed to return to school but not to take part in physical exercises. During this illness there were no growing pains, rheumatic fever, sore throat, or other evidence of rheumatic infection. For the next few months patient was able to take part in and enjoy children's games with her school-fellows, and does not remember that she became unduly breathless. During the last fortnight of the summer holidays she was abnormally easily tired and she lost her otherwise healthy appetite. When school re-opened, a week before admission, a new teacher allowed her to join the others at drill, but becoming too tired she was sent home. I went to bed, where she has remained ever since. At the time she complained of a "quick beating in the chest." No swelling of the legs was ever noticed, nor undue prominence of the abdomen. There was no vomiting.

Previous History. Her appearance in the early days of life was normal, and there were no suggestions which might have pointed to a congenital abnormality of the heart. Some years ago she suffered from "growing pains" and, from time to time, from sore throat. Three years ago she had diphtheria. She has attended school from the age of 5 years, and has appeared healthy in
all respects except as noted above.
Social: home conditions good. Home is dry but dark.
Family: patient is the eldest of four sisters, the others being 10, 8 and 3 years old respectively. Both they and their parents are alive and well.

State on Examination.
Intelligence average. Height: 4 ft. 9 in. Weight: 5 st. 3 lbs.
A well-developed, well-nourished child; skin pale; cheeks and ears cyanotic; lips and tip of nose very highly coloured. Temperature: 97.0°F. No clubbing of the fingers.
Circulatory System.
Patient complains of ‘rapid beating’ in the chest, not of breathlessness. She also states that she has some cough, which is however not observed to be frequent or distressing.
Pulse: arteries not thick-walled or torbaceous; pulse rapid, (110 per minute) totally irregular in time and force; wave fairly strong and well-sustained; arterial pressure 126/86.
Heart: precordial area rather more prominent than the corresponding area of the right side. Pulsations are readily seen in the 5th and 6th intercostal spaces, 4" to 5" from the midline on the left side; also in the epigastrium and in the neck. Cardiac pulsations are most readily felt 2" from the median plane in the 4th and 5th intercostal spaces, but may also be detected at the right border of the sternum, and, on relatively heavy pressure, in the arilla. These pulsations are of a strong, heaving, irregular character. Each systolic wave is followed by an early diastolic thrill of coarse character, most easily felt in the 4th intercostal space in the anterior axillary line. In the 3rd left intercostal space, close to the sternum, there is a short, sharp tap at the commencement of systole. diastole. Deep dulness to percussion extends 1" to the right and 5" to the left of the midline, and upwards to the 3rd costal cartilage. On auscultation a
loud, blowing, systolic murmur is heard, having its maximum intensity in the mitral area and propagated towards the axilla; it is followed by a long drawn, soft, blowing diastolic murmur. The 2nd sound in the aortic area is closed; in the pulmonary area it is very markedly accentuated; in the tricuspid area the first sound appears to vary, being generally pure, but sometimes giving the impression of a systolic murmur. The great veins of the neck are distended and beating strongly. There is no oedema.

Respiratory System.

The patient complains of cough & syrinx, but the former does not appear to be troublesome, and no specimen of the latter could be produced. Respiration is rapid (rate 30), regular and slightly laboured. Voice is clear.

Thorax: chest symmetrical and well-formed except for slight degree of precordial prominence. Movements are free and equal on the two sides. Vocal fremitus is normal and equal in corresponding areas. The note on percussion is resonant and everywhere equal in corresponding areas on the two sides, except that posteriorly there is slight dulness over both bases, right more than left. The breath sounds are pnce, vesicular; there are no accompaniments except for numerous coarse and medium crepitations over both bases, posteriorly.

Alimentary System.

The appetite is normally good, but has not been so during the present illness. Teeth and tonsils are healthy, the tongue moist and covered with yellowish fur. There has been no vomiting recently. The bowels are regular.

Abdomen: slightly prominent; movements are free. There is some tenderness in the right hypochondrium on deep palpation; there is no abnormal resistance.
Percussion shows extension of the liver dulness to a point 2" below the costal margin in the right nipple line. The spleen is not enlarged to percussion or palpation. The abdomen contains no free fluid.

Integumentary System.
The skin is healthy. There is no sign of any eruption or of subcutaneous nodules such as are sometimes found in acute rheumatism. There is no oedema.

Hematopoietic System.
There is no enlargement of any group of lymph glands. The blood examination shows: Hb. 90%, R.B.C. 5,020,000, Colour index 0.9, W.B.C. 13,200.

Urinary System.
Urine examination: colour lemon, specific gravity 1.030, acid, deposit of amorphous urate, contains no sugar, albumen, bile, blood, casts, blood corpuscles, acetoebodies.

Locomotory System.
Bones well developed. The joints are all painless and freely mobile; there is no effusion or swelling. Muscles show no abnormality.

Nervous System.
Intelligence normal. Apathetic state. Visual acuity normal. Fundus oculi shows no pathological changes. Eye movements normal. Pupils round, equal, react promptly to light and accommodation. Other cranial nerves intact. There is no hemor or chorea or any other abnormal movements. No paralysis or ataxia. Abdominal reflexes normal. Plantar reflex is flexor. Wrist, elbow, knee and ankle jerks present. No knee or ankle clonus. There is no sensory loss or subjective abnormality.

Provisional Diagnosis: subacute rheumatism with mitral stenosis and auricular fibrillation.

J.r. IV
Treatment as administered.

(1) Absolute rest.

(2) Diet: 11.区 酵素 vegetarian diet.
15.区 水 diet, including fish, rabbit, chicken, milk.

(3) 11.区 Tinct. Digitalis. M & t.i.d.
12.区 Stop digitalis.
12.区 Stop digitalis.
5. 区 Tinct. Digitalis. M & t.i.d.

(4) 14.区 Buiscin. gr. V t.i.d.
5. 区 Stop chisatin.

(5) 2. 区 Muri. Bism. & t.m. zir. noct.
11. 区 Stop Muri. Bism.
11. 区 Codein jelly zir. noct.

6. 区 Cod liver oil & malt. zir. t.i.d. 8. V.

30. 区 Stop tinct. cardam. co.
30. V Bis. oxycarb. gr. V
Sod. bicarb. gr. V
Be. Hydrocyan. Oil. M &
Tinct. Cardam. Co. M &
Aq. Chlorof. ad. M &
3ir. t.i.d.

