Entry for Wrightman Prize for Clinical Medicine

Report and commentary on 5 cases seen in wards 23 and 24 of the Royal Infirmary.

Kind permission by Professor Murray Lyon has enabled me to write these.

Subject: "Acute rheumatism and some of its sequelae."

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Craigdarroch
Bo'ness

Final Year 1952.
June 5th.
Case 1: Acute Rheumatic Fever

Patient's Name: James Turnbull
97 Whistern Road
Skenhouse.

Age: 23 years. Occupation: electrician

Recommended by: Dr. Paterson,
Henderson Terrace,
Gorgie.

Admitted to Ward 23: Monday 7th January 1952.
Discharged from Ward 23: 14th March 1952
to Castley Atricre Hospital.

Complaint on admission:
1. Pain and stiffness of 10 days duration in knees,
ankles, elbows, wrists and the small joints of his hands.
2. General malaise, headache, sweating — 10 days ago.

History of Present Complaint:
James Turnbull was a healthy, fit young man
until December 25th 1951, when he started that suddenly
to shiver and feel cold. He went to bed early that night
and when he got up next morning noticed that his arms
and legs were stiff and sore. He went to work — he
was working as a new builder at Portobello, installing
light fittings — and these aches disappeared by 1 p.m.
when he got home at night, however, the aches returned
— he described them as cramp-like pains in his shoulders
and thighs and calves.

He continued to go to work, although these pains
were really severe, until Thursday night (28th). Then he went to
see his Doctor who sent him home with a claim "to
sweat him." On returning home at 8 p.m. he felt severe
pain in the soles of his feet, and noticed his knees and
ankles to be swollen. He went straight to bed.
On the morning of December 29th he could not walk for the severe pains in his legs. His left wrist was now swollen and tender and the small joints of both hands were aching. He says that he "felt quite well in himself," but the pains were quite incapacitating.

He stayed in bed at home until 2nd January by which time all the pains had gone, so he got up and walked about, remaining indoors. He got into sleep that night for return of the pains and next morning his legs were again very painful, especially on movement.

Dr. Paterson came to visit him on Saturday January 6 to give him more tablets; he had been taking 2 tablets three times daily; he had noticed that when he had a fairly heavy sweat, the pain was considerably eased.

On Sunday 6th January his left wrist was very painful, fixed in flexion and swollen. He could not move at all.

On Monday 7th January his wrist was normal but his left leg was completely still and very painful.

He was admitted to Ward 23.

He has never had any trouble of this sort before.

Past History

No rheumatic history. No growing pains.

As a child, he had chicken pox, measles, mumps, and whooping cough. No scarlet fever, diphtheria, pneumonia, pleurisy.

When 5 yrs old he had a bronchotomy: he thinks that he had had trouble with his throat before this, but he never had a sore throat since.

He had an attack of influenza 1 year ago.

Some time ago he swallowed some sulphuric acid, that he had been syphoning and had some oesophagia for 2 weeks.

No other operations.

Ears, nose, mouth: no trouble: does not get head colds: often

Appetite: good

Weight: steady

Bowels: regular
Family History

His mother and father are both well. A sister died in 1932 in the R.I.E. of "rheumatic fever and chorea." Paternal grandmother had rheumatic fever when she was young. No other relevant facts: no tuberculosis in the family history.

Social History

The father works at McEwan's brewery checking beer onto lorries.

The patient likes his own job: he has had it since April 1951. He goes by car with his employer to fit up new houses.

He lives in a corporation house, family room, and he describes it as rather changing. There is a living room, bedroom, scullery and bathroom.

Until he moved to this house 10 years ago, he lived in McLelland St., Corrie.

He was in the Army from 1942 - 1949 and was stationed in Düsseldorf. He was in the Royal Signals and was walking on telephone lines etc. He enjoyed army life.

After admission,

James Turnbull continued to have fleeting pains in nearly all his joints which came and went. He gradually settled down but often he would be free from pain for some days, and then get an acute recurrence of pain in both hands, or wrists, or legs.

He continued to feel well in his general health but was not free of pain until well on in February; on reducing the dose of Selegiline the pain returned but eventually he did really settle down and was symptom-free.
Examination:

on admission:

General appearance:

James Turnbull is a tall thin young man and looks flushed with some circumoral pallor. His skin is hot and dry and his shoes not too well. Mucous membranes are part.

No other abnormality can be seen; no Cyanosis, rash, jaw.

He appears quite intelligent and is friendly and very willing to tell his story and is as helpful as he can.

Temperature: 100.8°
Pulse rate: 100 / min.
Respiration: 30 / min.

Upper Limb:

Shoulders: normal.
Elbows: not swollen but tender; full movement present.
Wrist: left wrist swollen but tender.
Hands: Interphalangeal joints appear swollen and cut tender.

Cardiovascular System:

Pulse: rapid, 100 / minute, regular in time and force.

The pulse wave is not well sustained, rather sloping in quality. Bessel wave not palpable.

Blood pressure: 150/90 mm. Hg.

Heart. Apex beat in mid clavicular line is 6th intercostal confirmed by palpation. Systolic small felt at the apex.

Heart Sounds:

Apex: soft blowing Systolic murmur heard which was localised, not heard in the axilla. No diastolic

The second sound in mitral area is increased.

Base: rushing aortic Systolic murmur heard over the heart which was heard also in the neck.

Oedema: None.

closed in other areas of precordium.
Respiratory System

Upper respiratory tract appears normal. No abnormal breath seen.

Chest: moves well on inspiration. Trachea central.

Respiration 25/minute.

Percussion note: resonant all over the chest, welllung.

Breath sounds vesicular, no accompaniments.

Coughing of fingers - absent. No cyanosis.

Alimentary System

Tongue: slightly pumped, moist.

Teeth: good condition.

Abdomen: moves freely, fairly firm but no evidence of recent loss of weight. Skin tone good.

Nothing abnormal felt on palpation.

No enlargement of spleen or liver or kidneys.

Rectum: Normal proctab.

Lymphatic System

No glandular enlargements.

Spleen not palpable.

Urogenital System

No abnormality found. No complaint.

Urine:

Colour: yellow
Specific gravity: 1025
Albumen - ve.
Sugar - ve.

No deposit. No blood.

Central Nervous System

Pupils: equal, react briskly to light and accommodation.

Fundus: no abnormality seen.

Cranial nerves: no abnormality.

Reflexes: all equal, brisk: but knees difficult to test for pain on movement.

Abdominal: + +

Plantar: \_ \_
Treatment and Progress

7-1-52. P. Ascorbic acid tablets 1 (50 mg.) t.i.d. Temp. 102.4°

8-1-52. B.S.R. found to be 90 mm. in 1st hour.

P. 1. Sodium salicylate gr 30 3 hourly.
Sodium bicarbonate gr 30 3 hourly.
2. Phenobarbitone gr 1 t.i.d.

9-1-52. James improved. Still complains of pain and swelling of his left knee; especially pain on movement.
Fluid can still be demonstrated in his knee joint.
B.S.R. 70 mm./hr. Temperature at night: 101°.

Blood investigation:
W.B.C. count 5000
R.B.C. 4,200,000
Hb. 75 %

11-1-52. Still swelling and pain in small joints of hands.
P. wrist swollen and very tender.
P. Sodium salicylate gr 10. added alternat. 3 hourly doses.
Temperature at night: 101°.

12-1-52. B.S.R. 60 mm./hr.
Temperature not rising above 98.4°

23-1-52. B.S.R. 10 mm./hr.
Haemoglobin 82 %.
Signs rise in temperature today to 99°.

26-1-52 B.S.R. 6 mm./hr. Temperature still rising to 99° at night.

Blood examination:
Wassermann Reaction ——
Kahn Precipitation Test ——
Gonococcal Reaction test ——

30-1-52. Symptom free. Temperature not rising above 98°.
Sodium salicylate and sodium bicarbonate stopped.

31-1-52. Pain returned in both ankles.

1-1-52. Severe pains in back of both legs. Left ankles am still and sore. Feet quite well in himself but has not been sleeping well for 3 days.
Complaint of soreness in his shoulders.
No longer any cardiac murmurs heard. Heart sound closed.
2-2-52. Right elbow and hand joints painful today. Temperature is now not 103° F.
  Rx. Sodium salicylate gr. 30. Sodium bicarbonate gr. 30. 3 hourly.

3-2-52. Turnbull looks much better; his colour has improved.

7-2-52. Blood exam: Haemoglobin 96%.
          W.B.C.  7,800
          B.S.R. 12 mm. /hr.

11-2-52. Patient discharged.
  Rx. Sodium salicylate gr. 20. Sodium bicarbonate gr. 20. 3 hourly.

14-2-52. Salicylates and soda bic. 4 hourly.

19-2-52. Blood exam: Haemoglobin 90%.
          R.B.C. 4,800,000
          W.B.C.  8,400
          B.S.R. 4 mm./hr.

Stop Salicylates.

21-2-52. Rx. and acetylsalicyl. gr. 20. 1st.

22-2-52. Allowed to get up. Weight 10 stones 2lbs.

1-3-52. Blood: haemoglobin 92%.

5-3-52. X-ray report:

  Hum. Sp. and pelvis.
  Appearance normal.

  Hands.
  Remains in soft tissue swelling best seen at the proximal interphalangeal joint, but no bone changes seen.
  Joint space is preserved.

  Knees.
  Normal appearance.

7-3-52. Weight: 10 stones 1/2 lbs. Temp. 97°. Pulse 80.

Very well. Discharged; to go Ashley Amishi Hospital for a period of convalescence.
Notes from the Ashton Memorial Hospital.

James Turnbull was admitted on 14-3-52 and discharged on 1-4-52.

Treatment consisted of graduated convalescence, full good diet with vitamin supplements, exercise in the fresh air, and occupational therapy.

He was symptomless from admission to discharge. His B.S.R. was 12 mm/hr.

Summary of Progress under Treatment.

1. His symptoms of rheumatic joint pains were controlled by salicylates in fairly heavy dosage; his temperature and pulse gradually settled, into a few remissions, and gradually the dosage of drugs was reduced and aspirin was given instead of the sodium salicylate. Appetite and well-being gradually improved.

It took 4 weeks for control of symptoms to be constant: he was allowed to get up after 2 weeks without symptoms or rise of temperature and was discharged after 2 full calendar months.

2. Cardiac signs disappeared once the acute phase of the disease settled. His colour improved.

3. Blood examination repeated shows a gradual improvement in haemoglobin, from 75% to 92%.

The B.S.R. is the most important guide to progress: it fell from 90 mm/hr on 3-1-52 to 44 mm on 19-2-52 and then was steady at 12 mm/hr after discharge & convalescence.
Diagnosis

Rheumatic fever.

Reasons for the diagnosis.

1. History of fleeting joint pains in many joints in a young man of 23. The joints were stiff, swollen, and very painful to move but would recover overnight and other joints be affected next day.

2. Relief of symptoms with sodium salicylate and sodium bicarbonate; also temperature controlled.

3. Increased temperature (101°F), pulse (100/min) and blood sedimentation rate (90 mm/hr.)

4. Soft blowing systolic murmur heard in the heart during the acute phase of the disease.

5. Little nephritic change in the joints apart from swelling; and this was temporary only.

Differential diagnosis.

The diagnosis is very straightforward in James Turnbull's case but the following conditions must be remembered as they can cause confusion in diagnosis—

1. Acute rheumatic arthritis.

This may occur in young adults, but is more frequent in females. The onset is usually insidious, even many months the patient noticing stiffness and aching in the small joints of the hands usually, but may be acute, rarely. The larger joints are seldom affected. The pains are not fleeting in character and are not controlled by salicylates.

