Sir,

I am greatly indebted to Professor Ritchie for allowing me to use his cases in wards 23 and 24 at the Royal Infirmary, Edinburgh for the purpose of competing in the Wightman Prize in Clinical Medicine. I have also to acknowledge the courtesies extended to me by Dr. McKinnon the house physician in allowing me free access to the various reports and X-rays on the cases I selected.

The cases are,

1) A case of syphilitic aortitis illustrating various cardiac symptoms.

2) A case of mitral stenosis with fibrillation to illustrate the treatment of cardiac failure, etc.

3) A case of congenital heart disease.

4) A case of arteriosclerosis to show how the condition may be classified into stages and a discussion on its treatment.

5) A case of chronic kidney disease with special reference to the condition of uraemia.

I am, Sir,

Yours faithfully,
JAMES C. JAMIESON. Aet. 42. Civil Servant.
Admitted. 3rd. March 1931.

Referred from Dr. Jamieson, Albany St.

Complaint. Breathlessness on exertion, about eight weeks duration.
'Attacks' at night, A fortnight's duration.
Swelling of the feet.

History of the Present Illness.

About a month ago the patient noticed that he was becoming somewhat breathless. This breathlessness came on after he had walked some distance and at times became so severe that he had to stop and rest for some minutes before he was able to proceed again.

At this time also he became aware of a 'tight' feeling over the lower part of his chest. It was never so severe that he had to lie down or try and obtain relief in any other way. Nevertheless he was conscious of its presence all the time, and also that it tended to become a little worse when he became breathless.

Finding that his work at the office was becoming a little too much for him he obtained leave for a fortnight. Most of this time he spent in bed or quietly sitting in the house. After this rest he returned to work for a day or two but again had stop because of a disturbing new symptom which had appeared. He is not very clear as to the exact date this appeared but it is apparently about a fortnight before the date of his admission.

On this evening he went to bed feeling moderately well but later awoke with an intense feeling of suffocation, he gasped avidly for breath, felt extremely distressed and frightened and got out of bed. In trying to get a comfortable position he soon found that leaning over the back of a chair was the most comfortable one and in this position he stayed till the suffocating feeling passed off in about half an hour. At this time he started to cough up an abundant frothy sputum which he states
contained no blood but was full of 'tiny black specks'. After an hour or so of this paroxysmal coughing he wiped off the clammy sweat which had collected on his brow and thankfully crawled back into bed. These attacks were repeated on two or three occasions till finally he states that he was afraid to go to bed. On Sunday the first of March the patient retired to bed at 10.45 p.m. About half an hour later he awoke in a typical attack, he immediately got out of bed, leant over two chairs and got his back well arched and hopefully remained in that position. The relief he expected however was long in coming and the attack did not entirely pass off till about two o'clock the following afternoon. The was the first occasion that he attack had lasted on till the daytime. He sent for his medical adviser who examined him and pointed out that his ankles were swollen. He advised him to seek expert advice and sent him up to the R.I.E. on the morning of March the third. Patient noticed that in the middle of an attack that the pain or rather the discomfort on the lower part of his chest tended to become worse. Also he changed his position from time to time in an attack and never liked to keep perfectly still. 

Previous Health.

In childhood he had measles, whooping cough, mumps, but never rheumatic fever, growing pains or St. Vitus dance. Twenty-six years ago he had a sarcoma of his upper jaw removed by Prof. Caird. Eighteen month later he had a recurrent growth removed but since then apart from the disfigurement and the difficulty in eating has had no further trouble in this region. He was accepted as fit during the war and served for three years with the troops in Dardenelles, Palestine and Egypt. During his service he
contracted dysentery and was in military hospital for three weeks with it. Later he also contracted malaria and this fever has been with him off and on up till about two years ago when he had his last attack.

**Personal History.**

Enjoys his work in a government office.

Used to smoke thirty cigarettes a day but has recently cut this down to ten. Says that he is a moderate drinker but rarely touches spirits. Patient doesn't confess to ever having had venereal disease.

**Family History.**

Patient is a bachelor.

His father died aged 66. His mother died of dropsy at the age of 34.

**EXAMINATION.**

Patient is a somewhat fat man of 41 years. He looks fairly well but is pale. The lobes of his ears and his lips are slightly cyanosed.

**Alimentary system.**

Tongue is clean and moist.

All the teeth in his upper jaw are false and he has to wear a special diaphragm in this region to close the opening in his hard palate made when he had the maxillary tumour removed. He has three unhealthy teeth in his lower jaw.

Owing to the deformity the patient has to choose his food carefully and his diet contains, perforce, much soft and well cooked food. He never suffers from indigestion. His bowels act quite regularly.

His abdomen is fat and moves but feebly on respiration. There is a pulsation in his epigastrium synchronous with ventricular systole. On palpation nothing abnormal could be found. His liver was of normal size. There was no tenderness over McBurney's point. The spleen was not enlarged. His sigmoid colon could be palpated with ease.
Genito-urinary system.
He passes a normal amount of urine. This contains no abnormal constituent and on centrifuging no casts or any abnormality could be found. The patient has never any trouble with his water and does not have to rise at night to pass any.

Nervous System.
The pupils responded well to light and to accommodation. On ophthalmoscopy the retinae were quite normal, there was no undue tortuosity of the vessels, no haemorrhages etc. The pupils were equal in size and shape, there was no evidence of any interstitial inflammation.

Movements of all limbs were normal in degree and strength. All the reflex 'jerks' were present and the Babinski sign negative. There was no examination on the Cerebro-spinal fluid carried out.

Respiratory System.
The lung expansion was equal on both sides. Bronchial fremitus was normal. The shape of the chest was not altered in any way. On percussion it was found that although resonance was mainly normal there was a definite hyper-resonance over the apices and the free borders of the lungs.

On auscultation a few sibilant rhonchi could be heard and also a few fine moist sounds over both bases. Vocal resonance was normal.

Cardio-vascular system.
Inspection. There was a slight suprasternal pulsation. The apex beat was not visible. The praecordium was normal in shape.

Palpation. The apex beat was located in the fifth left interspace in the nipple line. There was nothing distinctive about the apex beat. There were no thrills palpable.
Percussion. The upper border of the heart lay behind the third left rib. The left border of the heart was in the left nipple line. The right border was not enlarged.

Auscultation. In the mitral area both sounds were pure and closed. They were however markedly faint and at times were difficult to hear. In the aortic area the sounds were also closed and pure. In the pulmonary area the second sound was somewhat accentuated and reduplicated. This then was the characteristic state of affairs, but sometimes a very marked change occurred. This change was best heard best medial to the apex beat. In this change a third sound appeared to be added. It was heard just before the first sound and imparted a very marked gallop rhythm to the heart sounds. At first I thought that this was merely the reduplicated second sound but after further consideration I decided that in view of the fact that it occurred rather later than one would expect the reduplicated second sound to occur and that its point of maximum intensity was not over the pulmonary area but at a point somewhat medial to the apex beat that it was a true 'bruit de gallop'. In character also it was not sharp and short as in a reduplicated second sound but rather soft and dull. At one period of the patient's stay in hospital I heard this rhythm maintained for three consecutive days. Although I did not see the patient on his admission to hospital, in fact not till about three weeks after, I have it on the authority of the resident physician, Dr. McKinnon, that he could hear a friction rub over the base of the heart for two or three days. He also states that there was a systolic murmur over the mitral area at this time also. This systolic murmur is certainly not present now.

Serum Test. The bacteriology department reports a +++ Wassermann reaction.
The Pulse. The rate was about seventy beats a minute. The rhythm was entirely regular. The upstroke was rapid, the pulse was only moderately sustained and the downstroke was also fairly rapid. The blood pressure varied from time to time but was usually around

State of the Arteries.

Neither the radial nor the brachial were unduly palpable. The arteria dorsalis was not thickened.

Progress while in hospital.

From the date of his admission until the 14th April, i.e. about five weeks after admission he remained fairly well. He could read and converse with his neighbours in the ward without exhausting himself. The ward sister states that his appetite could not be better. On this day however He had one his paroxysmal attacks and I was fortunate enough to be present when it occurred.