5. 区 Stop homuth mixture.

J.C. V.
Progress Notes.

14. Urine only 130 cc. Blood culture negative.

15. Urinary incontinence.

17. Ankles pit on pressure. Condition otherwise unchanged.

19. Weight 5 st. 11½ lb. i.e. 8½ lb. more than on admission.

21. More marked breathlessness than hitherto. Oedema of ankles very marked, slight oedema to level of knees. Over the bases of both lungs the percussion note is markedly dull; breath sounds are bronchovesicular with numerous fine and medium crepitations. Examination of the abdomen for free fluid gives doubtful result.

22. Breathlessness particularly marked towards evening. Oedema more prominent.

24. The 2nd sound in the tricuspid area is a soft blowing murmur. Impossible to detect any liver pulsations. Liver dulness extends four fingers' breadth below the costal margin. Shifting dulness & fulness of both flanks.

27. General appearance much improved. Urinary incontinence has ceased. Examination of the left side of the thorax shows quivering beats of all the interscostal spaces from the 3rd to the 6th. Apex beat and radial pulse rates equal, but rhythm is totally irregular. The 2nd tricuspid sound is closed. Dulness at base of lungs is reduced; there are still crepitations at both bases, left more than right. Liver dulness extends 2 fingers' breadth below costal margin. There is no ascites. Oedema of the legs has practically disappeared. Weight 5 st. 6 lb.

28. Pulsations in the neck & epigastrium more marked than hitherto. Diastolic mitral thrill also more marked, palpable in 4th interscostal space from left nipple line to anterio axillary line, also in 5th and 6th spaces. Heart sounds unaltered. Abdomen prominent. Liver dulness, 3 fingers below costal margin, not tender on palpation. No ascites.

29. No oedema of legs. There is slight dulness over bases of both lungs from 8th rib downwards; there are a few
fine crepitations at the height of inspiration.

Cough: Height 5 ft. 3½ lbs.

Cough has suddenly become very troublesome. No pain on inspiration, no pleural friction. Sputum scanty, blood-stained; microscopic examination showed some R.B.C.'s and non-pigment cells.

Cough and blood-stained sputum persist.

Height 5 ft. 4 lbs. Liver dulness extends 2 fingers breadth below costal margin.

The easily palpable tap hitherto felt in the pulmonary area is occasionally reduplicated, and occasionally obscured by a short thrill. - ? Systolic or early diastolic? On auscultation, the first pulmonary sound is replaced by a rough short murmur, the 2nd sound is closed, very markedly accentuated. Mitral murmurs are unchanged. The 2nd pulmonic sound is closed.

It is possible to feel in succession a short, rough systolic thrill in the 3rd intercostal space 2" to the left of the midline, and a long, soft, murmuring thrill in the 4th I.C.S. in the anterior axillary line. The 1st pulmonary sound is replaced by a short rough murmur, the 2nd remains closed. Other heart sounds as before. Blood examination:

R.B.C. 5,850,000; H.b. 95%; C.i. 0.8; W.B.C. 15,400. Differential count: polymorphs 44.5%; lymphocytes 49%; monocytes 4%; eosinophiles 2.5%.

W.B.C. 15,800. Rheumatic nodules to be seen: one over the right olecranon, one over left olecranon.

Polygraph tracing.

Radial pulse ↑

Apex beat →

Patient vomited in the afternoon. Cough persistent and very troublesome. General condition otherwise unchanged.
Slight oedema of legs. First murmur is louder than before in the apical area, and has taken on a wheezing character.

Face very puffy, skin dry and wrinkled. Legs slightly oedematous up to knees. Marked oedema of the sacral region. Lower dullness extends 3 fingers below costal margin in the right diaphragm, the lower border being very firm, not tender. In the pulmonary area there is a systolic thrill and murmur.

Blood examination: R.B.C. 6,010,000; Hb. 92%; C.I. 0.77

Blood differential count: polymorphs 59%; 32% lymphocytes; 3% monocytes; in counting 200 white cells 15 normoblasts were seen.

Patient looks very much worse than ever before, the skin yellow & very dry, with dark lips and dull, very cyanotic flush of the cheeks. There is no dyspnœa. Marked oedema of the legs, thighs, groin, sacral region. The first murmur at the apex is louder than ever & much more wheezy in character, and is propagated over the whole cardiac area & towards the left scapula. Icteric index 33.

At 7.15 pm the patient suddenly started to cough violently, and to expectorate blood-stained frothy fluid. Dyspnœa became more & more marked although the cough subsided; at every breath there was a loud accompaniment of moist sounds from the mouth. The condition gradually grew worse & the patient fought for breath. At this time there were slow strong pulsations visible in the neck, not synchronous with, but slower than, the pulse. Patient complained of soreness in the abdomen. As heard through her garments, the breath sounds & accompaniments were equally loud at the two bases. There was no complaint of pain in the chest. Some relief was obtained by taking "mist, bromide," and
the patient became much quieter. She remained orthispnoeic, and cough was still troublesome; the rattling moist sounds from the throat continued. At 11 pm she became much quieter, the pulse slower and more regular. At 11.15 pm she died.

X-Ray taken shortly after admission.

The shape of the heart as a whole, globular, dilated to the right and to the left - is consistent with the diagnosis of mitral incompetence & stenosis.

The lack of translucency of the right lung is suggestive of pulmonary congestion; the angle of the ribs - almost horizontal - indicates that the photograph was taken during extreme inspiration, or else it is due to the long-continued dyspnoea.
A typical electrocardiogram showing auricular fibrillation: complete inequality of the pulse and absence of R-wave. In the three leads the pulse rate is equivalent to 120, 144, and 144 respectively. The increased S-wave in lead I and very large R-wave in lead III together indicate right-sided preponderance.

An electrocardiogram very similar to the earlier one: the three pulse rates are equal, though each lead shows the typical inequality of auricular fibrillation, much less markedly than before. Two R-waves are anomalous in lead I and the S-wave is not greater than in a normal record.
The irregularity of the heart is more marked than before. The S-wave in lead I is slightly greater than in the previous record, and the R-wave is smaller. The dimensions of the excursions in all leads tend to be smaller, particularly in leads II & III. The three rates are 88, 75 & 88.