2. Multiple arthritis in acute infectious diseases—especially gonorrhea, scarlet fever, pneumonia, pyelitis, typhoid or syphilis: rarely, dysentery and gout, indolent fever. Here the signs and symptoms of the primary infection would be present, the pains and arthritis not fleeting and not controlled by salicylate therapy.
3. Adult osteomyelitis is occasionally a difficulty if the rheumatism is monoarticular, but not in this case.

4. In serum sickness, occasionally polyarthritis with effusion occurs and this may closely mimic rheumatic fever but again the pains are not fleeting and controlled by salicylates. There is no history of serum therapy.

5. Onset of influenza often occurs with vague polyarthritis but does not last long before being followed by the influenza symptoms and signs.
Case II: Complete Heart Block of Rheumatic Origin

Patient's Name: Alexander Stewart,
60 Craigbrook Road,
Glasgow.

Age: 77 years. Occupation: semi-retired consulting engineer.

Recommended by: Dr. Membrino Meprees,
25 Castle Terrace.

Admitted to Ward 23: 29-1-52.
Died: 10-2-52.

Complaints on admission:
1. Breathlessness for many years, increasing recently.
2. Disability from 1 with cough and ankle swelling necessitating bed rest since October '51.
3. Drowsiness, mental confusion for 2-3 days.

History of Present Complaint:
This story was given by his daughter when she came up with him as Mr. Stewart was too distressed and confused himself to give a clear account of his illness.

He has had rheumatic fever three times, last in 1921, and has had "rheumatic heart disease" ever since.
This however did not interfere with his life and he led an active existence until about 10 years ago when he began to get breathless.

He continued to go to his office, with increasing difficulty, and recently not every day, until October 1951. At this time he noticed swelling of his ankles, a cough, and severe breathlessness.

On advice from his doctor he went to bed and has been in bed ever since except for a few days when he...
felt better when he would sit quietly by the fire.

Three weeks ago (8-1-52) his condition deteriorated and he became very short of breath and distressed. His ankles oedema increased and his arms and hands became swollen. His condition has varied daily since then, but has not been good. For 2-3 days he has been mentally confused. Mr. Stewart has never complained of pain.

He was admitted because his wife is no longer fit to look after him: she is 77 years old and crippled into rheumatism.

Past History.

Rheumatic fever three times, last in 1921.
No other diseases: occasional bronchitis.
He had a "tumour of R. breast" removed in 1931.
No other operations.

Family and Social History.

His wife is 70 as already mentioned, and semi-cripple. His daughter is married and very well.

Little other information is available: no history of tuberculosis in the family, or any other member with rheumatic history.

Examination.

General Appearance and attitude.

Mr. Stewart is a small man who is obviously very ill. He is cyanotic and very breathless, sitting propped up in bed, and pays little heed to what is going on around.

His colour is poor: his lips and ears a dull bluish red, the rest of his face pale and sweaty, with a slight yellow tinge. Nasal membranes look pale. His sclera look slightly jaundiced.

Respiration — Rate/minute: regular and even.
No finger clubbing seen. No other pathological features seen.
His mental state is difficult to assess, also his intelligence as he is difficult to rouse and slightly disorientated.

Cardiovascular System.

Pulse. Rate 30/minute. Actually irregular in time and force (difficult to feel when it is so slow.) The pulse wave is large and each beat is well sustained. The vessel wall of radial artery is easily palpable and hard but is not continuous.

Blood pressure. 140-70.

Peripheral vessels. Some venous engorgement in the neck - not severe.

Heart. Aperic heart felt in 6th intercostal space.

No pulsation can be seen in the chest. The heart sounds on auscultation are very weak and faint; all beats are transmitted to the peripheral circulation i.e. no pulse deficit.

Heart sounds appear closed but are too faint to exclude the presence of murmurs. Sounds are faint at apex preduction but, being slightly in 1st sounds intensity,

Signs of cardiac failure:
2. Liver: edge palpable below costal margin.
4. Ankle oedema: ++ +
   Foot oedema: ++ + Both markedly present
   - patient is in Congestive cardiac failure

Respiratory System.

Nose, throat. No obvious abnormality.

Chest. Well developed, not barrel-shaped. Moves quite well on inspiration, but respiration are shallow.

Trachea central, movements confirmed.

Percussion shows dullness at axillary bases.

On auscultation, breath sounds are normal vesicular except at the bases where they are faint: crepitations heard at both bases. Vocal resonance diminished in both bases.
Abdominal System:

Mouth: Rather dry but clean.
Teeth: None.
Tongue: Large and rather smooth, tremulous.
Abdomen: Moves quite well on inspiration; seems rather full and distended in the upper part especially - epigastrium and 1st hypochondrium. Very thin, little fat.
Skin: Rather dry and flaky and atrophic.
Muscle tone is rather poor; but nothing abnormal can be palpated except the liver edge which is also demonstrated below the costal margin by percussion. Enlargement is about 2 fingerbreadths. Spleen not felt.
Kidneys not enlarged.

Urinary System:

Bladder: Kidneys - nothing abnormal
No symptoms related to this system.

Urine:
- Colour: yellow
- Muriatic acid
- S. G.: 1016
- Alkaline slightly acid
- Sugar

Microscopy: no abnormal features. No c.b.c.s.

Lymphatic System:

Spleen: Not felt.
Glands: None felt enlarged.

Central Nervous System: Intelligence intact, but drowsy.
Pupils: Small, react. Mental state varies daily to light and accommodation slightly.
Conjunctival reflexes: present.
Cranial Nerves: No abnormality found; tongue though fibrillating was not extended centrally.
No facial weakness, loss of sensation.
Ocular Fundi: normal in appearance. No changes in vessels.

Reflexes: all present, equal, but feeble and slow.

Plantar responses: flexor.

Abdominal reflexes: all absent.

Locomotor system:

No abnormality found, but muscle tone poor.

Joints all appear normal.

Further Investigations.

1. Electrocardiogram. 6-2-52.

Report:

Complete heart block. Atrial rate 75/minute.

Atrioventricular rate 39/minute.

Left axis deviation with broadening of the QRS complex to 0.16 sec.

T waves inverted in lead I, low inverted lead II, low upright in lead III.

V1: broad Q, small initial deflexion, T upright.

V2 - V3: broad R. " " " T inverted.

V4 - V6: small R. broad S, upright T.

V4, V5: deep Q, small initial deflexion, T upright.

V2 - V4: small R, deep S, upright T.

V5: small R, deep S, inverted T.

V6: broad R, single initial deflexion, inverted T.

Record of left bundle branch block.

2. B.S.R. 2 mm 1st hour.

Hemoglobin: 10.2%
Treatment and progress.

29-1-52. Mr. Stewart admitted in gross congestive heart failure. Restriction: 6 oz. Blood was quickly removed, causing fall in blood pressure but no other change.

Rx. Mercuroyl 2 c.c. (1 M).
Rx. Penicillin 300,000. Distington daily.
Rx. Chloral hydrate. gr 30.

30-1-52. Still weak and very distressed. Pulse steady 30.
Rx. Digoxin 0.25 mg. t.d.s. Urine 4,740 cc.

Rx. Mercuroyl 2 c.c. (1 M).

B.P.

120/70.

Rx. Chloral hydrate. gr. 20.
Repeat mix. 20. I a.m. 20. I at night.

3-2-52. Mr. Stewart's heart failure is still present and he is still in poor condition; sets up a little better.

Pulse 29/minute. B.P. 130/60. Urine 1080 cc.

4-2-52. Penicillin stopped; total dosage 2,100,000 units.

5-2-52. The patient seems a little brighter; still not well.
Rx. Mercuroyl 2 c.c. (1 M).

6-2-52. E. C. G. done—see above.

Urinary output 1800 cc. B.P. 120/70

7-2-52. Condition slightly better but far from satisfactory.

8-2-52. B.P. 120/70. Weight: 105. 3 lb.

9-2-52. Little change in his condition. Rather restless.

10-2-52. B.P. 120/70. Patient a little better in morning.

Sudden death after lunch:

Digoxin 0.25 mg I. V. I in morning as condition deteriorated had no effect; then sudden collapse and death occurred.
Diagnosis.  I. Heart block — complete

Reasons for this diagnosis:
1. Steady pulse rate of 29-30 /minute. 1st sound varying in intensity.
2. History of chronic cardiac disease.
3. Electrocardiogram:
   - atrial rate 75/minute quite separate from the
     ventricular rate of 39/minute: the ventricular beat
     varies in intensity (as seen felt at the pulse) with
     varying relation to atrial contraction.

   The widening of the QRS complex is proportional to
   the slow rate — to maintain an adequate output each
   contraction must be a long, strong one.

   The inverted T waves are indicative of digitalis
   treatment.

   The rest of the picture seen will be discussed later.

4. Absence of any other pathological cause for
   increasing breathlessness and distress:
   i.e. no lung fibrosis, emphysema.
   no anaemia
   no signs of coronary disease

Causes — Rheumatic Heart Disease

1. History of rheumatic fever
2. Long standing chronic valvular disease of the heart.
3. Absence of other pathological causes.

Differential Diagnosis.

1. Incomplete (2:1) block.
   Here the ventricle responds to every 2nd atrial impulse and
   so the ventricle beats regularly at a slow rate — 30-40/minute.
   The 1st sound is very constant in character. Often (MUR) a
   change from 2:1 to 1:1 normal rhythm.
   The E.C.G. is complete proof.

2. Sinus Bradycardia.
   Occurs usually in young people and is usually symptomless.
It may also occur in convalescence after some fever e.g. pneumonia, with severe jaundice and with increased intracranial pressure whether due to cerebral hemorrhage or intracranial neoplasm, or infection.

There is no evidence to suggest that any of these conditions are present in this case.

Other causes of complete heart block.

1. Anterior infarction.

Mr. Stewart’s blood pressure on admission was 140/60 mm Hg but did never rise above 150 mm Hg. His systolic murmur was actually heard in heart block.

His cardiac area was palpable but not necessarily pathologically so in a man aged 77.

In his funds there were no changes and if he had had an atrioventricular block it is very likely that he would have had further changes (E.C.G.)

He had left ventricular enlargement but that is accounted for by chronic rheumatic valvular disease.

2. Coronary occlusion with or without arteriosclerosis.

There is no evidence of this being present.


5. Syphilitic involvement by gummatia or diffuse syphilitic endarteritis is now very rare: and no proof for.

6. Digitalis poisoning: other symptoms are present.

II. Congestive Cardiac Failure.


2. Presence of cardinal signs of cardiac failure.

(i) Enlargement of both auricles
(ii) Respiration of long breath
(iii) Enlarged Liver
(iv) Oedema of sacrum & ankles

3. Signs in chest—heart sounds faint and weak.

4. Mental deterioration and disorientation.

5. Alleviations:—Cerebral anoxia.
Case: III. Chronic Rheumatic Aortic Stenosis with auricular fibrillation.

Patient's name: Mrs. Christina Inglis

10 Whitecross Crescent
Inveresk
 Musselburgh.

Age: 63 years. Occupation: housewife.

Recommended by: Dr. Aitcheson,
Bridge St.
 Musselburgh.

Admitted to ward 23: 24-9-51
Discharged: 9-11-51

Complaint on admission:
1. Attacks of breathlessness at night for 5 years.
2. Breathlessness on exertion for 1 year, much worse in last 6 months.
3. Swelling of the ankles for 8 months.

History of Present Complaint:

Mrs. Inglis enjoyed quite good health until about 5 years ago when she began to get attacks of breathlessness during the night: these increased gradually in severity, making it necessary for her to get up and sit at the open window to get breath. She experienced no pain, but a gripping feeling and a pain not to be able to draw breath. These attacks would last from 3-4 hours and she was unable during them to speak or move. About 6-7 years ago she was kept at rest in bed for 3 weeks by her doctor, then gradually allowed up.