I was assisting to change the patient from one bed to another when I noticed that he had suddenly become pale and cyanosed. He started to gasp for breath and very soon vomited up the light meal he had eaten just before. He leant over the side of the bed and in about a quarter of an hour started to cough up an abundant sputum. This sputum was of a slightly pink colour. After about an hour the attack had entirely passed over leaving him in an exhausted and gravely disturbed condition. I took the patient's blood pressure during an attack and found it to be considerably raised viz. . His pulse was quickened also. Many moist sounds could be heard over both bases.

After this attack he remained perfectly well until a month had passed. Then on two successive days he had similar attacks. Unfortunately I was not present but they were apparently severe enough to seriously alarm the ward sister who administered oxygen etc. At this stage then the pat-
ient is tending to become worse instead of better. He is in alarmed condition as he does not know when to expect another attack.

Phthisis, finds that he is becoming increasingly breathless on exertion. One night he goes to bed and falls into a deep sleep but awakes shortly with a most intense dyspnoea. He gets out of bed and after struggling for breath and, he thinks, for his life, the attack passes off and he coughs up an abundant frothy expectoration.

This in a nut-shell is the history of our patient. We must first decide where the cause of the dyspnoea is situated.

1) Bronchial asthma. We knew that true bronchial asthma may be associated with bronchitis and may only come on in adult life. The patient's history and a careful examination of the lungs convince us that the cause of his paroxysmal dyspnoea is not bronchial asthma.

2) Similarly the history and the examination exclude pulmonary tuberculosis as the cause of the dyspnoea.

3) Uraemic dyspnoea, the so-called renal asthma, is excluded by a careful examination of the urine and the urea output etc.

4) We are thus left with the heart as a cause of the dyspnoea, and actually in his history we have a very good description of a typical attack of cardiac asthma. This term is not a very good one as it inevitably leads to confusion from time to time with bronchial asthma. Possibly the most graphic account of a severe attack of cardiac asthma will be found in Allbutt's book on the diseases of arteries. Pratt in the Journal of the American Medical Association also gives a good description. Probably the most impressive fact is that the patient will go to bed feeling very well, pleased that he has managed to do more on that particular day than he has for some time past. Actually it is this that is the cause of his intense dyspnoea.
Commentary.

1) **DIAGNOSIS.** A middle-aged man, who suffers from syphilis, finds that he is becoming increasingly breathless on exertion. One night he goes to bed and falls into a deep sleep but awakes shortly with a most intense dyspnoea. He gets out of bed and after struggling for breath and, he thinks, for his life, the attack passes off and he coughs up an abundant frothy expectoration!

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Causation of attacks of cardiac asthma.

The patient can maintain his supply to his respiratory centre by increasing his pulmonary ventilation during the day. He may or may not be aware of this breathlessness. One day the patient does a little extra work than he is used to doing and goes to bed quite pleased with himself. He falls into a deep sleep and this depresses the sensitivity of his respiratory centre to its carbon dioxide stimulus. Thus the increased pulmonary ventilation which is occurring during the day ceases. The anoxaemia which has now been established progresses to a marked degree before the patient is awakened. As soon as he does he feels suffocated and by forced breathing he tries to increase his pulmonary ventilation. This fact coupled with the fact that there is a reflex vaso-constriction puts up the blood pressure. Thus the heart which in the majority of cases is a diseased one has to contend with an increased amount of blood passing to it and also an increased peripheral resistance.

Acute pulmonary oedema may be described here as it is nearly always associated with cardiac asthma. It is due to a failure of the left ventricle to pass on the blood from the pulmonary circulation with resultant stasis of the pulmonary circulation and extreme congestion of the alveolar capillaries. An abundant exudate into the alveoli occurs and reduces the available respiratory surface considerably. At this stage one can hear, as was easily done in Jamieson's case, numerous moist sounds. Unless the condition causing the pulmonary congestion rapidly improves the patient may die. The late Prof. Lorrain-Smith used to say that the patient drowned himself in his own transudate. What impresses one most about acute pulmonary oedema is its fleeting character. It often starts and ends within a couple of hours, and the patient is soon better. Jamieson was reading his newspaper with interest four hours after a bad attack!
Predisposing factors of cardiac asthma. The inception of abnormal rhythms of the heart are commonly causes of this condition. Fibrillation, flutter and other forms of cardiac paroxysmal tachycardia are all said to be causes. In conditions where the left ventricle is hypertrophied and is commencing to fail are also fruitful causes. Thus in chronic interstitial nephritis, hyperpiesis, aortic valvular disease, etc. are all possible causes of cardiac asthma. A careful consideration of the results of the physical examination excludes all these causes. The left ventricle was hardly enlarged, there were no heart 'murmurs', the blood pressure was not high, and the electrocardiographic results all helped to exclude these causes. We are left with two very important causes of cardiac asthma namely syphilitic aortitis, possibly with involvement of the mouths of the coronaries, and cardiac infarction. We are not left with these two causes by a process of exclusion but by positive evidence which will be discussed below.

Syphilitic aortitis. In reading the pathology of syphilitic aortitis I have found that Carey Coombs article in last August's Lancets and Boyd in his Pathology of internal Medicine to be very clear and helpful. In the generalised stage of syphilis the aorta is together with all other tissues of the body affected with spirochate. It is stated that it is at this stage that the disease that the aorta and the myocardium are first attacked. The virus has a predilection for small arteries and it is along the perivascular lymphatics of the vaso-vasorum of the aorta that it is first to be seen. There is a small round cell and plasma cell infiltration of the lymphatics which extends into the media. There it destroys the muscular
and the elastic tissue. Over these areas the intima proliferates in an attempt to buttress the weakened media. This process may start as low as the abdominal aorta but usually it is the arch and the ascending parts that are most affected. Gradually creeping up the virus reaches that part of the aorta where the coronary arteries are given off. Here it strangles the mouths of these arteries although it does not affect the artery itself. Spreading to the valves of the aorta it thickens their free margin and widens the commissure. The replacement of the elastic tissue of the media by unyielding fibrous tissue is the main factor in the loss of the vessel's elasticity and thus leads to circulatory inefficiency. The strangulation of the coronary arteries leads to a poor oxygen supply to a left ventricle which is already struggling with the backflow of blood from incompetent aortic valves. As Coombs remarks surely a malign agency wanting to bring the action of the heart to a standstill could not light on any subtler way than this.

Cowan states that the symptoms of cardio-vascular syphilis come on about seventeen years after infection but may be much shorter or longer. With regard to the myocardium, Warthin has described a diffuse myocarditis in many cases of cardio-vascular syphilis. Coombs has found that it is a very rare lesion and even in the most suspicious lesions when blocks were sectioned and examined by Levaditi himself the spirochate was never found. The main symptoms of syphilitic aortitis apart from a valvular lesion are pain, dyspnoea - especially attacks of cardiac asthma, and more uncommonly arrhythmias. J.C. Bramwell states that 25% of cases of heart block are either directly or indirectly due to syphilis.