The irregularity is again very marked. The most striking difference as compared with the previous record is the greater rapidity of the heart action, the three rates being 140, 136 and 132. The excursions are also much larger than in the previous record.
Commentary.

This patient is admitted to hospital suffering from heart trouble which, to judge from her history, is of rheumatic origin; for we hear that years ago she used to suffer from frequent attacks of tonsillitis and from growing pains, the former of which may well have been the origin, the latter the product, of rheumatic disease, while there is nothing to suggest that the infirmity is a congenital one. She comes in fact as one of the unfortunate cardiac cripples who make up one third of all rheumatic patients.

The total irregularity of her heart action and pulse is indicative of auricular fibrillation, a great rarity at the age of 11 years; the heart is enormously enlarged, as is shown by percussion, palpation & auscultation; the cardiac thrill, the murmurs and their propagation being such as to suggest mitral stenosis & incompetence, with auricular fibrillation. Although her doctor makes no mention of his former diagnosis it seems probable that he knew of this condition when, a year ago, he kept her in bed for 5 weeks; and it need hardly be said that auricular fibrillation is more commonly associated with rheumatic mitral stenosis than with any other condition.

Her heart remained well compensated, as it would appear, and the rheumatic condition quiescent, until the latter half of August, when increasing lassitude and loss of appetite were probably symptomatic of a reemergence of active disease. This was bad enough, but worse followed when at school she was permitted to join the other children at physical exercises, with the result that hospital treatment became imperative.

At the time of her admission it was thought possible that a sub-acute bacterial endocarditis might have been grafted on to the primarily rheumatic condition. Such a hypothesis is hard to prove & harder to disprove:

J.C. X11
one sterile blood specimen is of little importance. Positive evidence would have been afforded by multiple systemic embolism, - by subcutaneous or retinal haemorrhages, Osler's spots, microscopic or macroscopic haematuria, central symptoms, - none of which was observed; neither was the clubbing of the fingers, generally found in this disease. Repeated haemopysis occurs so frequently in cases of mitral lesions as the result of pulmonary congestion and infarction, or following the bland embolism of a thrombus formed, perhaps, in the right auricular appendage, that it cannot be regarded as of direct significance. Such fever as occurred during the course of the illness was readily accounted for by other conditions present, such as pulmonary congestion, or later on rheumatic nodules. The rheumatic nodules are in fact valuable evidence against the existence of subacute bacterial endocarditis, for the two seldom co-exist. In view of all these facts the idea of subacute bacterial endocarditis was relegated to the background.

Had there been no excessive dilatation of the heart it might have been considered advisable to treat the patient with digitalis and quinidine, in the hope of restoring a normal rhythm; with the size of the heart as it was, quinidine was contra-indicated. It was therefore considered impracticable to attempt more than to restore compensation by rest & digitalisation, - unfortunately without success. Accepting Price's definition of decompensation as "dilatation in excess of hypertrophy" it is evident how completely decompensated the heart remained. Throughout treatment digitalis was of little value, and in fact the clinical findings as regards the extent of the liver dulness & the degree of oedema were never more serious, except shortly before death, than on Sept. 26th, the day on which digitalis medic
tion was first interrupted, - this in spite of its success in slowing the pulse to the neighbourhood of 80 per minute. Since then it was tried for another 14 days without favorable result, and again just before the end.

Two early signs of evilomen in this case were the presence of cardiac oedema, - which does not arise so readily in the child as in the adult, - and the exceedingly early age of onset of auricular fibrillation; but when in addition it became evident from the rheumatic nodules and the change in the cardiac murmurs that the condition was progressive the outlook became exceedingly black.

On the clinical findings alone it is impossible to diagnose as to the immediate cause of the sudden heart failure which heralded the end. The change in the child's appearance, her old, worn appearance, the increase in the oedema, the high icteric index of 33, were all very unfavorable signs. Then a sudden paroxysm of coughing may have been too much for a body that had expended all its reserves; the cause of the coughing may have been pulmonary embolism, though the absence of pain in the chest and of faint collapse make this unlikely; it seems more probable that considerable infarction of the lungs had occurred as the result of long-standing pulmonary congestion; the character of the sputum, - blood-stained, frothy fluid, not blood - proves that in addition to one of these two conditions there must have been considerable pulmonary oedema. The most likely explanation of the slow venous pulsations which were observed in the neck at this time is that they were due to an alteration in the character of the auricular contractions from fibrillation to something more effective; not being synchronous with the pulse, but at a very much slower rate, they cannot have been due to tricuspid incompetence alone.
The abdominal pain was probably due to rapid increase in the size of the liver; this, the pulmonary oedema, the extreme dyspnoea, and the ever-increasing cyanosis were proof of the progressive failure of the circulation which was the final cause of death.

*Rheumatic Subcutaneous Nodule (3 weeks old).*
Extract from the post-mortem report.

Serosa sacs all contained an excess of clear fluid.

Heart was considerably larger than usual, mainly owing to right-sided dilatation. The pericardial surface was smooth and glistening.

The right auricle was grossly dilated & its myocardium was rather thicker than usual. The tricuspid orifice admitted four fingers; the flaps of the valves were healthy except for the presence along the base of a number of minute recent vegetations. The right ventricle was dilated and its myocardium was hyperplastic. It was pale in colour but firm in consistence owing to oedema.

The left auricle was grossly dilated; the myocardium was somewhat thicker than usual. The mitral orifice was narrowed, admitting only one finger; the flaps of the valves showed evidence of old endocarditis and in addition there was a row of recent woody vegetations along the line of closure. The left ventricle was somewhat dilated and its myocardium thicker than usual. The inner layers of the muscle were somewhat pale, but the muscle was of good consistence.

The aortic valve was incompetent to the aorta but the cusps were welded together to some extent near their point of attachment to the aortic root. They also showed evidence of recent acute endocarditis.
Correlation of the clinical findings with the post-mortem report.

The presence of excess of clear fluid within the pericardial sac, combined with a smooth, glistening pericardium, is evidence of transudation, not exudation. This must have been due to circulatory failure; the fluid probably accumulated shortly before death.

The extreme degree of tricuspid incompetence accords well with the ante-mortem observation as to the size of the liver and the degree of cardiac oedema; but it is surprising that even shortly before death no venous pulsations were visible in the neck coincident with ventricular systole; it is possible that the retrograde impulse transmitted to the blood may have been dissipated in the dilated, fibrillating, right auricle.