She began to feel rather tired and exhausted at about this time, but improved after her rest in bed and was able to carry on her household duties.

During the last 18 months she has had very few nocturnal attacks but has had increasing breathlessness
on exercise during the day, and for the last 9 months she has noticed something of her ankles at night which has been gradually increasing in amount, and coming on earlier in the day.

For the last month she has had to keep her bed at home, propped up on 6 pillows, breathless and with constant ankle edema. Weekly injections were given by her doctor.

Her weight was always steady until 9 months ago. She has lost about 1 stone since then. Her appetite has been poor for the last month.

Her bowels are regular: she is never constipated.

She has had to rise 3 times heavily every night because of the ankle water.

She has no cough, no pain, no indigestion.

Past History.

When 17 years old Mrs Hughes had "rheumatism" - pains in all her joints.

When 36 years old she had pains in her legs and ankles and was in bed for 6 weeks, off exercise 9 months.

When 40 she had inflammation of the bladder and spent 3 weeks in bed with a fluid diet.

She had no trouble when she had her children, or afterwards.

Family History.

Her brother and father both died at the age of 65 - her mother of a "shock," her father of urinary retention.

She has 2 brothers - one is very well and one other has had pulmonary tuberculosis for 5 years.

Family: she had 5 children; her 3 sons are all well but both her daughters died of tuberculosis when 16 and 17.

Her husband was killed in an accident 15 years ago.
Examination.

General appearance and attitude.

Mrs. Inglis is a little old lady, who looks if anything older than her 63 years. She is very thin and wrinkled and pale and looks very worried.

Her lips and ears look cyanotic: her mucous membranes pale.

Although sitting propped well up in bed she is breathless and does not look well.

She is mentally alert and co-operative, willing to tell her story.

Cardiovascular System.

Radial Pulse: Rate is 78-80/minute, totally irregular in time and force. Some beats are well sustained, others very weak and poor. There is no semblance of regularity.

The vessel wall is palpable, not very hard or tortuous.

Blood pressure: 136/60.

Peripheral vessels: not palpable except the radial.

Veins in the arms slightly engorged.

Heart: Thrust seen in the 6th interspace outside the mid-clavicular line: open beat confirmed by palpation. 3 fingers' breadth outside this lies in the 6th space.

A rough thrill was felt over the 3rd and 4th ribs just to the Right of the sternum.

On auscultation: a rough harsh systolic murmur was heard all over the precordium including the mitral area best heard in the aortic area and propagated far up into the neck. The 2nd heart sound was soft in character.

No presystolic or diastolic murmur was heard in the mitral area.

The heart sounds varied in volume and were totally irregular: mitral rate 100; pulse rate 80.

Abdomen: + + +

Liver enlargement: 3 fingers' breadth. Lower costal margin
Respiratory System.

Noe and throat: No obvious abnormality.

Chest:

Very clear, moves well with respiration. Apical beat clear.

Trachea is central, movements confirmed, vocal fremitus normal.

Palpation revealed a resonant percussion note all over chest.

Breath sounds were normal, vesicular, no accompanying

There were no respiratory sound in lung bases.

No finger clubbing.

Alimentary System:

Mouth: rather dry, healthy.

Teeth: old fillings.

Tongue: slightly furrowed.

Throat: normal.

Abdomen: very firm, rounded, no skin dry and able to

move easily on respiration.

Nothing abnormal seen, muscles too quite good.

Palpation revealed enlargement to 3/6 of the liver.

Spleen not palpated, no other stomach felt.

Urogenital System

Kidneys, bladder not palpable or tender.

Urine: Colour: yellow.

Specific gravity: 1020

Reaction: acid.

Albumen: 1+ trace.

Sugar: nil.

Blood: nil.

Lymphatic System

No spleen or glandular enlargement.

Motor System:

No abnormalities of bones or joints.
Central Nervous System.

Pupils. Equal, react briskly to light and accommodation.

Fundii. Show no abnormality except slight general pallor.

Cranial Nerves. Show no abnormality.

Eye movements good; no facial weakness or sensory change: tongue protruded straight; muscle tone good.

Refrains. All present but rather feeble and difficult to elicit. Plantar responses flexor.

Temp. 98° F. Respiration 40/min.

Further Investigations.

1. Electrocardiogram
   Shows auricular fibrillation; ventricular rate 120/min.; left axis deviation.
   
   Head I: T wave inverted
   Head II: Slight ST. depression, inversion of T wave
   Head III: T wave low upright.
   
   V2, Moderate T, deep S (50 mm), T upright
   V4, Tall R, Moderate S, ST depression, T inverted
   V5, Small Q, Tall R, ST depression, T inverted.
   Gross left ventricular hypertrophy.

2. Blood: haemoglobin 65%.

Treatment and Progress.

24-9-51. Mrs. Hughes was admitted
   very breathless and in cardiac failure with fibrillation.
   
   Rx. Digoxin 0.25 mg. q.i.d.
   Rx. Aminophylline 2 G. t.i.d.
   Rx. Chloral hydrate 90.20.
   Nepenthe min. 20 / at night

25-9-51. Still breathless and I.V.
   
   Weight 7 stone 8 lbs. (Ideal for Ht. 5’4” - 9st. 12 lbs)
   Pulse, 100 (min.)

Mrs. Ingle's appears brighter, not so breathless.


3-10-52. Reduced. Rx. Digoxin 25 mg. b.d.
Weight: 6 stone 13 lbs.
Pulse rate: 70-90/min. Mrs. Ingle much more cheerful.

6-10-52. Rx. Ferrous sulphate gr. 3 b.d.

7-10-52. Pulse 70-80/min. Sickness subsiding now and flatulence.

Stop Ferrous sulphate.

Rx. Iron and ammonium acetate gr. 30 b.d.
Rx. Ascorbic acid 50 mg. b.d.
Rx. Magnesium trisulphate if required after meals.

10-10-52. Blood examination:
Hæmoglobin 76%.
R.B.C. 4,090,000 per c.mm.
W.B.C. 8,000

Differential diagnosis count:

- Polymorphs 61%
- Monocytes 19%
- Lymphocytes 27%
- eosinophils 7.1%

13-10-52. Pulse rate 70/min. Weight: 6 stones 11 lbs.

Digoxin stopped

14-10-52. Still good response to Mercurol.


Condition much improved. Good diuresis by mercurol, and urine still albuminous. Mrs. Ingle allowed up for 2 hrs. to be increased daily; she says she is feeling grand.

28-10-52. Mrs. Ingle feels much better, is able to stand up and down without breathlessness for 10 minutes.


She continued to improve; her pulse never regular but slowed to a steady 65-75/min.
By 9-11-52 her weight was 7 stone 2½ lbs, her exercise tolerance greatly improved, her pulse still irregular but ranging little from 70 to 90 and she could ambule better. She was discharged home on this day.

Summary of progress under treatment:
1. Her breathlessness was gradually controlled, to better with oedema and mild venous congestion, by the giving of
   1. Digitalis
   2. Mercurial
   with rest in bed.
2. Her pulse was slowed from 100 to 70
3. Her general condition improved, her haemoglobin rose
   with feroxous sulphate from 65% to 92%
   She was made sick with ferrous sulphate and so was
given iron + ammonium citrate.
4. By the time of discharge she could walk round the ward twice or three times, without any breathlessness.
5. Ankle oedema persisted largely treated through a
good diuretics was obtained, and it was ignored as
the other signs of cardiac failure cleared up.

Diagnosis: Chronic Rheumatic Valvular Disease

- of the heart
  - Aortic Stenosis
  - with auricular fibrillation
  - Chronic left ventricular failure and
    more recent right-sided failure.

Reasons.
1. Chronic rheumatic valvular heart disease.
   a) History of rheumatic disease when patient was 17 and 36.
   b) History of paroxysmal nocturnal dyspnoea with
      no evident hypertension.
      Blood pressure never above 140/78 mm Hg.
      Retina normal; peripheral vessels not generally
      palpable.
      No symptoms of dizziness, faintness, headache.
(a) Gradually decreasing exercise tolerance with no other pathology, e.g. no gross anaemia, severe infection, pulmonary disease.

(b) Presence of aortic stenosis which is most commonly primary.

II. **Aortic Stenosis**

(a) Heart signs: a rough systolic murmur heard in the aortic area to the right of the sternum in the 2nd intercostal space and propagated into the neck and all over the precordium even to the apex, in conjunction with a systolic thrill over the aortic area is diagnostic of aortic stenosis.

1. Left ventricular enlargement as seen
   (i) by downward and outward displacement of the apex beat
   (ii) by electrocardiography, showing left axis deviation.

(b) History of a gradual left-sided heart failure in the absence of hypertension or aortic regurgitation.

III. **Aortic Insufficiency**

(a) Pulse totally irregular in time and force.

(b) Heart sounds totally irregular in time and force.

(c) Apex pulse deficit (100:80)

(d) No evidence of hypotension or coronary insufficiency or hypertension; no other usual causes.

(e) Presence of cardiac failure a common consequence.

IV. **Chronic left Ventricular failure**

(a) History of attacks of Paroxysmal Nocturnal dyspnoea for about 5 years

(b) Increasing breathlessness on exertion → Orthopnoea on admission.

(c) Presence of aortic stenosis and left ventricular hypertrophy.
Recent right-sided cardiac failure.
(a) History of swelling of ankles in last month.
(b) Cardiac signs:
   1. Venous engorgement in neck: +++
   2. Liver enlargement: +++
   3. Ankle oedema: ++++
(c) Loss of appetite, recent indigestion.
(d) Arteriogram: with no other cause found.

Differential Diagnosis

In a woman of 63 complaining of paroxysmal nocturnal dyspnea and breathlessness on exertion and recent ankle swelling:

1. Hypertension:
   (a) No headache, dizziness, faintness, vision trouble
   (b) B.P. normal, funds normal.
2. Pulmonary disease causing breathlessness:
   e.g. fibrosis, advanced tuberculosis etc.
   No physical signs or symptoms.
   Unlikely at this age without being recognized before: no typical signs.
4. Myocardial degeneration
   No pain complained of.
5. Gross anaemia.
   Her haemoglobin 65/0 may seem out but it could easily cause all her symptoms.
6. Syphilitic heart disease
   No history of syphilis: W.R. -W.
   No signs of aorti incompetence, the usual lesion, or aorti aneurysm.
Case IV.  Rheumatic Mural Stenosis.

Patient's name:  Mrs. Jessie Weyde,  
8/0 Watt,  
9 Murdoch Terrace,  
St. Andrew

Age 33.  Occupation:  waitress.  
Recommended by:  Dr. Pole,  
142 Gilmore Place.

Admitted to ward 24:  5-11-51.  
Discharged from ward 24:  15-12-51

Complaint on admission.

1. Breathlessness of many years duration.  
2. Tiredness and depression for 3 days.  
3. Coughing up blood - 12 hours.

It is necessary in Mrs. Weyde's case, if a clear picture of her illness is to be seen, to describe her history from the beginning.

History.

1931.  When she was 13 she had an acute attack of rheumatic fever.  She was in bed for 1 year; her legs were mostly affected.  She then had 6 months convalescence before returning to school for her last year.  She was at that time quite fit for the normal school routine but did not run or play games or attend gymnastic classes.  She left school when she was 15 years old and got a job in the North British Rubber Co. as a clerk.  Going to Kennedy's Commercial College in the evenings.  Her health was good at this time but she got bronchitis every winter.
1940: When she was 22 an illegitimate son was born to her. She had no trouble in pregnancy beyond some slight breathlessness in the last trimester. The child weighed 8 lbs. 7 ¼ oz. In the puerperium she had breast abscesses on both sides which required surgical treatment.