**Thrombosis of the Coronary arteries and cardiac infarction.**

The cause of cardiac infarction are as follows. An embolus from an endo-
carditis, syphilitic obstruction of the mouths of the coronary arteries and atheromatous changes in the wall of the coronary artery itself.
The infarct varies in size and may or may not reach the pericardium. If it does a pericarditis results, if it reaches the endocardium a thrombus is formed on it.
The artery most commonly affected is the descending branch of the left coronary artery. This involves the lower and anterior part of the left ventricle and also the septum. Thrombosis of the right coronary artery often involves main branches of the bundle of His.
A point to be remembered is that although in the normal heart these are true end arteries, in the heart that has diseased coronaries there is a tendency for new collateral channels to be opened up i.e. there may be communication between the right and the left coronaries.
The clinical features of the syndrome are as follows,
The patient is a man above forty, possibly with a syphilitic history, he is attacked suddenly, commonly in bed at night. Immediately the infarction occurs three things may happen. Firstly he may die almost at once with fibrillating ventricles. (I believe that this has actually been recorded electrocardiographically) Secondly he may be attacked by a very severe pain. This is possibly the worst pain humans are called on to suffer. It is situated at the lower end of the sternum, it may be associated with nausea and vomiting. It is also accompanied with true shock, the face is of ashy hue, the pulse rapid and the blood pressure low. Thirdly there are cases where the main symptom is a sudden and intense dyspnoea associated with little or no pain. This to all intents and purposes is an attack of cardiac dyspnoea.
An examination of the heart after infarction will show a slightly dilated ventricle, possibly gallop rhythm, pericardial friction if the infarct
reaches the pericardium, frequently fever and a leucocytosis. There is usually no change in the rhythm of the heart. Such, then, in brief are the two pathological processes which might account for the symptoms observed in our patient. It will be noticed that it is quite possible for the symptoms of specific aortitis to merge into those of infarction. Further as Professor Ritchie has pointed out the clinical features of the end result of infarction may be produced by an ischaemic fibrosis of the myocardium consequent upon syphilitic sclerosis of the mouths of the coronary arteries. I think that in the case of James Jamieson there is no doubt that whatever is the pathological state of his heart it can be taken that it is due to syphilis. His Wassermann reaction is a triple positive one and in the light of his symptoms is most highly suggestive. To attempt to differentiate between the aortitis and the actual infarction is important both as regards prognosis and treatment. Let us consider a few points, Firstly. His attacks came on suddenly about a fortnight before his admission to hospital. They were repeated both before and after admission. These successive attacks do not necessarily mean an aortitis and not an infarction as it is quite often found that infarction is not always the dramatic state that the text-books describe and several 'attacks' may occur. Such a case is of course more often diagnosed on the electrocardiogram than on the clinical features. Secondly. On admission there was no temperature or leucocytosis that we are told usually occur in infarction. But again this is no proof that infarction has not occurred. A consideration of all facts of the case convince us that there is definitely a syphilitic aortitis involving the mouths of the coronary arteries.
and that although there is no evidence clinically that infarction has occurred there is some evidence that a process of slow or rather sub-acute ischaemia of the heart. Thus we have evidence that there was at some time a fibrinous pericarditis which is in favour of infarction, secondly that the heart is becoming progressively weaker as is shown by the development of canter rhythm (a common occurrence in infarction) and also that since admission the patient's heart has become progressively quicker in its rate. A further point to note is that cardiac asthma and gallop rhythm sometimes occur when heart-block is being established, but at present there is no evidence that this is occurring in Jamieson's heart.

Finally let us turn to the electrocardiogram which was taken four days after his admission to hospital and see if there is any indication given there as to the state of the heart.

The impulses start in the sino-auricular node and no cardiac arrhythmia is present. There are no extra-systoles. As regards the time of conduction the P-R interval is three twenty-fifths of a second, the Q-S interval is two twenty-fifths of a second and the R-T interval is five twenty-fifths of a second. Thus it is obvious that there is no conduction delay. In lead I, the transverse lead, the S wave is not fully formed and comes slowly down like a semicircle to an inverted T wave. In lead III, the lateral lead the S deflection is down and on rising is not fully formed but curves up to a normal T wave. In lead I the R wave is inverted and in lead III the S wave is inverted.

This change is rather similar to the type of curve that was described by Pardee as pathognomonic of cardiac infarction. Professor Ritchie and Dr. Gilchrist have shown that similar curves can be obtained in slow ischaemic processes and also that it is impossible to localise the infarct by the type of curve.
This radiogram was obtained at a later date. It will be seen that there is marked dilatation of the aorta. Although one does not ascribe to this view that all dilated aortas are syphilitic in this case the X-ray picture is confirmatory evidence of our diagnosis of specific aortitis.
The electrocardiogram shows us then that there is some myocardial disease but that it is not absolutely certain that infarction is the cause.

**Summing up our diagnosis.**

Syphilitic aortitis causing narrowing of the mouths of the coronaries and resulting a process of slow ischaemia of the heart muscle and also resulting in a fairly rapid heart failure.

**Prognosis.**

The prognosis is poor and the patient will never be able to return back to his work. At present the rapid rate of the heart is able to be controlled by medication but heart failure or possibly death in an attack of cardiac asthma will carry the patient off.

**Treatment.**

On admission the patient was put on a milk and chicken diet. This was gradually built up and he is now getting light diet.

As soon as the laboratory reported a triple positive Wassermann he was put on the following prescription,

\[
\begin{align*}
\text{P} & \quad \text{Liq. Hydrarg. Perchlor. mxxx} \\
& \quad \text{Pot. Iod. grxxx} \\
& \quad \text{Aqua ad} \\
& \quad \text{Sig. To be taken thrice daily.}
\end{align*}
\]

He has continued to take this all the time he has been in hospital.

In order to improve his appetite etc. he was also given the following,

\[
\begin{align*}
\text{P} & \quad \text{Ferri et ammon cit. mxxv} \\
& \quad \text{Liq. Arsenicalis. mvi} \\
& \quad \text{Tr. Nux Vom. miv}
\end{align*}
\]

Owing to the fact that his pulse rate was rising it was decided to give him digitalis. The tincture was given in twenty minim doses thrice daily.

<table>
<thead>
<tr>
<th>Pulse rate</th>
<th>Total digitalis</th>
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<tbody>
<tr>
<td>16th May 105</td>
<td>m60</td>
</tr>
<tr>
<td>17th 100</td>
<td>m120</td>
</tr>
<tr>
<td>18th 100</td>
<td>m180</td>
</tr>
<tr>
<td>19th 95</td>
<td>m240</td>
</tr>
<tr>
<td>20th 95</td>
<td>m300</td>
</tr>
<tr>
<td>21st 80</td>
<td>mxx</td>
</tr>
</tbody>
</table>

On the morning of the twenty first the dose was reduced to mxx thrice daily.
The table is shown to illustrate the fact that full digitalisation is usually attained when twenty minims or about five drachms have been administered. The dose is then reduced to a level which just keeps the patient digitalised. The full therapeutic action of digitalis will be discussed in another case.

The question that has to be decided is whether or not a full antispecific course of arsenic, mercury or bismuth, and iodides should be embarked on. Should his cerebro-spinal fluid be tested for complement fixation, the cells counted and the globulin estimated etc?

In view of the fact that the heart condition is a severe one I think it advisable that arsenic in an intravenous or intramuscular form should be avoided, plenty time to begin if there is any marked improvement in the immediate condition of the heart. The mild antispecific treatment in the form of mercury and iodides are all that the patient can tolerate at the present. There is no need to test his C.S.F. in the meantime as there is a complete absence of neurological signs. At a later date this might possibly be carried out.

Even with treatment the heart condition is poor and not likely to respond to treatment, other than the slowing effect of digitalis.

It is stated that all cases of syphilitic aortitis once subjective symptoms are complained of die within two and a half years. In addition our patient has very probably got some degree of infarction and his expectation of life is of the poorest.
Mrs. Annie Murray.  Aet. 51.

Recommended by Dr. Kirkness, Torphicen Street.

Admitted 29th March 1931.

Discharged 12th May 1931.

Complaint. Breathlessness on exertion for about one year. Swelling of the feet, eyelids and 'stomach' for about one fortnight.

History of the Present Illness.

About a year ago, in March 1930, the patient became very conscious of the beating of her heart. At the same time she found that she was getting very breathless and occasionally experienced a pain behind the sternum. At this time she never noticed that her feet became swollen.

She was admitted to the wards of Doctor Goodall where she stayed for three months and felt very much better. Later she was transferred to a convalescent home and stayed there for a month before she returned home.

Since the end of June last year she has been almost entirely confined to bed. Sometimes when she felt a little better she got up for a few hours and on five occasions went out of doors. Whenever she did this however she became very breathless and suffered from palpitations. Latterly she has been entirely confined to bed.

Her chief complaints during this time were the palpitations and also a bad cough which had come on since she had taken to her bed. She brought up small amounts of sticky sputum but is sure that it was never blood-stained.

Two weeks or so before admission she noticed that her eyelids were a little swollen and a day or two later that her ankles were also swollen. This was the first time that she had ever noticed it. At this time also she felt some mild abdominal discomfort after she had had a meal and was...
alarmed to find that her abdomen was increasing in size. She has never vomited nor has she noticed that her stools were abnormal in colour. Although she had formerly suffered badly from bleeding piles these have entirely ceased to trouble her during the past year. She has lost a lot of weight during the past three years.