The one important point on which clinical and pathological findings disagree is the state of the pulmonary valve, which is not mentioned in the post-mortem report because it alone was absolutely healthy. The aortic valve, on the other hand, showed stenosis and incompetence — though there were not suspected during life. It is therefore justifiable to assume that the murmurs heard in the 2nd left intercostal space arose at the aortic and not at the pulmonary valve. The error may be ascribed to the altered pathological anatomical conditions produced by the gross dilatation of the right atrium and the consequent displacement of the origin of the aorta towards the left; due consideration of this fact might have prevented the diagnosis of a lesion known to be a great rarity.
James O'Neil, act 63.

of "essential hypertension" leading to ischaemic changes
towards heart and brain.

Wightman Prize 1935

J. H. Brown

Contents: (i) Charts
(ii) History
(iii) State on Examination
(iv) Treatment
(v) Progress notes
(vi) Commentary
(vii) Electrocardiograms
JAMES O'NEIL, act 63, pensioner, born in Paisley.
examined: 15. VIII. 1934.

Complain: breathlessness; intermittent for some months; acute during the past 12 hours.

History

Present Illness. Until February 1934 the patient had always considered himself to be as fit as his contemporaries. In that month he was so troubled with breathlessness that he had to keep to his bed to call in a doctor, who treated him with such success that he was able shortly to return to his work. Nevertheless, breathlessness on night excitement, such as ascent of a gentle slope, continued, and in July, while the patient was on holiday in Ayrshire, became decidedly worse; he returned to his work on August 7th but after three days had to retire to his bed. As no improvement followed, but rather the reverse, his doctor recommended him for admission to R.I.E. on August 14th.

He does not remember suffering from palpitations prior to the day of admission. He has never suffered from dizziness or other cerebral symptoms, except for occasional headaches. He has never noticed any swelling of the feet or legs, and his weight has been constant for years. For some time past he has had to rise twice or thrice nightly to urinate, and sometimes he has had difficulty in commencing the act. His mouth often tends to be dry, and at such times there is a very unpleasant taste. He habitually suffers from winter cough, which has been no worse of recent years; during the summer he has been quite free.
Previous History. Forty years ago patient had rheumatic fever. He has always been a strong, healthy man, and during the war served in France with a Field Company, Royal Engineers, until in 1918 he received a gunshot wound in the head which damaged his left eye. At the time of his demobilisation in 1919 he was told that he had "smoker's heart." The vision of the right, i.e. the better eye, grew gradually worse, until in 1922 detachment of the retina rendered it completely blind. In 1923 the left eye was enucleated because of the pains he was suffering in the left side of the forehead. Since then he has mastered various occupations (e.g. making, Braille, typewriting) in a disabled soldiers' home at Hennington, where he still resides. In 1929 he fell on the stairs and was treated in the R.I.E. for 10 days, suffering from concussion.

Habits. Moderate drinker of spirit (gin); smokes up to 5 cgs. of tobacco weekly.

Family. Wife and five children alive and well. One child died at 2 years of age of peritonitis.

Social. Patient is well cared for in the soldiers' home at Hennington, where he carries on his occupation, chiefly rug-making.

State on Examination. Height: 5'7". Weight 10 st. 6½ lb.

Intelligence average. Blind. Slightly deaf. A well-developed man of good appearance; rather corpulent. Anxious expression; colour pale; hypnic eye; no venous congestion. Temperature 97.2.

Circulatory System.

Patient complains of violent palpitations; the heart going like a motor. Respiration rate 44; the difficulty, according to the patient, is in inspiration.

Pulse: arteries hypertrophied and tortuous; rate 120; regular in time and force; wave strong & well-sustained.
Sphygmomanometric reading 230/1140.

Heart: precordial area of normal form. Apex-beat is visible in the 6th left intercostal space, 6" from the mid-line. Weaker pulsations are visible in the neck and in the epigastrium. Apex-beat is of a strongly heaving character, regular in time and force. There is no palpable thrill. Area of deep cardiac dulness extends 1" to the right and 6" to the left of the mid-line; the upper border lies at the 3rd costal cartilage. A soft, blowing systolic murmur of maximum intensity in the mitral area is propagated slightly towards the axilla. In the tricuspid area the first sound is closed. The second sound is closed in all areas; there is accentuation in the pulmonary area, and very marked accentuation of an almost musical kind in the aortic area. There is no venous engorgement and no capillary pulsation.

Respiratory System.

There are no abnormal subjective phenomena, apart from the deep and laboured respiration, of frequency 44 per min. (On the evening of admission the Cheyne-Stokes syndrome was noticeable.) The severity of the dyspnœa almost prevents the patient from speaking. The thorax is well-formed and symmetrical. Movements large and free. Vocal fremitus is normal and equal in corresponding areas. The note on percussion is somewhat hyperresonant over the whole pulmonary area, and equal in corresponding areas. Breath sounds are vesicular, with slight prolongation of the expiratory part; there are numerous medium and low-pitched rhonchi; no crepitations; no friction.

Urinary System.

Patient complains of having to rise twice or thrice nightly to urinate, and of occasional difficulty in commencing the act. Urine is clear, amber-coloured.

J.O.N. III
of specific gravity 1022; acid; contains albumen, no sugar, no acetone bodies; no blood; no pus; no bile. Microscopically there are a few granular casts, no red or white blood cells. (Subsequent examination of the prostate revealed marked hypertrophy; there was no residual urine.)

**Alimentary System**

Appetite is good, and the patient is able to tolerate everything except whisky, which he vomits. The teeth are all extracted. The tongue is moist and covered with a brownish film. Tonsils healthy. Patient is in the habit of regulating his bowels with laxative medicines. He suffers from flatulence.

Abdomen is rather adipose; movements are free; there is no tenderness or undue resistance; the liver and spleen are not enlarged to percussion or palpation. There is no fluctuation or other evidence of free fluid. The rectal mucosa feels healthy.

**Nervous System**

Intelligence average. Patient is rather introspective. Sleep is satisfactory. Memory fair. Speech clear. The right eye is emaciated; left eye is blind, and the media opaque; the pupil does not react to light. Patient is rather deaf. All other cranial nerves intact.