1941: She returned to her job in the North British Rubber Co.

1945: She left the N.B. R. Co. to marry a Dutch sailor and went off to Holland with him, leaving her son George at home with his grandmother as mother.

She lived in an American-style flat in Amsterdam and was very happy there: but she found it damp, got bronchitis, and coined up blood in September 1945. She recovered from this very quickly: only a few dots had come up.

1947: She began to feel breathless and tired, had no appetite and started to lose weight. Her skin became pale and even sallow. Her housework became very heavy for her. Then one morning she woke at 3 a.m. coughing up blood. The bleeding was profuse and continued, so that she was taken into hospital. She remained in hospital until December and was then discharged on a maintenance dose of digitalis, 1 tablet daily. She was advised by her doctor to return to Scotland.

Dec.: She sailed to Hebrides in mid-December and her husband who had been at sea for all those years came with her to Inverlochy and got a job in the shops in Inverlochy.

He started drinking with friends, very heavily and shortly after this he disappeared and she has not heard of him since.

She now stayed with her mother and her little boy and made the best of a bad job.

She got a place in the Home Hotel in Shusha earn place as a waitress, can opener and
was quite fit enough for the hard work that was entailed; she did get very tired in the tourist season when she was on her feet for 10-12 hours every day.

August. She came home from work, feeling quite 1950. tired out and was taken at night by another haemoptysis — not however so severe as before. She spent a week in bed. She was sent to Spittal St. for investigation for tuberculosis, and a negative report was obtained. She had a bronchopneumonia diagnosed and went to the Western General Hospital where she was treated with digitalis and oxygen, penicillin.

Dec. She got home just before Christmas.

She felt well on her return home but took things easily as she got breathless very easily, especially going upstairs. She has had this for so long now that she cannot remember not being breathless on climbing stairs quickly, but she notices extra effort.

March. She returned to work feeling "champion."

May and she remained all summer, working a 7-day week from 7.30 am till 11.30 pm.

August. On Sunday on blowing her nose she saw blood.

Sept. 21 previous week had found her more tired than usual and she had become pale. Her Digitalis tablets had run out 3 weeks before and she had not bothered to get more.

3-9-57 Next morning she felt ill and stayed in bed.

On getting up after lunch she had a fairly severe haemoptysis. and was admitted to ward 24, with a sided loban pneumonia + decompensated mitral stenosis.

**Synopsis of treatment.**

Rx Penicillin 100,000 units 4 hourly.

Rx Digoxin .25 mg tid.

Rx SERIOUS sulphate 93 tid.

Temperature rapidly settled in 5 days.

Routine U/S. was found to be strongly positive so she was seen as an out-patient in ward 46 and treatment started with penicillin. It was suggested
(but before treatment with bismuth was started she should be admitted to the ward (24) for penicillin coverage while her teeth were removed as they were very carious.)

She was then discharged on 22-9-54 stabilised on digoxin, 25 mg. daily.

She stayed at home, feeling much better, but depressed and irritable. She managed to do shopping and lighter housework—not polishing or scrubbing—without being unduly breathless unless she strained.

November 4th. She began to feel poor—she could not sleep at nights, got a bad taste in her mouth, became swollen—"yellowish" and had a dull ache in her left lower chest.

November 5th. She was woken at 4 am by violent coughing up of blood—a large amount of blood. Large clots were coughed up and she was very frightened.

She was admitted to ward 24 in her early evening.

She says that during the last few months she has not been able to sleep at nights without pillows propping her up. Twice recently she has woken in the night wheezing and breathless.

On exercise if she gets breathless she tends to become dizzy with black spots in front of her eyes; her hands are clammy and she feels nervous, "butterflies in her chest."

She smokes 2 cigarettes a day occasionally drinks brandy.

Since admission,

Mrs Beagley says that she has improved enormously; her appetite is very good and her breathlessness goes.

Social History.

As a child she says she had "everything she wanted." Her father was in the fruit trade and she had a very good mixed diet.

Her home is a flat in a tenement—where she lives now with her father, mother and her son. Her younger sister is getting married on November 24th.

She had only measles and whooping cough as a child. No operations.
Examination.

General appearance and attitude.

Mrs. H is a very slender pale young woman and her very bad teeth are soon noticed. She is fairly thin but active and her muscle tone good. No signs of cyanosis or jaundice. No venous engorgement - pale. No finger clubbing.

She was breathless only on admission for a few hours.

She seems to be intelligent and co-operative and knows quite a lot about her condition.

Cardiovascular System.

Pulse: Rate 80 (normal), regular in time and force, was well sustained. Vessels wall not palpable.

Blood pressure: 120/80.

Peripheral vessels: No venous engorgement pulsation.

Heart: On inspection a strong thrush is obvious in the 5th space 1” outside the mid-clavicular line.

A thrush is felt over the area diaphragmaticri tone.

The apex beat is confirmed on palpation, loudest in character. No other faults felt.

On auscultation:

Mitrval area: a rushing, decrescendo presystolic murmur is heard which is propagated right sound into the axilla; a definite mid-systolic murmur is also heard. The first sound is slapping in character.

Aortia area: the mitral presystolic murmur is heard even in this area. The second heart sound is very loud in this and now especially in the 2nd left intercostal space at the edge of the sternal border pulmonary area.

No oedema or cyanosis.
Respiratory System.  
Nose + throat: normal  
Chest: moves well on respiration. Respirations rapid.  
Spumum is still good stained (6.11.51)  
Trachea is central, aper beat 5th space 1" outside R.  
Percussion reveals resonance all over lung fields.  
Breath sounds are normal vesicular; a few faint crepitation over both sides were heard.  
No finger clubbing.

Digestive System.  
Mouth: dry  
Tongue: furred  
Teeth: very bad - carious in every tooth: many are right  
down to the gums.  
Abdomen: moves well with respiration. Muscle tone good.  
No abnormality made out.  
Liver edge not palpable.  
Bowels: Regular.

Urogenital System.  
Kidneys, Bladder not palpable.  
Micturition: Periods regular at 21, quite a heavy loss.  
No trouble with micturition.  
Urine: Colour: amber  
5·6 (0·25)  
Reaction acid  
Albunen - -  
Sugar - -  
Blood - -  
Microscopy - -

Lymphatic System.  
No spleen, no glandular enlargement.

Genito-Motor System.  
No abnormality seen or complained of.
Central Nervous System:

Pupils: equal, react briskly to light and accommodation.

Fundus: show no abnormality.

Cranial Nerves: no abnormality.

Reflexes: all present and equal: tendon brisk.

Plantar responses flexor.

Abdominal reflexes present.

Temperature 98.9° F. Respiration 25
Pulse 80/min.
Weight: constant usually at 7 stone 9 lbs. 10 lbs.
Height: 5' 2".

Progress and Treatment.

6-11-51.
Mrs. Weight is still breathless and weak.

Rx: Penicillin - discontinue. 300,000 units daily.

Blood examination:

Koagulase 75%.
W.B.C. 7,200 / c.mm.
B.S. R. 28 mm.
R.S.C. 14,800,000 / c.mm.
Kahn ++

10-11-51.
Breathlessness now gone. Sputum no longer stained red.

Rx: Penicillin 300,000 b.d.

13-11-51.
Cerebrospinal fluid: ++ 73° culture.

14-11-51.
Blood culture - neg.

C.S.F. report: Protein 20 mg.

Sugar 60 mg.

Cells 3.

16-11-51.
Rx: Penicillin 600,000 units b.d.

19-11-51.
Dental extraction.

20-11-51.
Mrs. Weight feels quite well today, but her gums are a little sore. There is blood clot in the tooth socket, a haematoma on the inner surface of the lip.
22-11-51. On examination of the heart, a faint systolic murmur was heard in the aortic area.

Mrs. Weydel's mouth is still very tender.

23-11-51. Allowed up, she feels very well.

26-11-51. Weight 7 stone 6 lbs.

29-11-51. On attempting to climb stairs she felt very dizzy with rigidity in her arms and palpitations. She has had this before coming into hospital. Her jaw on left side is still sore, but the gums are clean and healthy.

Penicillin is still being given.

30-11-51. Weight 7 stone 12 lbs.

Mrs Weydel is up most of the day now, keeping well.

3-12-51. Had very severe dysmenorrhea on the 1st day of her period. Felt quite ill.

4-12-51. Felt drowsy, vomited twice; cramping pains in her stomach caused retching. Rx: Henrici 9/4.

5-12-51. Vomiting again: she does not remember any more. Thin: opii. min. 20, given twice.

4-12-51. W.B.C. 1 p.m. 8,000
        4 p.m. 8,700

B.S.R. 12 mm. /hr.

C.S.F.: Cells 2 /cu. mm.

Protein 30 mg

Sugar 54 mg

Chlorides 696.

Rx. Sulpha 250 mg loading dose.

1/2 G. 4 hourly

Rx. Potassium citrate 90 20 1/2 hourly.

Sodium bicarbonate 90 20 1/2 hourly.

6-12-51. Stop Penicillin.

7-12-51. C.S.F.: W.B.C. : 0.001 /cu. mm.

Cells 2 /cu. mm.

Gold test 0000000000.

10-12-51. She now feels much better but tends to get dizzy.

Recovers well of the 5th, 9th and 12th of November.
Her appetite is returning to normal again, her
dizziness is decreasing.

11-12-51
Weight: 7 stone 13 lbs.

14-12-51
8 stone 2 lbs.

Steady improvement.
She went to board 46 to be examined: nothing
was found. She had been absolutely astonished when
and how she had been able to find out her state of
health as she had had no symptoms or signs. To start
bisulphate therapy on January 15th as her gums are
healed.

On walking a lot she tends to be breathless
and yields, sweating, with palpitations, but this
is improving.

Her appetite is now "enormous."
She was discharged home.

Summary of treatment and progress:

Her slight decompensation was quickly settled by a course of
digitalis with rest.

Penicillin was given in large doses in view of her
crepitus teeth, which would have been an ideal starting
place for a subacute bacterial endocarditis.

The carious teeth were removed under penicillin
cover.

Two weeks after she had a hemoglobin reaction
and so the penicillin was stopped and sulfadiazine
started.

She made uneventful recovery after this and the
discharge was very well, but still becoming easily
breathless on exercise.

She has gained about 6 lbs in weight and is
eating well, undergoing treatment for syphilis.
Diagnosis: Mitral stenosis of rheumatic origin.

Reasons for diagnosis:

1. History of rheumatic fever when 13 years old.
2. Recurrent haemoptysis in a young woman with no tuberculosis — proved by chest X-ray — clean. Sputum culture — negative.
3. Breathlessness on exertion, increasing.
4. Physical signs:
   Typical findings: apical mid-diastolic and presystolic murmurs propagated well into axilla. Auscultation of 2nd sound at 2nd left interspace by sternum.
   Sharp first sound ("slapping") at apex.
   These are diagnostic of mitral stenosis.

Differential Diagnosis:

Haemoptysis in a young woman.

1. Tuberculosis pulmonary
   Fever, temperature would be raised and cough + sputum + physical signs present.
2.
Case V

Chronic rheumatic endocarditis
with subacute bacterial endocarditis.

Patient's Name: Stewart Boyd Taylor
63 Halves St,
Admore Place.

Age: 14 years. Occupation: schoolboy.
Recommended by:
Dr. Henry, Admore Place.

Admitted East Fornax Sanatorium 26-9-51.
Readmitted to Ward 23, R.I.E. 5-10-51.