Previous Health.

She seems to have had rheumatic fever when she was a schoolgirl but is unable to recall how old she was or what form the illness took. Patient was attacked with scarlet fever and also with mumps in her early childhood. Apart from these illnesses she has been a healthy enough woman. She has never had pneumonia or pleurisy and doesn’t catch cold easily.

Obstetrical History.

This is far from satisfactory. Her first baby was a girl and was born thirty three years ago. She is alive and well. The next baby was a boy who died of pneumonia at the age of two years. Thereafter comes a succession of six miscarriages, she carried the first four months but all the rest were aborted at about the third month. She cannot remember when the last miscarriage occurred but thinks it was just before the war.

Family History.

Her mother died at the age of 61 of an abdominal swelling. Her father was killed in the Indian mutiny of last century.
Examination.
The patient is a petite woman of moderate intelligence. Her lips are deeply cyanosed and there is a marked malar flush of a deep purple colour. Although her breathing is rapid she is not orthopnoeic and lies back on her pillows quite comfortably. Her ankles and the lower parts of her leg are swollen and the swelling pits on pressure. The swelling is not gross.

Cardiovascular system.
The pulse is wholly irregular in time and force. The characteristics of the pulse wave naturally varied greatly. The sphygmomonometer reading of her blood pressure was 110/75. The radial and brachial arteries were slightly thickened.

The Heart. On inspection the apex beat was seen to be diffuse and to vary in force from time to time. On palpation this was confirmed and the exact position was found to be in the fifth interspace 4½ inches from the mid-line. Inspection also showed that the veins at the root of the neck were engorged - the external jugular was plainly visible from the mandible to the clavicle - and showed marked pulsation. On percussion the upper border was found to be at the level of the second right interspace, the left border was four and a half inches from the mid-line and the right border two inches from the mid-line.

Auscultation. The first sound at the mitral area was replaced by a soft blowing murmur which could be followed back into the axilla. At the mitral area the second sound was not well heard but was found to be closed. About one inch internal to the mitral area (apex beat) there was a very short but quite distinct mid-diastolic murmur. It was soft in character. At the pulmonary area there was accentuation and reduplication of the second sound.

The electrocardiogram will be discussed later.
Respiratory System.
As has been stated although the breathing was increased there was no distress or orthopnoea. The chest wall was very thin. Expansion was equal on both sides but was feeble. There was no alteration in the vocal fremitus. Resonance was unimpaired. At the base of both lungs some coarse crepitations could be heard. The sputum is a tenacious muco-purulent one and moderately abundant.

Alimentary System.
Tongue is slightly furred but is moist. There is a solitary molar in the upper jaw and the lower jaw is edentulous. Patient has a plate for the upper jaw but none for the lower.
The abdomen is greatly swollen. On palpation it was fairly tense. In the upper abdomen the liver was found to extend down to the level of the umbilicus on the right side but did not appear below the costal margin on the left. Percussion confirmed this. Dulness began again about two finger breadths below the umbilicus and extended down to the pubes. On each sides it extended up to the costal margin. A fluid thrill was easily elicited. On changing the position of the patient on to her side the flank that was uppermost became markedly resonant while the extent of the dulness on the lower side increased. i.e. shifting dulness on percussion. The girth of the abdomen was 38 inches.
The X-ray report showed the presence of some stones in the gall bladder but no lesion in the stomach, colon or rectum.

Genito-urinary system.
Kidneys not palpable. The urine was brown in colour and was of S.G. 1026. There was a trace of albumen present and usually some mucus. No casts or other abnormality on centrifuging.
A vaginal and bimanual examination showed no abnormality. The uterus was of the senile type, small hard and anteverted.

Nervous system.
The cranial nerves were all intact. The pupils responded equally to light and accommodation. There was a marked arcus senilis.

There was no motor or sensory loss in the limbs. The reflexes were present and normal.
DISCUSSION.

1) DIAGNOSIS.

Auricular fibrillation is not usually a difficult condition to diagnose. The characteristic pulse, the deficit between the apex beat and the pulse and lastly the electrocardiographic and polygraphic findings all make the condition a simple one to diagnose.

In the case of Mrs. Murray we noted that the pulse rate was wholly irregular both in time and in force. We noticed also that there were more cardiac contractions as shown by the apex beat than there were pulse waves at the radial artery. Actually this would almost enable one to diagnose the condition right away but there are several conditions which very rarely might confuse the diagnosis.

Let us first consider the electrocardiogram.

This is quite characteristic. The p wave is entirely absent and its place is taken by a great number of small irregularly placed and sized waves. It is impossible to count them correctly on this particular record but I estimated that they were occurring at the rate of 520 a minute.

There is no lengthening of the Q-S interval nor of the R-T interval. There is however an inversion of all the T waves in all leads. However as the record was taken after digitalis administration one does not put too much stress on this finding. (See later under signs of digitalis overdosage)

The electrocardiogram showed that the condition present was auricular fibrillation.

The following conditions have to be excluded,

a) Sinus arrhythmia. Naturally in this particular case this caused no trouble but in young patients with possible fibrillation it has to be excluded. The patient is asked to hold his breath and if the irregularity
stops it is due to sinus arrhythmia.

b) Multiple extrasystoles are occasionally the cause of trouble especially if they are arising from different foci in the heart. The electric records and the polygraph soon clear this error. The p waves will be found to be present and are of course occurring at the same rate as the ventricular responses. (Note. The McKenzie polygraph seems to have been entirely superseded by the electrocardiogram. In my clinics etc. at the Infirmary I have never been shown a record nor have I seen the instrument demonstrated. Therefore I have not attempted to make tracings for this and other cases. Why this should be is difficult to understand as it is presumably easier to get a polygraph done than an electrocardiogram). This showed a healthy gastro-intestinal tract but showed the

c) Auricular flutter may be only diagnosable by the electrocardiograph. The p waves are present and about twice as frequent as the ventricular responses.

d) A theoretical error would be the coexistence of a partial heart block with many dropped beats and a rapid heart. Again the mechanical aids to diagnosis would be necessary.

e) It has been recorded that auricular fibrillation has coexisted with complete heart block. Thus the auricles were beating at over 500 beats a minute while the ventricles were beating at the rate of 60. One would imagine that such a diagnosis would have only academic interest. Having diagnosed auricular fibrillation we turn our attention to the state of the abdomen. Is it possible that the ascites is due to some other cause than heart failure? Could a malignant condition of the abdominal cavity especially of the pelvic organs not coexist with the heart condition. That the ascites is due to heart failure is made very probable by a consideration of the following facts.
Firstly the liver was enlarged and tender, but it was entirely smooth and tended to keep its normal shape. Although one has read of cases of malignant 'hepatomas' enlarging a liver yet retaining its normal shape the possibility of this is over-rulled by the fact that she has no gastric disturbance and has a very good appetite.

Secondly a careful bimanual failed to reveal any pelvic cause, the ovaries were not enlarged and the uterus was shrunken. The cervix was typically that of an ageing woman. The breasts were atrophic and shrivelled and showed no evidence of any malignant change.

Other abdominal conditions were eventually ruled out by an X-ray examination. This showed a healthy gastro-intestinal tract but showed the presence of some gall-stones.

An analysis of the abdominal fluid on examination showed the presence of a few lymphocytes and mononuclears. There was no growth on culture and no malignant cells found.

We thus conclude that the ascites is due to cardiac failure with chronic venous congestion of the portal system.

The striking features in this case are I think the absence of orthopnoea and the maintainence of a good appetite. The typical picture of heart failure with the patient finding every breath an effort and suffering from anorexia etc. is quite absent.

The diagnosis then is centred in the heart. The auscultatory findings are in accordance with a mitral stenosis with fibrillation. The mitral systolic murmur denotes incompetence as well and the mid-diastolic murmur is characteristic of stenosis when fibrillation has occurred.
I will next discuss three points, firstly the etiology of fibrillation, secondly the administration of digitalis and lastly the treatment of cardiac oedema all in particular reference to this case.