Motor functions are unimpaired. There are no abnormal movements, atrophy or paralysis. Muscles tone fair. Abdominal and cremasteric reflexes very feeble. Plantar reflexes are flexor. Elbows, wrists, knees and ankles jerks all brisk. No ankle or knee clonus. There is no loss of sensory faculties.

**Locomotor System**


**Integumentary System**

The skin is healthy. There is no oedema.

J. O'N. IV
Haeemopoietic System.
Hb. 90% C.I. 0.9. The lymphatic glands and the thyroid gland are not enlarged.

PROVISIONAL DIAGNOSIS.
Essential hypertension, leading to ischaemic changes in kidneys, heart and brain.

Treatment as administered.
(i) complete rest. The patient was kept in bed for 21 days, and then only permitted to walk to and fro in the ward.
(ii) diet. A low protein diet.
Mag. sulph. 3ij mane.
(iv) digitalis. 15. VIII. Ta. digit. M xxv t.i.d.
20. VIII. Ta. digit. M x t.i.d.
22. VIII. Stop digitalis.

and on re-admission:
(i) complete rest.
(ii) light dry diet.
(iii) drug treatment:—
28. XII Tinct. digit. M xx t.i.d.
29. XII Theobrom. et Sod. salicyl. qr. x t.i.d.
2. XII Tinct. digit. M x t.i.d.
11. XII Stop theobrom. et Sod. salicyl.
16. XII Quinidine sulph. qr. vii 4 hourly
17. XII Quinidine sulph. qr. vii 8 hourly
21. XII Quinidine sulph. qr. vii 4 hourly
23. XII Stop quinidine.
Tinct. cardam. ca. 2 oz. mouth. p.i.b. 3ij t.i.d.
Three spirits 3ij noct.
31. XII Syr. Cocillana Co. 3ij t.i.d.

4. I  Mist.  pect.  sedat.  3 fl.  q.i.d.

7. I  Stop  Tinct.  cardam.  co.  2  aq.  mouth.  pip.

27. I  Stop  Elixir.  Heroin.
Stop Three Spirits.

Theobrom.  at  sod.  salicyl.  gr.  1/2  q.i.d.
Laboratory Investigations.

Urea range test.

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<th>20. VII. 1934.</th>
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<th>27. VIII. 1934.</th>
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<tbody>
<tr>
<td>Vol. m. c. c.</td>
<td>Sp. Gr.</td>
<td>Conc. %</td>
<td>Vol. m. c. c.</td>
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<tr>
<td>6 a.m.</td>
<td>600</td>
<td>1012</td>
<td>1.4</td>
</tr>
<tr>
<td>7 a.m.</td>
<td>95</td>
<td>1015</td>
<td>1.7</td>
</tr>
<tr>
<td>8 a.m.</td>
<td>240</td>
<td>1011</td>
<td>1.2</td>
</tr>
<tr>
<td>9 a.m.</td>
<td>140</td>
<td>1004</td>
<td>1.5</td>
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(From these figures it is easy to calculate that from 10 p.m. to 9 a.m. the weights of urea excreted were, in the first instance, 13.59 grams, in the second instance 10.90 grams.)

"Blood Chemistry."

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<th>15. VII.</th>
<th>17. VIII.</th>
<th>25. VIII.</th>
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<tbody>
<tr>
<td>Creatin:</td>
<td>2.9 mgm. %</td>
<td>2.8 mgm. %</td>
<td>2.6 mgm. %</td>
</tr>
<tr>
<td>Co.-comb. power</td>
<td>68.5 volo. %</td>
<td>50.5 volo. %</td>
<td>62 volo. %</td>
</tr>
<tr>
<td>N.P.N.</td>
<td>56 mgm. %</td>
<td>146 mgm. %</td>
<td>145 mgm. %</td>
</tr>
<tr>
<td>Albumen</td>
<td>3.66 Grm. %</td>
<td>266 mgm. %</td>
<td>1448 mgm. %</td>
</tr>
<tr>
<td>Globulin</td>
<td>1.25 Grm. %</td>
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<td></td>
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<tr>
<td>Cholesterol</td>
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The urine was examined on 20 occasions, apart from the above tests. It contained albumen in every specimen from the date of admission till 26. VIII. 1934, more from 29. VII. 1934 until 9. IX. 1934, since when there has always been a trace. Granular casts were present on 3 occasions: 15. VIII. 19, VIII. and 18. IX. There were never any R.B.C.'s, generally W.B.C.'s; fresh specimens contained masses of coliform bacilli.

Sphygmo-graphic Tracing.
Laboratory investigations on re-admission.

"Blood Chemistry."

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<th>27. XII. 34</th>
<th>31. XII. 34</th>
<th>28. I. 35</th>
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<tbody>
<tr>
<td>N. P. N.</td>
<td>28 mgm. %</td>
<td>80 mgm. %</td>
<td>26 mgm. %</td>
</tr>
<tr>
<td>Albumen</td>
<td>2.8 gm. %</td>
<td>2.3 gm. %</td>
<td>2.6 gm. %</td>
</tr>
<tr>
<td>Globulin</td>
<td>57.5 vols. %</td>
<td>69.5 vols. %</td>
<td>64 vols. %</td>
</tr>
<tr>
<td>CO₂-comb. power</td>
<td>26 mgm. %</td>
<td>24 mgm. %</td>
<td></td>
</tr>
<tr>
<td>Urea N.</td>
<td></td>
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</table>

The urine was examined almost daily throughout the patient's second stay. There was found to be generally a trace of albumen, sometimes '+' or '++', occasionally none. In the centrifuged deposit there were occasionally granular casts, on one occasion they were very numerous; there were almost always a few (white blood or) pus cells, very often spermatozoa.

The amount of albumen, while varying, tended to rise towards the end of the patient's stay.

Sphygmographic tracing on re-admission.

Blood examination: R.B.C. 3,860,000. W.B.C. 10,100
Hb. 75% C. I. 10.
Progress Notes.


22. VIII. Pulse occasionally misses a beat. Frequency of radial pulse and apex beat identical.

23. VIII. Partial heart block confirmed by electrocardiogram. Normal beats between the pauses vary in number from 3 to 7. P-R interval (lead II) varies from .24" to .42".

Patient feels quite well and is unconscious of the irregularity of his heart's action.