Complaint on admission:
1. General irritability, restlessness, anorexia - 6 weeks.
2. Loss of weight, stomach ache - 4 weeks.
3. Severe frontal headache spreading to top of head.

Seven vomiting and prostration - 1 day.

Again it is necessary to tell this boy's story from the beginning to see its development of his present condition.

History.

1942. When Stuart was 5½ years old he contracted scarlet fever which was immediately followed by an attack of rheumatic fever. He has been told of this, remembering very little. He was off school for 6 months at this time. Of this he thinks 4-5 months were spent in bed, at least 3 months being complete bed rest.

His parents seem to have been told at this time that his heart was diseased as he was not allowed to play games with the other children.
1947. He was well until 1947 when he became ill and was taken to the Infectious Diseases Hospital as a case of pneumonia, and rheumatism was diagnosed eventually. He thinks he was in hospital for 3 weeks, and then 2 months at home.

1947-1948. He had "burst ear drums".

After this his parents moved out to New Mill Burn and he attended school regularly; he does not remember being ill except once with pain in his knee - he was ill for a week at this time.

He seems to have had odd joint pain on and off since this time.

1948. He got tookeh and when his mother took him to see 4 different dentists each refused to extract his cavious teeth on hearing his story.

1949. He returned to Edinburgh, to Harley St. with his family. He has attended school regularly and is now in his 3rd year - with boys of his own age.

His activities have been normal; he is in the Technical and Commercial class and gets on well. He has played the bagpipes for 2 years and is in the band of his Scout Troop. He has played Rugby Football (in his summer) for his school and also Cricket. He has never been breathless.

1951. He was on holiday in August in West Hinton August and helped with the hay, working in the fields with another boy. On the 26th he experienced pain in his back - sharp, spreaded up behind the scapulas spreading half the length of his back. He was in bed for 5 weeks then up and about.

Just before this time his parents noticed that he was irritable, restless, easily annoyed. He said himself that he was 'grumpy' and said 'nobody could do anything right'.
He lost his appetite and began to complain of slight headaches all over his head: he sweated heavily at night, did not feel well. He lost quite a lot of weight after his holiday, and looked pale.

September. He was ready to return to school when, 2 days before, he got severe headaches. He did go back to school, but had to return home after half a day, complaining of pain in his right side area. He stayed off school for 1 week with slight headaches and vague stomach ache.

He returned to school 1 day and had to stay away next day - he felt weak and ill.

His doctor came, told him he had a chill and was fevered and had to must stay in bed.

September 24th: In the evening he was sitting reading a newspaper when he got a very severe headache in his left temple, radiating to the top of his head.

He went through to his room and lay down on his bed. The pain stayed in his head and moved down to his neck on the left side. When he tried to get up he vomited all that he had eaten that day. He vomited several times.

When his mother came in she sent for his doctor who sent him up to the R.I.C.

Stated does not remember very well what happened that night or the next day - but the pain in his head was very severe and he could not help screaming and struggling. He vomited on coming to the next night.

(History supplemented with notes from Frank Fontaine
Sarcasticum and Ward 20 R.I.C.)

24-9-51. On admission to Ward 23 the following points noted:

Card. Pupils normal in reaction, equal in size.

Fracture: none.

Reflexes present & equal.

Patient responded 2 & 2.
Next rigidity + + +
Kernig's sign for.

Cardiovascular system.
Rate 120. B.P. 150/75.

Respiratory system.
No cyanosis, proper breathing. Nothing abnormal.

The patient was so restless that 1.5 phenoldehyde 31st evening September 25th. Patient was put on to sedative hemiobid gr. 5 and was still very restless.

Lumbar puncture.
C.S.F. Protein 140 mg%.
NaCl 706 mg%.
Sugar 49 mg%.
Blood stained.
C.S.F. Culture 25/9/51 negative.

September 26th. No change in patient's condition.
Repeat lumbar puncture.
C.S.F. Protein 40 mg%.
NaCl 702 mg%.
Sugar 50 mg%.
Error contaminated with blood.

Blood examination:
Haemoglobin 72%.
W.B.C. 6,400.
B.S.R. 50 mm. in 1st hour.

Presumably he was diagnosed as a tuberculous meningitis for he was sent to East Fortune Sanatorium.

September 27th. Lumbar puncture.
C.S.F. Protein 200 mg%.
Sugar 60 mg%.
Chlorides 720 mg%.
September 28th

Gastric function.

Respir.: Protopic 220 mg.

Sugar 60 mg

Chloride 720 mg.

Confirmatory of subarachnoid hemorrhage.

Stent was then sent back to Wadg 20, R. He remembers this quite distinctly: his headaches were still very severe.

Very slight:

Negative except for horizontal uncorrected on right.

Examination. Ward 20.

* Central Nervous System.

Pupils: equal, round, react normally.

Aud. normal.

Ocular movements: 3 & 4 on 6 dys.

Mark rigidity:

No facial weakness. 1x 1x 1x 1x Normal.

Limbs: tone good; reflexes, power.

Reflections present and equal. Plantar 2\&2.

Cardiovascular System.

Pulse regular, not collapsing, 96 per min.

Heart: Apex beat possible, outside and cranial line.

Diastolic normal, ascending area with 1st. systolic (no diastolic) normal at base.

Respiratory System.

Nothing abnormal.

Abdominal System.

Liver and spleen both palpable. No tenderness.

September 29th.

Stent renders feeling very well with severe headache.

Still, and he noticed double vision for a while when he was in ward 20. He was sweating and restless.

He was treated with sedatives and analgesics.

Sodium chloride 9 gr. 3 4 hourly.

Also oral sedatives on 29th and 30th September.

October 1st.

Improved today. Still mild attacks of headache with distress. No demonstrative neurological sign except slight 1\&2 facial weakness.
R. Sodium Amytal gr. 3.
R. Phystostigme 1 mg. io bid.

October 2nd.

Central arteriogram: taken.

Medication: Morphine gr. 1/6

Operation Notes.

Right Carotid Angiography

Anesthesia: Pentothal induction 0.5 gr. The nitrous oxide and oxygen mixture was used. Anesthesia satisfactory but uneventful.

Procedural Notes: The right common carotid artery was entered percutaneously without difficulty. Plaque bron needles were used.

6 cc. of 35% water-soluble were injected for anterior-posterior and lateral phase films.

When repositioning the head for lateral exposure, a radiograph caused dissection to be closed in the artery and a considerable hematoma was formed. Further 2 injections of 6 cc. of 35% water-soluble was used for anterior-lateral early and late phase films were given.

Total运æurious... 10 cc.

In view of the large hematoma and auscultation, it was decided to inject the left common carotid at the same session. This should be done later.

After this procedure, patient became dyspneic and gastritic and his general condition deteriorated.

October 3rd. Na amyntal gr. 3.\t4 hourly.

October 4th. Condition is still unsatisfactory for angiography. His spleen was definitely enlarged.

October 5th. Pulse temperature 104°, increased in both rigidity and severe frontal headache. Tachycardia. Night right sided papilledema. No other signs.

Repeat: Central arteriogram.

"There is no definite evidence of intracranial pathology but vascular anatomy is a little unusual."
The bifurcation of the internal carotid artery is low in position and unusually closer to the sinus appears to be an unusual vessel arising from the anterior cusp, extending anteriorly but difficult to visualize when it passes the middle cerebral. Little help is given from the antero-posterior projection. Acromionality is seen in the late phase film.

Stuart was sent back to ward 28 - readmitted 5-10-51.

Since admission Stuart says that he feels much better, his headaches continued for some days and gradually lessened so that on October 17th he could say they were gone. His appetite which was very poor before admission has improved very much and he feels brighter, more interested in things, better tempered "cheerier" and generally stronger. He says he has grown since admission. In the last week he felt less tired all the time. Now he feels quite well but not able to get up yet.

He never remembers having had a sore throat: his teeth have been bad for 4 years. 3 years ago he read a bout of bad mouthwash - see history - this gradually disappeared.

Stuart says he has never had bronchitis, diphteria, pneumonia. He has no trouble with night sweats.

Bowels acted normally before admission - constipated since.

He has never been breathless; he had noticed a pain "stitch" in his side on running, occasionally.

He is slightly deaf in one ear since preparation in 1948.

He has lost weight since his hospital. Has had occasional nose bleeds for years. Very many in the last 72 - for no apparent reason.

Family History.

Father: he is 43 and is fairly healthy. He had rheumatic fever when away and has had "heart disease" ever since. He is able to manage his business - is not handicap greatly.

Mother: well
Sister - 18 - works in Kings Square banking office.
No other relevant history: no tuberculosis.

Social History.
Patient's father is manager of a butcher's shop: his mother has just started to look after foreign students, as housewife.

He appears to be comfortably off: the house is big with room

Over town room. Stains smell a bedroom with his brother

Shoe shop: bagpipe and 2 or more other bags; one of his friends has had known 'chest troubles' for 2 years and is still
nder observation.

He previously living at Hillside Farm by Edinbane.

His first home is Edinburgh, new Stevenson Road in

George - a semi-detached bungalow. He had rheumatism from then.

Examination.

General appearance.

Stuart looks younger than 14 and is very slightly pale and

thin. He walks steadily: his eyes are large and intense in his

brown face; his arms are very thin, his fingers long and

tapering and white, lying deeply on his chest. His face is

slightly flushed and his skin clammy; he breathes easily, freely.

Mental state and behaviour.

His intelligence is above normal for his age - he responds

readily and intelligently to questions and was very co-operative

- he does not avoid students listening to his heart if they

learn something from it. He seems a very normal boy.

He is fully aware of his condition, knows that he has

a 'bad heart' but his attitude to it is difficult to

assess: he is fairly philosophical.

General Nutrition. etc.

Stuart is very thin and wasted. Appetite is

now improving: has never had indigestion, cough,

symptom or breathlessness.
Colour. His face is flushed with fever but his hands are cold.
No cyanosis.

Nose: Very pale.

Skin: Moist, otherwise normal. No petechiae, oedema or oedematous tissues.

Nails: Normal. No clubbing.

Hairs: Industry shape but is thin.


Cardiovascular System:

Pulse: Regular 80/min. Heart is quick up and down, there is no regularity, it is not well sustained i.e. is unloading in type.

The force is quite strong. Aorta wall is not palpable.

Blood pressure: 120/70.

Pericardial sounds: No pulsation or venous engorgement.

Heart:

The heart is seen to thrust against the chest wall with every beat. There is a diffuse motion over the left 3rd, 4th, 5th and 6th ribs maximal over 5th or 6th ribs. Below the nipples.

A violent thrust is felt at the apex which moves down and to the left. It is maximal in the 5th intercostal space on the side mid-clavicular line. A finger placed here feels the impact of the ventricles contracting down with each beat. Just outside the intercostal muscles.

A thrill is felt at the apex and also faintly in aortic area.

Heart size as determined by percussion (which on a thin child is usually accurate) is generally enlarged.

Heart Sounds:

1st sound: Entirely obscured in the central area by a loud rushing murmur which starts in the right and crescendos through systole, even slightly muffling the 2nd sound.

A rough early diastolic murmur is also present here.

Both murmurs are heard over a wide area and are propagated into the arterial area where the presystolic and systolic murmurs are heard very clearly.
In the anterior area the first heart sound is obliterated by
a rushing soft murmur - the second sound is very loud
and accentuated. No diastolic murmur is heard here.
The systolic murmur is heard over a wide area, propagated
well up into the neck and down the l. side of the sternum.
It becomes faint at the 4th costal cartilage
It was thus heard to be different from the second
murmur as the two were not continuous and of different types.
The sounds are indicative of two conditions of the
heart valves, taken in conjunction with the history of
the patient five years ago:
1. Mitral stenosis with incompetence.
2. Aortic disease - probably stenosis with incompetence.