**Etiology of fibrillation**

Fibrillation may be said to arise in the large proportion of cases in three main conditions. It may arise as the result of cardiac rheumatism. This furnishes the largest number of cases. And it arises especially in cases of mitral stenosis. The rheumatism causes a myocarditis and an endocarditis. If the mitral valve is attacked the consequent stenosis which may occur leads to dilatation of the left auricle. Sooner or later this leads to fibrillation. In reading various articles on the etiology one is struck by the absence of any pathological change being described in association with fibrillation. Indeed one review of the subject states that in 150 cases there was no constant pathological change which could be stated to occur with any degree of regularity. Presumably the cause lies in the chemical or physical properties of the heart muscle.

The next largest group of patients were those who showed only age as a common characteristic. Elderly people may have fibrillating hearts with no morbid changes either in the myocardium or the arteries of the heart. The significance of this is quite obscure.

The next group of patients who may have fibrillation are those with hyperthyreoidism. Again the exact cause is quite obscure.

The majority of cases then fall into these three groups but there are a host of conditions which may cause fibrillation in isolated cases. Such causes are syphilis, infections especially fibrillation in a patient dying of pneumonia, after alcoholic and other dietetic indiscretions, and many other causes. We can only state that fibrillation may be expected in states but what governs its onset is entirely unknown.
What category can we place Mrs. Murray in? Was she an example of the rheumatic type or of the elderly group?

Certain physicians of the Edinburgh School would certainly quibble at the diagnosis of a mitral stenosis in a woman over fifty years of age, especially in view of her obstetrical history. That a woman who had had two live children and five miscarriages and who had a mitral stenosis could live till over fifty before her heart broke down would seem quite impossible to them. I can remember one physician who asked a clinic why one NEVER saw a case of mitral stenosis in a patient over forty and on receiving no reply said 'Why, they have all died of heart failure before they have reached that age'. However in the past three months in the wards I have seen at least two definite cases of mitral stenosis and have banished from my mind the statement that you must never diagnose mitral stenosis in an elderly patient.

We will assume that Mrs. Murray did have rheumatic disease and it is possible that a combination of age and myocarditis of the left auricle following mitral stenosis have eventually brought on the fibrillation. The onset of the fibrillation was possibly about a year ago when she first noticed the praecordial pain and the breathlessness.

**The Administration of Digitalis.**

The action of digitalis is twofold, it acts via the vagus and also directly on the heart muscle. Its actions can be summarised as follows:

*By the Vagus.*

- It slows the pace maker, it SHORTENS the refractory period of the auricular muscle, it depresses conductivity in the conducting tissues.

*Directly.*

- It has a depressant action on the conducting tissues, it is thought to increase the strength of systole and lastly it **lengthens** the refractory period.
It will be seen therefore that the action on the auricle is twofold, by the vagus it will by shortening the refractory period actually increase the rate of the impulse. By its direct action it will tend to slow the rate. The ultimate action however will depend on which action predominates and in most cases it is found that the vagal action predominates and thus the rate of the impulse actually increases in digitalis therapy. Thus the beneficial effects of digitalis in fibrillation are not located in the auricle, it is the depressant effect on the conducting tissue which is the important action. Both the vagal and direct actions tend to depress the conductivity in the conducting tissues and especially in the bundle of His. The ventricle thus responds to much fewer auricular impulses. Although the late Prof. Cushny thought that in the early stages of digitalis therapy the vagal action predominated and in the later stages the direct action predominated this has not been entirely confirmed.

Method of Administration. The tincture of Digitalis is the drug which is most commonly employed in this school. The method used is a slow one, about \( \mu \text{xv-xx} \) of the tincture are given trice daily till the pulse rate falls to a requisite level. When this has been attained then a maintainence dose of about \( \mu \text{x} \) is used.

Many other methods of administration have been evolved and possibly the most commonly used of the quick methods is Eggleston's massive dose method. The amount required to digitalise the patient is calculated from the weight and from the fact that for each pound of weight .15 of a cat unit is required. Having found the total then fractions of it are given at six hourly intervals, the fractions commonly employed are a half, a quarter, an eighth, and a residual dose. This method is not recommended by clinicians in this school but apparently is in use elsewhere.
To show the effect of Digitalis on the pulse rate in Auricular Fibrillation.
In Mrs. Murray's case digitalis was commenced as soon as she was admitted. It will be seen on reference to the chart that her pulse rate fell very rapidly and after having received 135 she was put on a maintenance dose. This amount was rather small as it is usually found that it requires 20c.c. or about 3v it digitalise a patient. Nevertheless it had a striking effect on the rate of the heart causing it to slow down some fifty or sixty beats a minute.

In ordinary cases the maintenance dose is continued indefinitely but in Mrs. Murray's case it had to be stopped on the ninth day after administration because of a very significant finding.

Signs of overdosage etc.

Anorexia, nausea and vomiting during the administration of digitalis are symptoms which are very suggestive. Although they do not necessarily mean overdosage or intolerance in every case they are symptoms which need very careful watching.

But if these symptoms arise in conjunction with certain heart signs then digitalis is best immediately discontinued. The classical signs of digitalis poisoning are as follows. Extrasystoles occur and show a special tendency to coupling. i.e. every second beat is an extrasystole. This is an absolute sign that digitalis must be discontinued. Another indication of toxic action is the onset of heart block. Any grade may be met with up to complete dissociation of the ventricular contractions. In the electrocardiogram a very characteristic change occurs. The T wave becomes inverted in all leads and there is also a tendency to a depression of the interval before the T wave.

Turning to Mrs. Murray we find that on the ninth day after the commencement of digitalis therapy the pulse fell to sixty. This was taken to be an indication that a major degree of heart block was tending to occur.
and the administration was immediately stopped. Two days later the pulse rate rose to eighty beats a minute so we can assume that this was a true toxic effect of digitalis. The patient suffered from no gastro-intestinal symptoms at all. With reference to the electrocardiogram I pointed out that the T wave was inverted and also it is seen that there is a depression before the T wave. The electrocardiogram was taken a week after the digitalis was stopped and the problem is how far these changes can be ascribed to the toxic effect of the drug. Unfortunately I can get no information as to how long the electrocardiographic changes persist after the withdrawal of the digitalis. However, the changes present in Mrs. Murray's electrocardiogram are quite typical of overdosage.

Of course by 'overdosage' we mean that in this particular case the patient was not able to tolerate an amount of digitalis that would not in many other cases have sufficed to digitalise them. It is impossible therefore to lay down with any degree of accuracy or safety the actual amount of digitalis which is required in any given case. Each patient must be carefully watched and the toxic effects looked for, it took only 145 to digitalise Mrs. Murray but in another of our cases we see that it took 300.

Quinidine is a drug which justly deserves a place in the treatment of fibrillation. It acts mainly by depressing the conduction time and increasing the refractory period of the heart muscle. In this case there were two contraindications to its administration. The heart was failing and the muscle of the auricle due to the advanced stenosis of the mitral valve was not at all in a suitable state for quinidine therapy. Its main indication is in fibrillation of recent origin and in cases where heart failure is absent. Quinidine acts by converting the circus movement of fibrillation into normal rhythm. The heart must always be fully digitalised before it is used.
Lastly let us turn to the consideration of the treatment of cardiac oedema. The patient usually finds that the most comfortable position is when he is sitting up in bed well supported with pillows. In some cases one may treat the patient by getting him into an arm chair. A difficulty that is sometimes experienced is that in this position sleep may be difficult. Usually however the patient soon overcomes this difficulty. It is suggested also that the lower limbs should be lightly bandaged and light massage be started. This is done before oedema has appeared.

The diet needs careful attention. The main indication is to avoid indigestion and flatulence and thus it is obvious that the fluid intake must be reduced to the minimum. Not more than two pints of fluid must be given per day and it is advisable to curtail this if possible to about 30.

Some physicians advise a very light diet containing milk, cream, jelly, and farinaceous foods. They state that the gastric juice is not able to digest any of the heavier protein foods. Prof. Murray Lyon does not agree entirely with this. He advises that the fluid be greatly restricted and given in the form of milk and also that salt be eliminated as far as possible. He thinks that the soft sloppy foods are far more apt to cause flatulence and indigestion than an ordinary light diet. The patient thus receives a light dry diet which may consist of anything he fancies. He will himself avoid those foods which cause flatulence etc. The fluid in the form of milk should always be taken in between meals.