27. VIII. Heart condition similar. Lead II of the E.C.G. happen to show two ventricular extra-systoles, one immediately following upon, and one coincident with, a P-wave.

3. IX. P-R interval varies from .24" to .44". As the general condition of the patient is good & his circulation satisfactory, he is allowed up.

6. IX. Amyl nitrite was administered, to investigate the reaction of the heart. (Preliminary control showed an identical condition with the previous days.) From the first moment of inhalation the action of the ventricles became regular. Inhalation lasted 20" but the regular action persisted for nearly 3 minutes, after which time there was a return to the former condition of partial heart-block. Owing to the increased rate of heart beat during & immediately subsequent to the inhalation, the T wave frequently coincides with the following P-wave; but where it is possible to distinguish the two, the P-R interval was found to be .40".

12. IX. Patient complains of sharp constant pain in the right side over the lower ribs. There is no pleural friction, no tenderness or resistance in the abdomen. There is no change in the urine. No explanation of the origin of the pain is found. Temperature rose to 100°F and with the rise in temperature the heart block disappeared. Patient's condition is otherwise good.

J. O'N. X.
13. E. Pain is gone. Regular heart-ach on continue.

13. E. Partial heart bloc re-established, but disappeared when the patient rose and dressed. Dr the evening only an occasional beat was missed (about 1 in 30), also after he had returned to bed.


2. E. Right plantar reflex is definitely extensor.


16. E. Re-admitted. Patient gives the history of never having felt well since leaving the hospital; nevertheless he had been able to get about until Nov. 11th when he got well through; since then his condition has got steadily worse. He is glad to be back.

On re-admission he looked distressed, the face being drawn and white; his hands, particularly the left, deeply and irregularly congested, very cold and pale, not oedematous; the terminal phalanges of deep purple colour. There is no oedema of the face, nor obvious swelling of the veins of the neck. There is a bruise, very marked oedema of both legs & scrotum of the scrotal area; the anterior abdominal wall is not affected. The patient is orthopnoeic; there is marked respiratory periodicity of Cheyne-Stokes type; the pulse is regular and feels softer than the sphygmomanometer would lead one to expect; the voice is poorly sustained. There is some unproductive, moist sounding cough.

All over both lungs numerous crepitations are to be heard; the percussion over both bases being impaired; heart sounds are broncho-vesicular and partially masked by rhonchi. The apex beat is 5" from the mid-line, in the 6th intercostal space; the heart sounds are pure in all areas, except for
a soft hoarse systolic murmur, mostly readily heard at the apex. The tongue is very dry and slightly coated with a white film.

27. The periodicity of the respiration is much less marked; inspiration is short & rapid, and followed immediately by a rapid grunting expiration and a relatively long pause before the next inspiration. P.R. 190/126. E.C.G. shows left ventricular preponderance, the one striking change since the date of discharge being the reduction of the P.R. interval from 0.38" to 0.20".

28. Although the patient has been restless and very depressed he continues to take his food with an appetite. Respiration is slower but of the same laboured character. The hands as a whole are of a yellow-brown, faded colour, dotted with blue patches, especially towards the tips of the fingers; they are not oedematous. When the hand is held head high, the yellow-brown colour turns paler, and most of the blue patches almost disappear, a few only, particularly those on the left little finger, remaining unchanged. There is little cough. The oedema has extended to the penis, which is enormously swollen. The lower abdomen gives a dull note on percussion. The plantar reflex is flexor on both sides.

30. General condition much better; oedema slightly reduced, respiratory rhythm normal. E.C.G. shows slow fibrillation.

31. Oedema much reduced. Colour of fingers almost restored to normal. Pulse regular. E.C.G. shows a P.R. interval of 0.28" (maximum). In lead II there is a blocked auricular extra systole.

6. E.C.G. shows a coarse auricular fibrillation which approaches flutter.

7. E.C.G. shows 4:1 auricular flutter. Carotid vagal pressure was applied, without response.

10. Auricular flutter persists. Intravenous injection of quinidine sulphate, gr. 2½, caused transition to coarse fibrillation, which persisted until the following day.

J. ON. XI
11. XII. The apical murmur is harsher & broader than on admission.

13. XII. Auricular flutter (4:1) was changed abruptly to auricular fibrillation by the intravenous injection of quinidine sulphate, q.t.i.

14. XII. Patient's general condition relatively satisfactory. Respiration is regular in time but varies considerably in depth, the changes showing regular periodicity. Hatting remains of the oedema except to a slight degree at the ankles.

E.C.G. shows 4:1 flutter.

17. XII. Oedema increasing once more, the legs up to the knees pitting deeply on pressure. Pulse is regular, the E.C.G. showing normal auricular deflections with a prolonged P-R interval (0.26s).


25. XII. Oedema extends to the scrotal area, but not so high as on admission. Pulse is regular, the E.C.G. shows normal auricular deflections with prolonged P-R intervals. The tracing is less typical of left ventricular hypertrophy.

26. XII. E.C.G. shows auricular fibrillation.

29. XII. Oedema considerably reduced. E.C.G. shows heart block with dropped beats, the P-R interval varying up to 0.44s.

30. XII. Dyspnoea troublesome during the past 24 hours; there is also persistent, dry cough. There is dulness over both bases, with feeble breath sounds & many crepitations.

31. XII. Patient very breathless; respiration shows marked periodicity of Cheyne-Stokes type. Mental condition very confused. There is no evidence of ascites.

9. I. E.C.G. shows a change from flutter in the morning to normal rhythm in the afternoon, with a P-R interval of 0.38s.
18. I. The cardiac rhythm continues to vary between flutter, fibrillation and heart-block with dropped beats from day to day; the oedema also continues to fluctuate; but the general appearance of the patient - drawn and haggard - suggests that he cannot live long. The mental condition is very confused and he does not know where he is; but he wishes to go home. The plantar reflex on the left side is flexor, on the right extensor.

22. I. Oedema very extensive; physical & mental condition are worse.

25. I. E.C.G. shows a condition of irregularity, which only on account of the rhythmicity of the changes cannot be designated ventricular arrhythmia.

28. I. Oedema extends to the lower thoracic spine; there is considerable ascites and (?) bilateral pleural transfusion effusion.

29. I. Patient discharged home at wife's request.

Information was subsequently received that the patient continued to go steadily downhill, and died on 9th February, 1935.
Commentary

The first question which requires attention is: what is the cause of the patient's dyspnoea? - for this is the symptom which brought him to hospital.