Signs of Cardiac Failure:
1. Neck veins: Not engorged
2. Liver: Not palpated
3. Lung bases: No rales
4. Ankle oedema: Not found

Respiratory System
Nose and throat: Sounds slightly blocked at nose.
No abnormality seen.

Chest. Respiration: 20 per minute; normal. well
Trachea central, movement confirmed
Percussion - both lung fields resonant
Breath sounds: Normal vesicular. No complications
No cyanosis, finger clubbing.

Alimentary System
Mouth: Healthy
 Lips: Rather dry
 Tongue: moist, clean
 Gums: slight gingivitis seen.
Teeth: heavily carious, many decayed.
Some teeth badly discoloured and decayed.
Abdomen.

Aperistalsis generally felt, especially in lower half, but little subcutaneous fat. Skin rather thin and atrophic.

Movements on respiration.

On palpation, a large pelvic colon was felt.

The spleen was enlarged slightly on paralyse by

1. Percussion

2. Palpation — 2 to 2½ below rib margin.

It was slightly tender.

Liver not palpable.

Urogenital System.

Secondary sex character undeveloped — small for a boy of 14.

Pubic, no axillary hair.

Herpes. Now found.

Kidneys and bladder not palpable or tender.


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Central Nervous System.

Pupils. Divided. Read briskly to light and accommodation.

Rendi: normal.

Cranial Nerves normal except for ch. depression below.

Reflexes: all present and brisk.

Plantar responses V & V.

Gastrointestinal System.


Lymphatic System. No abnormality seen or complained of.
Further examinations.

October 24th.

Shrewd looks much better: he is brighter, more interested in what is going on. He seems fuller of mischief than before—a good sign in a boy of 14. He is not so flushed and less languid. He says he feels much better.

Cardiovascular System

Pulse regular 60/min.

Heart. Apears beat as before.

Mural area. 1st sound obscured by presystolic and systolic murmur. Which is musical and high pitched. 2nd sound soft. Murmur propagated into the aorta where it is even softer.

Mural area. 1st sound obliterated by a soft "whiffing" murmur in systole. 2nd sound closed, short and loud. Propagated into both x.'s and left subclavian arteries. Soft high pitched diastolic murmur heard over 2nd right interspace—nowhere else.

Abdomen.

Spleen just felt behind the costal margin, coming down on inspiration. Not tender.

October 31st. Shrewd looks much the same.

Cardiovascular system

Pulse regular 60/min.

Heart. Apears beat as before.

Mural area. The murmur is rushing and loud now. 2nd sound is soft and blunted at the finish suggesting an early diastolic. Propagated to axilla.

Mural area. The systolic murmur is replacing the 1st sound, loud, rasping. 2nd sounds loud +. At 3rd left interspace a faint hissing high pitched diastolic murmur is heard midway between 2nd sound and the beginning of the systolic murmur. Propagation as before to neck of left subclavian.
November 7th.

Stuart got up last night for the first time since admission, and though he felt fairly well, though weak, his temperature ran by 10 F to 98°, it looks much better this morning.

Cardiovascular System.

Pulse: Regular 102/min.

Heart: After beat unchanged.

Sounds: Ribs of extraneous heard.

Atrial area: Loud rushing "whistling" murmur replaces 1st sound. 2nd sound is a double a character, soft mumbled.

No diastole murmur heard. Propagation -> axilla.

Aortic area: Harsh systolic murmur replaces 1st sound. 2nd sound loud and sharp.

Pulmonary area: Systolic rasping murmur. Explosive 3rd sound.

Left 2-3rd Space - faint shifting and diastolic murmur.

Abdomen:

Spleen is slightly enlarged on percussion, no longer palpable.

Skin:

Two small red areas appeared yesterday on upper lateral aspect of Stuart’s R: forearm. Not tender, painful or itchy.

About size of sixpence. No raising of skin on circulation.

November 8th.

Stuart’s face is much better; he has lost his translucent body, his colour is much improved.

Cardiovascular System: unchanged from yesterday. Rath 80/min.

Progress: seen by:

1. Stuart’s general appearance:
   a. His colour has improved.
   b. Fair, looks fatter, eyes less sunken.
   c. More cheerful, lively, active.
   d. Patient feels better, eats better, headache gone.

2. Clinical Findings:
   a. Pulse temperature settled to normal.
   b. Spleen has returned - no longer palpable.
   c. Haemoglobin 52% -> 72%.
   d. Heart murmurs are changing less & less in character.
Treatment and progress.

6-10-51
R. Penicillin subcutaneous 600,000 units 2 i.d.
Temperature dropped from 103° to 99.6°. Pulse 120.
R. Luminar gr. 1 b.d.
R. Neomycin 2.5 6 hourly.

7-10-51
Temperature fell to 98° 97°. Pulse 115.
He is restless and anxious.
R. Chloral hydrate gr. 10 at 8 p.m.

8-10-51
Temperature not rising above 98.4°. Pulse 105.

Blood examination:
Hæmoglobin 52% 
B. S. R. 2 mm/hr.
R. B. C. count 4,200,000/m. W. B. C. 3,400 /cm.

9-10-51
Temp. below 98.6°. Pulse 90-100.
R. Neomycin stopped.

12-10-51
Slight improvement. Blood culture for culture.
T. 97-98°. Pulse 90-100.

13-10-51
Blood exam.
Hæmoglobin 64% 
B. S. R. 6 mm/hr.
W. B. C. 4,200 /cm.

16-10-51.
T. 97-98°. Pulse 100.
R. Ascorbic acid 50 mg b.d.

21-10-51.
Blood culture negative. + 97°. Pulse 95.

26-10-51.
Improvement continues.
R. 7 c. Farmos Supitet gr. 3 t.d.

29-10-51.
Blood exam.  
T. 97°. Pulse 90-100.
Hæmoglobin 72% 
B. S. R. 24 mm/hr.
W. B. C. 6,100 /cm.

2-11-51.
Sedatives stopped. T. 97°. Pulse 90-100.

4-11-51.
Penicillin stopped. T. 97° Pulse 90-95.

6-11-51.
Allowed up at 5 p.m.

Stuart improved very well, his temperature and pulse remained constant and after a graduated exercise regimen, checking his pulse and temperature was discharged home on 7-12-51. (Hb. 90% taken)
Diagnosis. Chronic Rheumatic Endocarditis

with recent Subacute Bacterial Endocarditis

Reasons for Diagnosis.

In Chronic Rheumatic Valvular Disease of the Heart:

(a) History of acute rheumatism when patient was 52 years of age. Recurrent attacks in 1947 and vague limb pains on and off since then.

(b) Clinical findings

Examination:

General. Thin, underdeveloped, delicate boy, heart heard.

Sounds are changing in character due to the vegetations of the subacute bacterial endocarditis but the following were usually heard:

Mitral area: (i) Systolic blowing murmur indicating flutter in incompetent blood regurgitating into a valve in systole.

(ii) loud early systolic and presystolic murmur (both heard) indicative of a slight degree of mitral stenosis; also accentuated second heart sound.

Aortic area: (i) loud loud systolic murmur propagated well up into the neck — possibly an aortic valve stenosis.

(iii) loud aortic diastolic murmur indicative of a slight degree of aortic incompetence.

Rales is of contracting type, hearing fourth and sixth heart sounds.

Heart is generally enlarged by palpation and percussion.

II. Subacute Bacterial Endocarditis.

a. History:

(i) Previous rheumatic history, chronic valvular disease.

(ii) General symptoms for 6 weeks before admission:

Fatigue and sweating at night — low grade pyrexia

Pallor, anaemia

Restlessness and malnutrition

Loss of weight.
(b) Clinical Findings:

(i) Pallor and anaemia. Haemoglobin 52%.
(ii) Temperature 100° - 102° on admission.
(iii) Changing heart sounds. Quality was different day by day.
(iv) Splenomegaly with no other cause.
(v) Embolic phenomena.

1. Myocardial anaemia base of brain replaced to give subarachnoid haemorrhage.
2. Petechiae in sores, conjunctiva, mouth.

(vi) Rising B.S.R. rate 2 - 4 - 24 mm/hr.
Rising R.B.C. count 3,000 - 6,100 / cu. mm.

The following were searched for and not found:
1. Blood cultures repeatedly negative.
2. Petechiae in nails, conjunctiva, mouth.
3. Clubbing of fingers.
4. Others nodos on fingers or toe-tips.
5. Red cells in urine.

Differential Diagnosis.

1. Of Chronic Rheumatic endocarditis.

Here is really no question of diagnosis here. Hi attack of scarlet fever followed by rheumatic fever, an acute attack, at the age of 5½ after which his parents were told that he had a damaged heart valve or valves is typical.

A further attack (in 1919) was to be expected.

The findings are quite inconsistent with anything but chronic rheumatic endocarditis but are rather confusing as the fever and quality of his manner are rare changes.

2. Of Subacute bacterial endocarditis.

1. Streak persisted as a case of meningitides with intense puru lous headache and vomiting.

On exam. Kernig's sign was +, there was marked rigidity and no other abnormal neurological signs except an
upgoing left toe - the Babinski on the left side. 

Here the following had to be considered:

1. Infections meningitis of meningococcal or tuberculous origin. The latter was especially possible as Stuart has a painful and chest 
biohazard - probably therapeutic.

This would have given identical signs and symptoms if had been excluded by the fact that the lungs were clear, he had no
changes in the chest or symptoms, and also the cerebrospinal fluid findings.

In tuberculous meningitis the fluid is clear, cells are
increased even up to 800, protein is raised to 400 mg/L sugar
is decreased and chlorides about normal. The tubercle
beads are usually obtained from the sphenoidal wall which forms

In meningococcal meningitis, was much more unlikely
but could be considered if higher temperature would have
been present, and CSF showed cloudy fluid cells 300-2,000
mostly polymorphs, protein++ sugar+ and chlorides decreased.

2. Infection from middle ear disease. No signs.

3. Subarachnoid haemorrhage from a ruptured aneurysm.
   It is a possibility and may in fact be the diagnosis but is
   unlikely as a

4. Subarachnoid haemorrhage from rupture of a cryptic
   aneurysm from an embolus hits into the general picture
   perfectly - the gradual onset of low grade pyrexia, marked
   loss of weight with anaemia and splenomegaly in a boy
   who has chronic varicose disease of the foot.

4.2 Prodromal Symptoms

Another possibility to be considered is

A. Severe nutritional anaemia for this can give a
    functional systolic murmur at the apex and base of the heart,
    reticuosis due to capillary damage, splenomegaly and low
    grade pyrexia: in severe pallidocyanic anaemia subarachnoid
haemorrhage may be got but this is very rare. However
the presence of the endocardial exudate and heart murmur
confirms the diagnosis of organic changes in the heart valves and
the abscess is thus secondary to an endocarditis.

6) Tuberculosis. This was excluded on examination
and investigation of embrosginal fluid.

Thus the history and clinical features pointed to an
infective endocarditis. 1926 is of 3 types.

I. Acute Rheumatic Endocarditis.

1. Has usually had a new acute phase, occurring during an
acute attack of rheumatism with joint pains etc. Heart had noise.
2. Heart rate is more rapid, pulse is leucocytosis: Heart had angina
3. B.S.R. is usually very fast. Streaks seen through modulus
4. Heart murmurs could correlate.
5. Embolic phenomena are rarely seen: vegetation are small

II. Ulcerate (Malignant) Endocarditis.

1. Usually seen as the course of an acute illness pneumonia, typhoid
2. Fever is hectic with rigor and drenching sweating
3. Emboli are septal and produce abscesses
4. Rapidly fatal

III. Subacute bacterial endocarditis.

Sant's story is typical and the findings also.
Blood cultures would not have been expected to be positive
when the specimen was taken after penicillin therapy.
The disease has not been of long enough standing to produce
fnger clubbing.