In the treatment of cardiac oedema one has to avoid the more drastic methods which are permissible in say an acute nephritis. Hot baths and packs which have a marked depressive action on the heart have to be entirely avoided. Naturally the skin must be kept healthy by judicious tepid sponging and drying. Again in acute nephritis there is good indic-
-ation to get rid of fluid by the bowel and thus strong purges are given. In the treatment of cardiac failure this is not advisable and all that should be done after a good initial purging is to keep the bowels well open by a laxative or a morning saline. These then are the simpler general measures, rest, posture and dieting. Sooner or later however one will be forced to use diuretics and the main problem is to decide which of them to use.

Digitalis itself by toning up the heart and by its slowing action has often a marked diuretic action. In the present case it had only a moderate effect increasing the amount of urine by about 500c.c. per diem.

The Guy's pill is often of use in dealing with cardiac oedema. It consists of a graineach of powdered leaves of digitalis and squill and of pil. hydrarg. One pill is equal to fifteen minims of the tincture of digitalis and if it is to be administered any tincture that is being given must be stopped.

The striking feature with most other diuretics is that they do not act the same in every case. In one patient they may cause an excellent diuresis in the next only a very moderate one. Or they may have no effect or even lessen the amount of urine being excreted. Thus one finds that in treating cardiac oedema it is best not to persevere too long with one drug if the results are unsatisfactory but to change over to another drug till one is found that is suitable.

Calcium Chloride is a salt which often gives surprisingly good results. It has to be given in fairly large doses, Prof. Murray-Lyon recommends 2-5 Grammes three times a day. If this has no effect then one has to turn to one of the so-called 'specific' diuretics. One could only wish that they really were specific.

Urea is probably the simplest and is given in moderately large doses, it
To show the action of Salyrgan on Mrs. Murray.

Salyrgan was given in 10cc. saline.
is given well diluted in orange or lemon juice to mask the unpleasant metallic taste. One to two ounces daily are given. Prof. Lambie has noticed that certain patients to whom he gave urea and who did not respond particularly well to urea later responded remarkably well to novasurol. There is a large group of xanthine derivatives that have been exploited by manufacturing chemists and are known by many names, caffeine, theocin, diuretin and euphyllin. They are given in doses averaging about five grammes daily and may or may not have an effect on the oedema.

Novasurol is a drug which has been very popular in oedema cases. It contains about 34% of mercury. The diuresis it promotes is very quick and starts within three hours. It is given intramuscularly in .5 - 2 c.c. doses. Lambie states that many patients who have responded to no other drugs will respond to novasurol. The drug however must be considered a dangerous one as it may cause a nephritis or a colitis. It is therefore contraindicated in all cases of heart failure due to or co-incident with renal conditions.

Salyrgan is a compound mercury and sodium salt. This drug was used in the case of Mrs. Murray. It is given intramuscularly or intravenously in .5 - .2 c.c. doses. The chart on the opposite page shows its effect on Mrs. Murray. It will be seen that its effect gradually tails off after a week or so. This is another problem that one has to face in cardiac oedema is that a drug tends to loose its effect in a very short time. It has been suggested that the exhibition of ammonium chloride enhances the action of salyrgan and should be given in fifteen grain doses twice daily. Some physicians do no give small doses of salyrgan every day but give larger doses of two c.c.s twice weekly.

Sooner or later one has to consider the mechanical removal of fluid and when this is started it can be said that the end is not far off. The
abdomen must always be tapped before the muscles loose all their tone. It is needless to anaesthetise the patient either locally with novocain or ethyl chloride. The sharp prick of a Southey or larger tube does not hurt much. The fluid is drained off slowly and particular attention is taken to look for any appearance of shock. Pituitrin or adrenaline should be immediately given.

In Mrs. Murray we noticed that so marked was the ascites that we had to take particular care to exclude a coincident malignant cause. Her legs were only very moderately oedematous. Paracentesis of the abdomen was carried out on the day of admission. A point midway between the umbilicus and the pubis was selected. Seven and a half pints were removed and the patient became very collapsed. A fiftieth of strychnine and a 1/250 of strophanthin were administered and the patient soon rallied. On two other occasions the abdomen was tapped and a total of twenty four pints were removed.

Prognosis.

She will never recover from this attack of heart failure and will be bedridden for the remaining months of her life. Her own doctor will try and relieve oedema as far as possible and keep her comfortable. She will very possibly die of some intercurrent infection.

Previous Health. Chickenpox, measles, bronchitis. When 3 years old she had diphtheria. In 1926 she was in the Sick Children's Hospital on account of her 'heart'. She cannot remember ever having growing pains.

Family History.

Father died at 53 of 'sleeping sickness'.

Mother age 80 is alive and well.

Three sisters and one brother alive and well, one brother and sister died...
HELEN SMITH.  Act 13½.  Schoolgirl.

Address. School Brae, North Queensferry.

Referred from

Admitted 29-4-31.

Complaint. 'Rapid beating of the heart'
'Breathlessness'

History.
The patient states that her mother says she was a strong baby until she had diphtheria at the age of 2½ years. After that time and as long as she can remember herself she has noticed that whenever she gets excited she begins to feel her heart beating very quickly, also when she does anything in the slightest strenuous she quickly gets breathless. She has never noticed that she was blue in colour and never remembers her mother telling her she was at any time.

Apart from a period when she was in the Sick Children's Hospital she has attended school regularly. Since the family have gone to stay at Queensferry however she has only been going to school for half the day.

A week before admission she found that she was feeling very tired and was made breathless very easily. The doctor was called in and he put her to bed for a few days. She then felt much better but was sent up to the Royal Infirmary to see if she could be admitted for a short period and get a 'good rest'.

Previous Health. Chickenpox, measles, bronchitis. When 2½ years old she had diphtheria. In 1926 she was in the Sick Children's Hospital on account of her 'heart'. She cannot remember ever having growing pains.

Family History.
Father died at 52 of 'sleeping sickness'
Mother age 50 is alive and well.
Three sisters and one brother alive and well. One bother and sister died
from diphtheria.

**Examination.**
The patient is a rather pale child but she exhibits no cyanosis. She rather resents being examined and is of the 'spoilt child' type.

**Alimentary System.**
Her lips are of good colour. Except for two septic stumps her teeth are in good condition. The tongue is clean and moist. The tonsils appear to be clean and healthy. Her abdominal wall was thin and moved freely on respiration. On inspection marked aortic pulsation was seen. On palpation nothing abnormal was found.

**Respiratory System.**
On inspection the chest is seen to be of a typical rachitic type. It is very flat and shows a marked Harrison's sulcus on both sides. On percussion it was found that the lungs were normal in resonance and on auscultation the breath sounds were found to be of a harsh vesicular type. There were no accompaniments.

**Nervous system.**
Apart from the fact that she was a 'nervous' type of child examination revealed no organic or functional disease.

**Genito-Urinary System.**
The urine was lemon in colour, of specific gravity 1023, but showed no abnormality at all.

Patient had not reached the Menarche.

**Cardio-vascular system.**
The praecordium was of the rachitic type. There was a certain amount of bulging and at the level of the upper part of the xiphisternum there was a horizontal sulcus to be seen - the Harrison's sulcus.

The apex beat was very diffuse and was clearly visible over an area
about 2½ inches in diameter. It is very forcible in character. On palpation over the mitral area no thrill can be felt, but when one placed one's hand over the base of the heart there was both a marked diastolic and systolic thrill. The apex beat was found to be five inches from the midline in the seventh intercostal space.

On percussion the upper border of the heart was found to be at the third rib. The right border two inches from the midline and the left border five inches from the midline.

Auscultation. Practically over the whole heart a loud rough and continuous murmur can be heard. It shows marked diastolic and systolic accentuation but there is no recognisable first sound at all. In the pulmonary area a closed second sound can be heard. The systolic murmur is propagated into the vessels of the neck and is also clearly heard at the angle of the left scapula.