Four possible causes, each capable of obvious physico-chemical explanation, suggest themselves at once: - (i) respiratory disease hindering the free interchange of gases at the alveolar surface; (ii) severe anaemia of any type; (iii) gross cardiac failure, causing imperfect circulation of sufficiently oxygenated blood; (iv) acidosis in any form.

Evidently the lungs & bronchi are not the seat of the trouble, for on first admission there was complete absence of symptoms or signs suggesting respiratory disease or pleural effusion. Anaemia is excluded by inspection of the mucous membranes, coupled with examination of the blood. If there be cardiac failure, then it follows that in the previous period of relatively good health the blood pressure had been higher than at the time of admission, when the systolic pressure was at 200 m.m. of mercury; but the further course of the illness, during which his head was afforded rest and the chance of recovery, shows that his condition improved while the blood pressure tended to fall; moreover, heart failure would be likely to be, and in fact in the later stages of the case was, coupled with venous congestion and oedema of the lungs & of the dependent parts. Thus only the fourth explanation, acidosis, remains. And of the causes of acidosis - persistent vomiting, starvation, diabetes, poisoning, anaemia, - anaemia alone cannot be readily excluded by perusal of the history and by a few tests.

This line of argument therefore strongly suggests anaemic acidosis as the cause of the dyspnoea; it is with surprise that we find that during the acute illness of his first admission the CO₂ - combining power of the blood was 68.5 volumes %, a figure which is well within the normal range & indeed rather nearer the higher than the lower limit of normality.
It is true that the non-protein nitrogen was raised to 56 mgm.
% and that it fell during treatment; but latterly, when the
patient was moribund, the non-nitrogen was only 26 mgm.%
and the CO₂ combining power 64 vol. % - facts which prove
conclusively that uremia cannot be held responsible for his
condition. All other signs and symptoms which might have been
held to corroborate the diagnosis - inconstant albuminuria with
granular casts; the abnormally brisk tendon reflexes; the history
of headaches; the rise of blood pressure; the hypertrophied heart
and arteries; the increasing mental confusion; the brown-frayed
tongue; - all are capable of other explanations than uremia.
A diagnosis we are forced to reject, or at least to find
inadequate.

May there then not be other causes of dizziness, incapable
at present of such obvious explanation, but well-established
by clinical observation? One such is cerebral haemorrhage, in
which respiration tends to be hurried, noisy and stertorous, t
with increasing intracranial pressure to become irregular,
grouped, or of the Cheyne-Stokes type. Another such cause
is found in "essential" hypertension; and in this connexion
it has been written that when breathlessness is out of all
proportion to the degree of cardiac failure, and where no other
cause for it exists, "the breach of the usual relationship is im-
portant clinically, because if it is quickly observed it will very
often lead almost at once to a correct diagnosis." (Devis). We
may without hesitation reject cerebral haemorrhage as the
diagnosis in this case, but essential hypertension remains as
a possibility.

Consideration of the cardinal symptom of dizziness has
thus brought us to the consideration of hypertension, which,
as it is interesting to note, was one of the few pathological
signs which the patient presented throughout the whole of
his five and a half months of illness. Hypertension is, as Fishberg (Hypertension & Nephritis,
1934) says, only a symptom, and he enumerates 19
conditions in which it may be found; but most of these can be rejected at once, and we only need to consider "essential" hypertension, chronic glomerular nephritis, and, as some may add, chronic interstitial nephritis. The final attitude to be adopted with regard to the last named is still uncertain, but it is a sign of the times that Gray ("Nephritis & Allied Diseases, 1933"), Harris ("Diseases of the Heart, 1933") and de Besselow ("Creonan Lectures, 1934") approaching the question from entirely different angles, all agree in discarding the term altogether; so also does Fishberg; as Gray says, "older descriptions of 'chronic interstitial nephritis' obviously include both cases of chronic [i.e. glomerular] nephritis and others, which are now fairly widely regarded as examples of non-nephritic ('essential') hypertension. Lowenthal (1927) described a case which he considered by to be true chronic interstitial nephritis, but he believed it to be unique in the literature. (Zeitschr. f. klin. Med.). Admittedly there are many cases in which it is exceedingly difficult to distinguish during life, or even after death, between cases of essential hypertension and chronic [glomerular] nephritis; nor is this surprising, for, however different the pathological factors may be, renal damage is believed to be due in the one case to thrombosis of the affected glomerular arteries, and in the other to obstruction of the glomerular tufts; and it is this difficulty of distinction which has given rise to, but no longer justifies the use of, the term 'chronic interstitial nephritis.' In the case under discussion we have therefore to decide between chronic [glomerular] nephritis and essential hypertension, - one point, namely, the marked dyspnoea in the absence of severe uremia and of signs of marked cardiac failure, having already been adduced as strongly in favour of the former.

Statistically, 'essential' hypertension is much the commonest cause of raised blood pressure, - commoner by far than chronic nephritis; Gray in his series of 500 consecutive post-
mortality found it to be the cause of death in no less than
40 cases, 24 of which terminated with cerebral haemorrhage; in
the same series there were only 10 cases of chronic nephritis,
of whom died of uremia and the tenth of pneumonia. On
this basis a point in favour of essential hypertension
as the diagnosis in our case is the course of the illness, for
the patient died of heart failure, not of uremia.

The age and sex of the patient do not help us; at his age
death from either cause is not uncommon, and essential
hypertension is only slightly more common in males than in
females.

Many cases of nephritis are believed to run an insidious
course throughout, and therefore the absence in this case of any
history of acute nephritis with renal oedema and haematuria
will not serve to distinguish between the two. Neither can we
derive any help from the old diagnosis of "smoker's heart," or
from the account of the loss of the second eye; we can only say
that if one of these two conditions was responsible, then it was
essential hypertension, for chronic nephritis of much degree
would not have spared his life for 12 years.

The question of duration has thus further interest for us,
that from the very marked hypertrophy of the cardiac atrium
we can be certain that the high blood pressure is of long
standing, and we know that the average duration of essential
hypertension is greater than that of chronic nephritis.