Red cells in the urine, a sign of embolization to the kidney
with focal embolic nephritis would have to be sought for
in a centrifugal specimen daily, as they are very transient.
Another described sign is the cafe au lait
skin. Sant's does not show this — only about 20% of long standing cases do show this phenomenon.
Commentary.

These five cases illustrate the course of an attack of acute rheumatic fever in a young man of 23 and four complications of rheumatic fever—chronic rheumatic endocarditis, two with super-added pathology—heart block, complete—in an old man of 77, and subacute bacterial endocarditis in a lad of 14.

It is proposed to discuss briefly rheumatic fever with reference particularly to James Turnbull containing two cases with past history of rheumatic fever in the other cases and secondly to discuss the complications of rheumatic disease as shown by the other four cases pointing out typical and atypical characteristics.

Summary of Cases.

   Duration: About 10 weeks.
   Result: Cure—As far as can be seen.

II. Alexander Stewart, 77. Complete heart block with rheumatic aortic stenosis.
   Duration: Heart block—3 months.
   Endocarditis—Probably 30 years.
   Result: Death.

III. Christina Luggis, 63. Rheumatic aortic stenosis with aneurysmal dilatation.
   Duration: Endocarditis—Probably 27 years.
   Aneurysm— Probably 1 year.
   Result: Improvement in condition.

   Duration: Probably 20 years.
   Result: Great improvement in condition.
V. Stuart Taylor. 14. Chronic rheumatic endocarditis
subacute bacterial endocarditis

Duration: chronic rheum. end. 8½ years
subacute bacterial end. 3 months.

Result: great improvement in condition

Case of S.A.B. E. as far as can be seen.

Rheumatic fever.
This is an abnormal tissue reaction in a sensitised person
to the toxic products of the haemolytic streptococcus
which is characterised by febrile and toxi attacks with
the presence of multiple disseminated focal inflammatory
lesions in many joints: these joint lesions are
typically fleeting in character and tend to disappear
on dosage with antipyretic drugs - of the salicylate group
Great tendency is to affect the heart.

Incidence. This is difficult to assess as in many cases
the condition may be manifested only as slight "growing
pain" which are not reported to the doctor, ever.

It is a common disease, however.

Pathology. As the disease is a manifestation of
allergy, as would be expected, the pathological lesions
are a proliferation of fibrous tissue. The typical lesion
is described as a granuloma and is best seen in the
myocardium as an "Aschoff body", an area of swollen
collagen fibres, is surrounded by a characteristic layer of
rheumatic endothelial cells which may probably arise
from the connective tissue cells. Lymphocytes and
plasma cells are present peripherally and eventually there
is a fibrous tissue reaction with fibroblastic proliferation
and a dense scar is formed.

Thus, for an area of peri-articular tissue and
there is often a resorption into the synovium of the affected joint.

Heart - pericarditis. (See opposite)
Aetiology.

It is not fully known, but certain factors must be considered:

1. Age incidence

This is usually maximal from 6 to 10 years.

James Turnbull is thus not typical case: he is very young old to develop rheumatic fever for the first time at 23, but of course, it can occur at any age.

It is not known how Mrs. Stewart was when he had his first attack:

Mrs. Ingle was again in a slightly older age group when she developed it at 17.

Mrs. Weir was also 13 at onset while Stuart Taylor was more typical in that he was 5½ when he had his first attack.

2. Sex incidence

It is said that boys are not more susceptible than girls.

3. Heredity

Many cases do show a family incidence: of these five:

James Turnbull’s sister died at the age of 13 with "rheumatic fever and chorea", and his paternal grandmother had the disease when she was young.

Alexander Stewart: no available details.

Christina Ingle: no history in family of rheumatic disease.

Jessie Weir: no history in family of rheumatic disease.

Stuart Taylor: father has rheumatic heart disease.

This would seem more than coincidence - if 2 cases have definite family history and no details in one case available.

Pathology in the heart.

The valvular (stenotic) lesion is a true inflammation; it is thought that the increased damage to a child’s valve is due to the relatively greater vascularity compared with an adult valve. The mitral valve is most commonly affected, then the aortic, rarely the pulmonary tricuspid. There is intense scarring of the valve: then small firm vegetations are formed on the valves and chordal tenderness and cicatrization follow inflammation with distortion and tightening of the valve. This may take many years to develop fully.
Rheumatic fever is said to be more prevalent in the poorer people with faulty nutrition, poor housing, overcrowding and dampness. It is very difficult to prove. There are many "hospital class patients" but their living conditions vary. Mrs. Heydty lives in a tenement, James Turnbull in a Corporation house; but Stewart when he got rheumatic fever was living in a good bungalow with no possible overcrowding.

Little information is available about diet but Mrs. Heydy and Stuart both say definitely that they had good all round diets in their childhood with plenty of milk and fruit and vegetables.

5. Infection: 

This plays a strong part in this. The patient has to be sensitised to the Streptococcus hemolytics (Group A) before allergic phenomena are developed as a reaction between antigen and antibody.

This may be obvious, as in Stewart Taylor's case when rheumatic fever followed closely on an attack of scarlet fever, or no evidence may be found, as in James Turnbull's case: he had had no scarlet fever or any other illness attributable to the hemolytic streptococci. In other cases no evidence exists.

Course of the Condition:

It is shown typically by James Turnbull's story. Severe antecedent pains, temperature becoming higher at night, rapid in recovery, and then swelling and painfulness in many joints, all being controlled with salicylates.

The systolic murmur heard in the cardiac area is often found during the acute illness, associated with a tachycardia and may only be due to dilatation of the
of valve rings. This alone can show whether there has been real pathological damage to the heart.

It is unusual for severe damage to occur during an attack; usually this occurs some weeks after the attack and steadily grows worse.

In these cases: Various thrombi must be watched to see if cardiac signs develop: he was rigid and symptom-free at time of discharge.

Alexander Stewart was said to have suffered from rheumatic heart disease for many years. Probably 5 to 10 years at least would elapse before it started to be even slightly breathless on exertion, and then over the years this shortness of breath would increase until it was so short of breath that his heart started to fail as it could not pump enough blood through his narrowed valve, the lungs being greatly congested.

Mrs. Hope was many years in developing signs and symptoms of cardiac embarrassment.

Mrs. Wright on the other hand was prevented from taking severe exercise from the start of her rheumatic fever attack. Although she managed to do a good day's work later on she was always breathless on climbing stairs so really annoying and in fact hardly considered that abnormal.

Stewart's heart was well compensated after his acute rheumatic fever episode as he played cricket and football but when he did overdo it he got an infarction on his damaged valves after working and playing hard all day when on holiday.

**Diagnosis and differential diagnosis**

Have been dealt with in each case after description of history and examination.
Treatment:

There is now some doubt as to the benefit or otherwise from prolonged absolute bed rest in rheumatic fever and most people advocate it as the safest form of treatment. There are several points which are carried out in every case.

1. Bed rest until symptoms and signs gone.
2. Control of symptoms by adequate doses of sedatives and non-steroidal anti-inflammatory drugs, and examination of necessary; splints fixed to affected joints.
3. Careful watch of pulse rate and temperature; the pulse rate only being allowed up when these have been steady at a normal level for some time.
4. Building up of general health and nutrition by
   a. Fresh air
   b. Good diet with plenty of calories, protein, fruit and green vegetables.
   c. Administration of iron and of calcium.
   d. Supplementary Ascorbic acid if necessary.
5. Graduated slow convalescence, watching pulse rate.
6. Careful thorough treatment of any complications or concurrent diseases or infections.


   Cough, colds: Penicillin

   Sulfasalazine.

7. Removal of local sepsis after the disease is over, e.g. bed sores, boils.

If these were carried out in every case then few would be prone to complications.

Unfortunately the ideal life of sunshine, fresh air, exercise and good food that is so necessary for these patients is not possible except for a short period of convalescence.
In James Turnbull's case these measures were carried out and assessment of progress made by pulse and temperature charts and repeated blood sedimentation rates and haemoglobin estimations.

He required

Rx. Sodium salicylate q 30
Sodium bicarbonate q 30 f 3 hourly

before his symptoms abated.

He also was given

Rx. Penicillinum q 2 did be established.

As he was worsened about his condition.

The salicylates were stopped only 2 weeks after symptoms had disappeared and pulse and temperature were steadily normal.

In Stuart Taylor's case, had he had his carcinoma
been removed when trouble began in June it is quite probable
he would never have developed infective endocarditis
as they were a potent source of bacteria. The extraction
would have been perfectly safe under penicillin cover.

In Mrs Wegg's case, she died with her teeth
extracted under penicillin cover and did very well.

Prophecy of Complications.

Unfortunately nothing so far can be done to
prevent a boy like James Turnbull from developing
chronic endocarditis. It can only be recommended
that he live as healthily as is reasonably
possible: persuaded him to take plenty of exercise
without ever overtiring himself and to eat a good diet
and avoid too many late nights, heavy smoking or
drinking.

Of course the argument is that it is not
wanted to make a cardiac cripple out of a young
man: Should he be told he might develop trouble?
or should we remain unaware of the facts that we may well lie in wait for him that he can hardly avoid anyway?

It is a difficult problem and probably depends on the mentality of the patient.

It may be sufficient in some cases for the doctor to keep a careful eye on such a patient and to treat thoroughly any infection that may arise.

Chemotherapy

It has been suggested that prophylactic chemotherapy e.g. by Salphenazin 1-2 g daily from September to June every year (the danger period) is an answer.

But against this come

1. Toxic effects of prolonged dosage: these are not fully known.

2. Organisms may well develop resistance to the drug.

The question of chemotherapy in these cases is not still within the realm of experiment: it may yet be necessary.

Toxidermy

This should always be done if it could lead to the cure of the patient were not a rheumatic, but always had always under cover of pemphigus to prevent a bacteriemia.

Prognosis for James Turnbull

He may make a complete recovery without any permanent cardiac damage and lead a perfectly normal life.

Or he may easily get another attack of rheumatic fever with contact with streptococcal infection.

He may develop late chronic rheumatic valvular disease of the heart, symptoms and signs of which may not develop before he is an old man.

The prognosis is always guarded: for the acute
attack, it is good - as has been already proven by his apparent total cure.

If he never develops streptococcal infection again, his prognosis is very good.

If he does, or has a 2nd attack of rheumatic fever, he is very liable to develop chronic heart disease.
85% of patients who get rheumatic fever will develop chronic valvar disease of the heart in later years.

**Rheumatic Heart Disease.**

This may be an integral part of rheumatic fever and can therefore not be called a complication, but symptoms suggest chronic rheumatic valvar disease of the heart may develop as seen above, many years after the initial attack.

Shift divides rheumatic heart diseases into 4 types:

I. Endamitting type, usually fatal with various rheumatic fevers, rare in older children.
   - Remittance active
   - periods of remission flaring up at intervals

II. Chronic active - gradually progressing - most cases here.

III. Chronic inactive - years with no signs or symptoms.

**Incidence:**
- In adult population it accounts for 30% of heart disease.
- In children, the % is higher - 90% of defective hearts are caused by rheumatism.
- It is therefore a most important subject.

The other few cases will now be discussed, the salient features emphasised and its typical or atypical features stressed, and a prognosis given. This is of course very difficult in most cases.
Case II. Mr. Stewart: Complete Atrioventricular Heart Block
in a Rheumatic Aortic Stenosis.

His previous history is a little vague and it can only be assumed that he has had some symptoms of rheumatic heart disease in recent years only as he had an active career and is now 77 years old.