The pulse. According to the chart the rate of the pulse varied between seventy and hundred beats per minute. It was regular in time and force and showed no sinus arrhythmia. The upstroke was forcible and the pulse collapsed suddenly i.e. a Corrigan pulse. There was no thickening of the arterial wall. The blood pressure was 140/30.

The heart is originally a single tube but as a result of unequal growth the part destined to become the ventricles passes caudally and the part that is to become the atria passes cephalwards. The primitive thus looks S shaped. The atria bulge round the bulbus cordis and the sinus venosus lies behind the ventricle. The heart has now roughly the shape of an adult heart.

As yet however there is only a hollow tube inside and this becomes divided up into various compartments by the growth of certain cushions and septa.
Congenital abnormalities of the heart and the great vessels are by no means rare. At the Sick Children's hospital I saw four cases of 'congenital hearts' and was impressed by the fact that it was sometimes only with great difficulty that the physicians could decide that it was a congenital abnormality and not a rheumatic infection that was causing the symptoms and signs complained of. During the past three months in Professor Ritchie's wards I have seen two cases of congenital heart disease, one case of complete transposition of the viscera including dextrocardia, also there was a case seen in the out patient department which I did not see of coarctation of the aorta.

One can say that congenital heart disease is of academic interest rather than from any interest in the treatment which is practically nil. I will discuss very briefly the development of the heart and the vessels then enumerate the possible malformations before discussing the diagnosis and treatment of this case.

Last year in a course of embryology from Professor Robinson he showed us numerous sections of all stages of development of the heart of mammals, etc. I will give a summary of his teaching as to the development of the heart in man.

The heart is originally a single tube but as a result of unequal growth the part destined to become the ventricles passes caudally and the part that is to become the atria passes cephalwards. The primitive thus looks S shaped. The atria bulge round the bulbis cordis and the sinus venosus lies behind the ventricle. The heart has now roughly the shape of an adult heart.

As yet however there is only a hollow tube inside and this becomes divided up into various compartments by the growth of certain cushions and septa.
Rough Diagrams of development of the heart. (After R.H. Hunter.)

From the dorsal and ventral margins of the atrio-ventricular canal a fold of endocardial cushion forms called the septum primum. Then it has grown down and the ventral part breaks down and thus forms the foramen ovale. It is the second pouch of the right atrium except at the point where the septum primum meets the endocardial cushion. Thus the venous return enters the ventricular foramen and is not closed by the venous valves but by the growth of a septum arising in the bulbus cordis which separates the right and pulmonary arteries. Meanwhile a sinuses of the heart has occurred in the ventricle, a septum has grown up to form the coronary sinus and left the aorta. This sinus therefore forms the ventricular foramen and is partly closed by the venous valves or valves which allow pass from the right to the left atrium but not in the opposite direction.

It is known as the foramen ovale which is often the site of pathological processes at the upper part of the aorta. The heart is divided into four chambers which are separated by either valves or septa. At birth the foramen ovale is closed but in an appreciable number of persons there is an intact foramen ovale of small size which has caused no symptoms during life.

I will not describe the aortic arches but mention that from the sixth arch a portion of the outgrowth is retained as the ductus arteriosus which unites the left pulmonary artery to the end of the arch of the aorta.

From this brief description we can understand that congenital abnormalities-
From the dorsal and ventral margins of the atrio-ventricular canal a pair of endocardial cushions arise and from this growth the atria are separated from the ventricles. From the cephalic end of the atria a septum arises and slowly grows down to the endocardial cushions. It is called the septum primum. When it has grown down and met the cushions its central part breaks down and thus forms the foramen ovale. A septum secundum arises in the right atrium and passing down fuses with the septum primum except at the point where the foramen ovale exists. Here the septum secundum forms a flap like valve which allows blood to pass from the right to the left atria but not in the opposite direction.

Meanwhile a similar process has been occurring in the ventricles. A septum has grown up from its floor but fails to reach the atrio-ventricular cushion. This opening therefore forms the ventricular foramen and is not closed by a second septum but by the downgrowth of a septum arising in the bulbus cordis, which separates the aorta and pulmonary artery, meeting the crescentic edge of the interventricular septum. In adult life it is known as the pars membranacea and is often the site of pathological processes attacking the bundle of His.

The heart is now divided into four chambers which are separated by either valves or septa. At birth the foramen ovale is closed but in an appreciable number of postmortems patent foramen ovale of small size albeit but which have caused no symptoms during life.

I will not describe the aortic arches but mention that from the sixth arch a portion of the outgrowth is retained as the ductus arteriosus which unites the left pulmonary artery to the end of the arch of the aorta. From this brief description we can understand that congenital abnormalit-
ies can occur almost anywhere in the heart and can be slight so that they never cause symptoms or can be so severe that they are incompatible with life.

The lesions found are of great number therefore and all we will discuss are the commonest. Maumde Abbott in Osler's system of Medicine is extremely full on the subject but I'm afraid I had to abandon my attempt to read her articles at an early stage!

1) The heart is originally formed by the fusion of paired primordial vesicles. If these persist then double heart may exist.

2) Ectopia cordis results from the failure of the body wall to fuse round it. It is not compatible with life. A man is known to have carried his heart in his neck.

3) Dextrocardia results when the original rotation of the heart tube is to the left instead of to the right. We mentioned that we had seen a case of this condition. An athletic boy of seventeen had been refused by an insurance company as a risk. His doctor sent him up to see Prof. Ritchie who found that there was a complete transposition of viscera. The question was whether or not the heart was diseased in any way. It was found to be quite healthy.

4) Persistent foramen ovale exists as we have pointed out in a considerable number of people. Some say as high as 25% - naturally the opening is very small. By itself it is of no consequence and leads to few clinical signs or symptoms. It is the presence of a large foramen which has caused the condition of paradoxical embolism to occur. This is a rare condition but beloved by all authors of text-books of pathology.

5) A persistent opening between the ventricles also occurs. Again its presence is compatible with perfect health. The physical signs are the well known Bruit de Roger, a systolic thrill and murmur.
6) Patent ductus arteriosus also occurs. It often gives rise to a continuous murmur throughout systole and diastole.

7) Stenosis of the pulmonary artery is probably the most easily recognised and the most serious abnormality of the heart. It is frequently associated with other congenital abnormalities. The signs are a systolic thrill and murmur and possibly an absent second sound in the pulmonary area.

8) Coarctation of the aorta is a condition where there is a minor or major degree of narrowing of the aorta at a point just distal to the origin of the ductus arteriosus. It is alleged that in a very large proportion of autopsies on children show a mild degree of this narrowing and frequently many adults. The signs of a collateral anastomosis is a very striking one. Those arteries that are supplied by branches above the stenosis anastomose with those below and thus many enlarged and visible pulsating arteries can be seen coursing over the chest as they pass down to anastomose with arteries lower down. The case I mentioned as having been seen by Dr. Small in the out patient department was in an adult woman so the condition is compatible with a fair degree of longevity.

As to the causation of these abnormalities few investigators nowadays agree with the older theories of foetal endocarditis. That a foetal endocarditis does occur is probably not disputed and most textbooks of pathology contain pictures of right sided cardiac lesions of the newly born child. However it would appear improbable that all the multifarious abnormalities could be produced by inflammatory causes. And also histological studies fail to reveal any signs of this supposed inflammation in the majority of cases. Probably the majority of physicians agree with Sir Arthur Keith that the cause is much more obscure, being lost in the ill understood factors which govern foetal growth and structure in general.
The symptoms of a congenital abnormality naturally vary greatly in their prominence. There may be none or on the other hand one may see an example of the classical 'blue baby' in whom life is of a few minutes or hours in duration. It is impossible to exclude a rheumatic basis for some of the symptoms. Speaking generally one can say that the two main symptoms are breathlessness on exertion and possibly a degree of blueness. Or the patient may be conscious of the tumultuous beating of his heart. On examination of such a patient one would confirm the breathlessness and the cyanosis and would probably find a degree of finger and toe clubbing which might vary from a mere shininess at the base of the nail or be of the classical type. An examination of the blood is frequently helpful if it shows the increase of red cells that is so often quoted as being a common sign in congenital heart disease. The size of the red cells usually is somewhat larger. The examination of the heart would reveal that there was some abnormality present.