In chronic nephritis we should expect to find a copious
urine of relatively fixed, low specific gravity, with constant
albuminuria, and that in the urea range test there was
poor concentration and poor dilution; whereas in fact we
find that during the patient's first stay in hospital, oedema
being absent, the volume of urine averaged just under 1000
c.c. daily; that the specific gravity varied from 1022 to
1004; that the albuminuria was quite inconstant; and that in
the urea range test the specific gravity varied between 1018
and 1004, the urea concentration varying between the poor
Maximum of 1.5% and the satisfactory minimum of 0.5%.
It is on the question of concentration more than on any-thing else that Gray lays stress as a point of differentiation between essential hypertension and chronic nephritis. The key study ... fundamental difference. "The only conditions other than chronic nephritis which produce the same lowering of concentrating power are conditions in which a degree of atrophy equivalent to that found in chronic nephritis is present, - e.g. polycystic kidney.

Price (Text-book of Medicine) stresses the same point, adding that "Ischaemic symptoms do not develop unless the condition [i.e. of hypertension] is complicated by true nephritis."

The patient's complaint of nocturia was evidently not due to polyuria, as might have been supposed; it may have been due to his enlarged prostate, or to irritation resulting from bacilluria.

A final point in favour of essential hypertension is the absence of serious anaemia, the haemoglobin on admission being 90% with 4.74 million red cells.

The term "essential" hypertension is apt to mask the fact that nothing is known with certainty as to the aetiology of the condition; but it is believed that the immediate cause of the raised blood pressure is increase of tone of the peripheral vessels, particularly of the arterioles; that the consequence of persistently increased pressure is firstly medial hypertrophy, and later medial degeneration and intimal-stenotic hyperplasia; and that thus ischaemic changes may occur in any part of the body, such changes being most readily deduced during life from impaired function of kidneys, heart and brain, and most evident after death in kidneys, pancreas, spleen and liver.

The question of this man's renal function has already been discussed; it remains to refer to the signs of symptoms referable
to heart and brain.

Considering the latter organ first: the briskness of the tendon
reflexes, taken alone, could not be regarded as pathological;
but, taken in conjunction with the plantar reflex which, though
generally flaccid, was for two brief periods extensor, points
to imperfect function of the pyramidal tracts. The corresponding
impairment of sensory function was detected, except that his
hearing was dull. But the higher mental functions, though
not constantly deteriorating, tended to fail and were finally
almost completely abrogated, so that the patient did not know
where he was, nor to whom he was speaking. The objection
may be made this cerebral failure might be due to progressive
insufficiency of the circulation as a whole, but it must be
remembered that frequently in cardiac failure the mental
powers remain distressingly alert.

The first striking change in cardiac function occurred early
in the first period of treatment when the rhythm became
characteristic of "partial heart-block with dropped beats;"
or, as it is sometimes called, "heart-block of second degree."
The digitalis which had been administered was insufficient
to account for the change, which gradually tended to become
less extreme, until the only sign of block was the electro-
cardiographic demonstration of a P-R interval prolonged
to 0.40" ("heart-block of first degree.") On re-admission
seven weeks later it was surprising to find that the P-R
interval was no more than 0.20", that is, practically
normal. But from then onwards there occurred a kaleido-
scopic series of changes in which regular rhythm, heart-
block with dropped beats, auricular fibrillation and
auricular flutter alternated in bewildering manner. During
the first period of second degree heart-block experimental
proof was obtained that amyl-nitrite, probably by virtue
of its vaso-dilator action, was capable of restoring normal
rhythm and temporarily stabilising the P-R interval at 0.40;*
hence on the effect of quinidine in auricular flutter was

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found to be a temporary change to auricular fibrillation. (See
electrocardiograms). But as the end drew near worse and
more anomalies crept into the records, until finally they
showed something approaching ventricular arrhythmia. It is not
clear to what extent these latter changes should be attrib-
uted to the direct effects of localized ischemia on the my-
cardium, and to what extent similar changes might have
occurred if the heart had been failing from other causes
than essential hypertension; but in view of the amyl
nitrite experiment and of the subsequent spontaneous
reduction of the PR interval to normal duration it seems
justifiable to assume that a vascular lesion, partly revers-
sible, and situated in the auriculo-ventricular functional
zone, was responsible for the phenomenon of heart-block;
and, that being so, that similar lesions existed throughout
the heart. Nor can we confine ourselves to the heart, for
it seems most probable that the disorders of renal and
cerebral function were associated with identical changes.

Not only do the vascular changes impair the power and
the reserves of the myocardium, but they also, by diminishing
the efficiency of the peripheral vessels, and, perhaps, of the
vessels supplying the vital centres in the medulla, create con-
ditions in which a higher blood pressure would be essential
for the maintenance of efficient circulation. The combination
of diminished power with increased demand explains the
frequency of cardiac failure in essential hypertension. Dilata-
tion of the left ventricle, and later also of the right, lead to
pulmonary congestion, swelling of the lung, ascites, and
edema of the dependent parts; all of these phenomena
being present in the later stages of the present illness.

From the therapeutic point of view the most interesting con-
dition was that of auricular flutter with a regular 4:1
ventricular response; for in that condition the danger of
change to 3:1 or 2:1 response must always be borne
in mind; such a change would in this case have spelt dis-
aster. It is true that the prolongation of the P-R interval proved the reluctance of the ventricles to respond to any auricular stimulation, and therefore the danger never appeared to be a very imminent one; nevertheless it was considered the improbable occurrence of more rapid response was a greater danger than the administration of quinidine, with the resulting depression of the cardiac action.

In essential hypertension the dangers of cerebral haemorrhage is always very real, more insidious but none the less sure is the onset of cardiac failure; or it may be, though this is sometimes denied, that even in an uncomplicated case vascular changes in the kidney may progress so far as to cause renal failure and death from uremia; finally, it is certain that intercurrent disease in a hypertensive patient is apt to have unduly deleterious effects, as was exemplified in this case when the patient took a chill and thereby precipitated his final illness. After his first discharge from hospital, when his case was first reviewed, it was considered that his span of life must be counted in months; and so it proved. It seems doubtful whether medical science will ever be able to cure an advanced case of essential hypertension.
6.15.1934

Controls after amyl nitrite.
10. Aug. 1934
Control before quinidine 0.25 g, 2 1/2 hours later.

10. Aug. 1934
After quinidine. I

11. Aug. 1934

12. Aug. 1934