In Mr. Stewart's case the valve mainly affected was the aortic one and only in the last few years that he has had symptoms has his exercise tolerance been reduced.

Symptoms and Signs.

The first symptom is breathlessness which is accounted for by the heart being unable to discharge through the narrowed aortic valve all the contained blood in its ventricle with the result that there is congestion and clumping back of blood in the lungs which causes breathlessness.

Last October 51 he most have been decompensated as his breathlessness increased and he got swelling of his ankles. The heart was failing after many years of pushing blood through a narrowed semi with no complaints on signs of embarrassment.

Red post became essential.

Three weeks before admission it is likely that the conducting mechanism of his heart gave out; this is very likely to occur with damage to the valves.

A heart block was obtained and he became very breathless; he gradually became mentally confused, a sign of cerebral anoxia and was in a very poor state on admission, in gross cardiac failure, his ventricles beating at 30/min.

Explanation of Signs.

The irregularity of the pulse and heart beat is caused by the
regular relationship of auricular and ventricular contraction: should the auricles beat just before
the ventricles, then the blood flow into the ventricles
will be maximal and a strong contraction will be
obtained (Baroreflex effect: increased contraction
with increased filling). Similarly if the auricles are
observed, then will be poor filling before ventricular
contraction and a weak beat will be heard.

The combined signs of cardiac (congestive)
failure are explained by increased venous pressure owing
to defective function of the right ventricle. The venous veins
become congested, the liver enlarges and oedema
eventually develops in dependent areas due to
1. Increased venous pressure — sparsity of blood
in peripheral vessels — increased tendency to leak out plasma.
2. Tissue anaemic damage (capillaries).

The heart sounds were too weak and faint to be able
to be examined and the sounds analysed but it is
probable that a rough systolic murmur in the aortic area
would have been heard. Unfortunately there is no clue as to
this from the doctor.

The electrocardiogram requires some comment: the lengthening
of the QRS interval complex is necessary when such a slow
rate is present, to maintain circulation.

The leads are put round the heart to detect areas
of maximal deflection over a hypertrophied ventricle and
here the V6 lead together with large deviation on
the standard leads points to left ventricular hypertrophy.

The most interesting feature of Mr. Stewart's
case is the fact that he lived to be 77. This
is known when rheumatic disease of the aortic valve is
compatible with a long life and he is a good proof.
Prognosis.

Had a prognosis been written down on Mr. Stewart's entry to hospital it should probably have been accurate for it is an accepted fact that while aortic stenosis may be compatible with a long life, once cardiac failure sets in the prognosis is very bad. Actually it is unlikely that the patient will live months— even with excellent treatment. This was fully borne out in Mr. Stewart's case as really nothing could be done to help him and his death was inevitable.

Case III  Mrs. Inglese, 62 Aortic stenosis with auricular fibrillation.

This is another example of the longevity associated with rheumatic aortic stenosis. This patient had no complaint until only 5 years ago when she began to get the classical symptoms of left ventricular embarrassment—paroxysmal nocturnal dyspnoea.

Diagnosis in this case could be made on the history above— the negative symptoms of hypertension helping, but the signs in the heart are also classical and diagnostic of the condition.

The reason for a heart to have compensated for a defective valve and then breaking down is not known. She may have been unusually hard-worked 5 years ago when she first became symptomated conscious but often no reason can be found.

Her cardiac failure has been slow slow in its onset, retirement to bed only being necessary one month before admission although she had noticed ankle swelling 9 months before.

The onset of auricular fibrillation probably accounted
for the determination of her condition 1 month ago; or it might have come on 9 months before admission; there is no evidence from the history of a sudden collapse and fainting, dizzy spells, palpitation etc.

Signs in the heart:
The rough aortic systolic thrill and murmurs are typical. One point is that it is possible that there might have been a slight arterial stenosis and that the obstruction mitral murmur was caused to disappear by the phlebitis of the aorta; this, however, is not likely.

The cardiac failure signs have been discussed.
The electrocardiogram is very similar to Mr. Stewart's as would be expected, and it confirms the diagnosis of auricular fibrillation.

Treatment:
Is of cardiac failure:
Digoxin 0.25 mg q.i.d. to improve heart function
Aminophylline 26-5 i.d. to reduce bronchospasm
Mersalyl 2 cc. three weekly

The digits did show her heart sank and made her more compensated; but failure to remove the actual essence in spite of good response to mercurials suggested that it was not entirely cardiovascular derangement.

Quinidin was contraindicated here as a method of curing fibrillation for 3 reasons:
1. Toxicity to a damaged myocardium—here the cardiac failure.
2. Long standing auricular fibrillation—the danger of a clot being liberated from the auricles.
3. Perhaps fatal effect.

The most unusual feature of this case is the co-existence of auricular fibrillation with aortic stenosis, as
it is much more common for a mitral stenosis to be complicated by auricular fibrillation: it is really rather rare in aortic stenosis.

**Prognosis.**

This tends as in Case II II to be bad. But Mrs. Ingham made quite a good improvement — rather surprisingly, and very happily — and she could walk quite a distance before becoming breathless on discharge. In spite of this apparent recovery of the heart however the prognosis is not good and it is very unlikely that Mrs. Ingham will live many months: however she may be lucky and live for some time to come.

Death may occur from cardiac failure or embolism (rare) from the pulmonary artery which is usually full of thrombus. Similarly a hemiplegia may occur from the left auricle.

**Case IV. Mrs. Weydt. Mitral stenosis.**

This is a very different case: Mrs. Weydt is a young woman of 33 and the outlook is different.

**Course of the Condition.**

It is quite usual for a young woman with what may be called 'subclinical mitral stenosis' to go through pregnancy with no symptoms except some breathlessness in the latter months as Mrs. Weydt did: She was younger at the time (22) and her heart managed to compensate.

Only in 1947 did she first have serious trouble when she had her 2nd haemoptysis. At that time when she
was discharged from the hospital in Amsterdam she was on a maintenance dose of Digitalis.

The recurring attacks of haemoptysis are a sort of safety valve in mitral stenosis: thus, Weydt said that she felt much better after a haemoptysis had occurred and she had recovered from it.

Miss Weydt has had a most unlucky history and she is amazingly cheerful, enjoys her work and although aware of the dangers of her condition is very hopeful.

This attack of haemoptysis quickly settled down, and her breathlessness disappeared, when digitalised and statified.

The discovery of the posterior Warthin reaction was a great shock to her and to anyone else a complication on the picture.

Her treatment with penicillin for this unfortunately precipitated a Forster's reaction: this is harmless but not dangerous and she quickly recovered.

Removal of her teeth was a very necessary part of treatment for although it is unusual for a person of this age to develop subacute bacterial endocarditis it is not impossible. There was no trouble about the extraction except that her gums took some time to heal with blood on the spaces but they did heal and she is looking forward to getting her false teeth in about 1 year's time.

The bismuth therapy for her syphilis was now to go ahead: it is dangerous to give bismuth while there are raw areas in the gums because of violent toxic local effects.

Signs:

Are typical of a mitral stenosis. The aortic murmur was listened to carefully to see if there was any
possibility of a syphilitic aortitis infection complicating the picture but there was no sign of a obstructed aortic valve on discharge.

It is interesting to note how the C.S.F. W.P. was ++ on 14.11.51 and ++ on 7.12.51. This (it is to be hoped) is accounted for by the disappearance of the Hackenhoff reaction.

It would be interesting to have an electrocardiogram of Mrs. Heyoldt's heart to see how much of right auricular dilatation as this can be enormous and can be seen clearly on a ray also.

Prognosis.

Mrs. Heyoldt should do well: although she breathes on severe exercise on discharge, if she takes reasonable care she will improve and probably be able to return to some lighter work by the summer months. It would be very unadvisable for her to go back to the (same hotel) as a waitress; she should look for a sedentary job somewhere.

The expectation of life is not very good, however. It is unlikely that she will live much beyond 40 years. She will tend to develop auricular fibrillation and congestive failure at any time and the prognosis is therefore guarded.
Case V. Stuart Taylor.

Subacute bacterial endocarditis
on chronic rheumatic endocarditis.

The most interesting feature in this case is the difficulty in diagnosis: this has been already discussed.

Course of Stuart's condition.

Although his parents were told that his heart was damaged after the 1st attack of rheumatic fever, he was able to go to school and later play Rugby football and cricket etc. with the other boys.

The teeth decaying were very important as in Sept. 1951 when he was admitted. He must have had a bacteraemia and the organisms settled in his cardiac valves, which already damaged were an ideal pace.

This chronic infection caused a depression of red cell formation in the bone marrow and in became anaemic, tired, weak and anorectic, losing weight.

At about this time an embolus must have come off one of the valves and been carried up into probably the cerebral artery where it would lodge in a point between 2 vessels: this infected embolus caused the vessel wall to become thin and damaged and it started to leak - the start of Stuart's headaches.

He probably had 1 or 2 more emboli lodged in his spleen or kidney which caused no symptoms.

He awoke one day with severe headache, without loss of consciousness due to a fairly severe subarachnoid haemorrhage from rupture of an aneurysm.

As he was at the time sitting reading, it must have been an inferior haemorrhage causing meningism and so a wrong diagnosis was made of subarachnoid meningitis, considering his contact that he had in his patient.
Explanations of Signs

Splenomegaly

1. Cardiac congestion
2. Left atrium from embolus
3. Chronic infection

Cardiac failure was noted by auscultation or x-ray which caused sudden deterioration in condition:

Temperature rose to 104°
Tachycardia remained.
Hemoglobin fell 72% - 52% in 3 weeks.

Under treatment by penicillin the bacterial infection was gradually controlled, and pulse and temp were again normal.

Changing heart sounds:
Due to vegetation on valve, causing stream flow - fibrinoid, cells, bacteria and leucocytes form a vegetation.

Subacute bacterial endocarditis

3 factors must be present.

1. Damaged heart valve
2. Infection in the body
3. Decreased resistance of the patient.

We have seen how these all came about.

Occurrence. It is commoner in late teens and early twenties. Accounts for 3% of all organic heart disease.

In damage may be:

1. Rheumatic
2. Congenital
3. Syphilitic
4. Arteriosclerosis

In a young person 1) or 2) are likely.

Infection may from tonsils or teeth as in this case.
In any source of chronic infection - bowel, kidney, genital tract.
Organisms.
Acute endocarditis.
1. haemolytic streptococci
2. pneumococci
3. gonococci.
4. Streptococci viridans.
Subacute endocarditis
1. usually Streptococci viridans.
2. or any other organism.

Pathology.
Bacteria invade the damaged heart valve(s) and small thrombi form; there is little local reaction or repair.
Site of the vegetations: On the lips of the valves or proximal attachments and spreading onto
the left auricle endocardium.

Course of the disease.
Stuart presents a typical story.
Certain typical features are interesting:
1. lack of renal signs or symptoms
   - often one gets 1) silent infection
     - often with haematuria
   2) septicemic nephritis,
2. If the endocarditis is acute, abscesses are formed.
3. The lack of Osler's nodes is unusual.
4. No true petechiae were seen or raised haemorrhages
   - another unusual feature.

However, the rest of Stuart's story tells planning of an
undoubted susceptibility to a rheumatic infection and
always reaction in most of his tissues which unfortunately
leads to permanent heart damage. He then developed
one of the common complications of rheumatic heart
disease i.e. SANE from the 1st acute phase
of which he has recovered very well.
**Diagnosis**

Immediate: Is very good. Stuart is much better and looks well.

Bad: Is very poor. It is likely that his life will be cut very short due to:

a) recurrence of S.A.B.C.
b) cardiac failure

But the prognosis is often very difficult and one must just hope that it will be better in this boy's case.