Turning to the case of Helen Smith we see that up to the age of two and a half her mother considered her a healthy baby so there could have been no gross abnormality at birth. During childhood she seems to have had more than her fair share of childhood's complaints and we notice that bronchitis was one of them. Her main symptoms are dyspnoea on exertion and consciousness of the heart beat. This is practically all her history except that there is no history of any rheumatic manifestation.

On examination of the heart one is struck by the continuous murmur and thinks of a patent ductus arteriosus. The second sound in the pulmonary area is present but absent in the aortic area. The character of the pulse i.e. its collapsing character seems to indicate that there is some aortic incompetence. The enlargement of the right heart would fall in line with a diagnosis of patent ductus arteriosus. Prof. Ritchie has pointed out
that it is rare to find a single congenital lesion and also that it is
unwise to pursue the profitless task of diagnosing every and each lesion
present. A diagnosis of 'congenital heart' is often sufficient.
Although it is impossible to exclude a rheumatic basis for some of the
lesions present we have no positive evidence that there has been any
rheumatic infection. One would point out however that such a heart is
one which would very easily fall to rheumatic infection or other more
serious type of infection. For instance it has been shown that in sub-
acute bacterial endocarditis—'kriegsendokarditis' of German authors-
attacks the aortic valves in the majority of cases and in an appreciable
number of cases the aortic valve was a congenitally bicuspid one.
The Prognosis is probably poor in this particular case. Both her
right and left heart are considerably enlarged and dilatation and heart
failure are bound to occur sooner or later. She will probably not survive
beyond the age of thirty. She will be the easy prey of any intercurrent
infection such as pneumonia or severe influenza.
The Treatment.
This is mainly expectant. Her life must be so ordered that there is no
severe strain thrown on her heart at any time. Both physical and mental
effort must be of the mildest.
The diet of course will be so planned to avoid flatulence and so cause
any possible embarrassment to the heart. She is at present being given
radiomalt and iron in attempt to improve her general condition.
She will probably not be advised to marry as childbearing would almost
certainly cause the heart to fail.
It is wise however not to impose too many limitations on her life which
one could easily make intolerable by refusing to let her play with other
children etc. A carefully planned life with not too many strictures is
the aim to be planned for.
Patrick Crombie.  
Admitted.  7th. March 1931.  
Dr. Malone, Rankeillor Street.  
Complaint. The patient collapsed and was brought into the R.I.E by the police.

History of present illness.

About six months ago the patient was employed in connection with some building work being carried out at Craigleith hospital. One Saturday evening at this time he was walking along to his house when he became unconscious and wakened up to find himself in a bed in Leith Hospital. It transpired that he fallen down in the street and had been picked up by two young men and conveyed to the hospital.

He was not hurt and states that he is quite sure that he didn't bite his tongue. His stay in hospital was comparatively brief and he was allowed out in three days. The old man, he seems much older than his years, is hard of hearing and not very intelligent and it was rather difficult to get much sense out of him.

Apparently he had not returned to his work after this occurrence but had merely lead a quiet and not very strenuous life.

On the seventh of March he was walking along Rose street when his mind becomes a blank till he found himself in a bed in the R.I.E.

On both these occasions he was merely walking quietly along the road with no particular worry troubling him.

He knows of no reason why he should have become unconscious. He has felt perfectly well since his admission to hospital.

For the past year or so he has noticed that on looking up a height he has become 'dizzy' and that he had to quickly look down to prevent himself from falling.
Previous Health.

As child had measles and scarlet fever. But apparently he had never a days illness in his adult life until three years ago when in connection with his work he slipped and fell off a ladder. He fractured three ribs and received a hard 'bang' on the head. This accident did not lay him up for long, in fact he didn't take to his bed at all.

Patient has always been a good trencherman and confesses to a partiality for plenty meat.

He has never suffered from headaches. It is only on the rarest occasions that he has to rise at night to pass his water.

The patient did not serve with any force in the Great War.

Personal History.

Up to the time of his first attack he was a very heavy smoker and used to smoke about four ounces of tobacco a week. Since that time under the advice of his doctor he has cut it down to about two ounces a week.

A moderate drinker of beer and ale.

No history of any venereal infection.

Family History.

Patient is a bachelor.

Both parents died at the age of 49, he does not what they died of.

Five brothers alive and well.
Examination.
When the patient was brought into the outpatient department he was recovering consciousness and by the time he had arrived at the ward he had regained it completely. A brief examination at this time showed no obvious cause for loss of consciousness. All reflexes were present and normal; he had not hurt himself or bitten his tongue, and rapidly made a recovery.

Alimentary System.
The tongue was moist and clean. His lips were somewhat cyanosed but this was not observed elsewhere. There were no teeth in the upper jaw but the few teeth in the lower jaw were very septic.
The abdomen moved moderately on respiration. On palpation nothing abnormal could be elicited. The liver and spleen were not enlarged.

Nervous System.
The pupils reacted well to light and to accommodation. They were equal in size and quite regular. His eyesight is good for an old man and he can read newspaper print without any difficulty. On ophthalmoscopy the only thing to note was that the arteries where they crossed the veins tended to compress them. The veins were slightly more tortuous than normal.
Patient complains that he has become somewhat deaf since his admission to hospital. With a speculum one can see that the cause of this is due to much wax in the meati.
Routine examination of the limbs showed no impairment in power, no loss of sensation, no spasticity. The knee and ankle jerks are present but reinforcement is necessary to obtain them. The abdominal reflex is present. The Babinski sign was negative.
Genito-Urinary system.
Patient has never any trouble with his water. He never has to rise at night to pass it. The twenty-four specimen of urine is of average amount, is pale in colour and acid in reaction, it shows no abnormality whatever.

Respiratory System.
On inspection the chest showed the typical appearance of an emphysematous condition. The antero-posterior diameter tended to become the same as the side to side diameter. Movement was poor. There is definite decrease in the vocal fremitus. On auscultation the breath sounds were found to be weak and expiration to be prolonged. There were numerous sibilant rhonchi to be heard but no crepitations. Vocal resonance was impaired. The respirations were increased. The lung resonance was markedly increased.

Cardiovascular system.
On inspection the apex beat was just visible about six inches from the midline in the sixth interspace on the left.
Palpation confirmed this and showed a heaving and diffuse character of the apex beat.
Percussion was not satisfactory as the condition of the chest wall and the hyperresonant lung precluded an accurate percussion of the heart boundaries.
Auscultation. In the mitral area the first sound was loud and booming, in the aortic area the second sound was short and sharp and in the pulmonary area the second sound was reduplicated. The sounds were closed in all areas.
Very infrequently there was a premature contraction of the heart which was followed by a long pause.
The pulse.
The upstroke was strong and slow, the pulse was well maintained and the downstroke was also slow. The rate of the pulse varied a little from day to day but was usually in the region of 70-80. The blood pressure also varied but the average reading was 180/126. Rhythm was mainly regular.

The arteries. Both the radial and the brachial arteries were thick and tortuous. The arteria dorsalis pedis was also thick and tortuous. The superficial temporal artery could be easily seen and was very tortuous.

Laboratory tests.

Wassermann negative.

Urea Nitrogen. 11 mgm.%
Creatinine  2 mgm. %

Hypertension, arteriosclerosis, and hyperleak.

Laboratory tests.

Wassermann negative.

Urea Nitrogen. 11 mgm.%
Creatinine  2 mgm. %

First. The stage when the blood pressure is raised but there is no sign of cardiovascular involvement.

Second. This high pressure has brought on changes in the vessels and the heart.

Third. The cardiovascular system fails and gives rise to diverse but easily explained symptoms.

The first stage.

It is not the least improbable that there are many factors which may either together or singly raise the blood pressure. There is thus no need to subscribe to one and one only theory as to the cause of the raised pressure.

Professor Barger has extracted substances from meat which had a very marked pressor effect. Others have shown that certain substances which can be obtained from meats when these undergo putrefaction are absorbed but in a healthy person are excreted. In people with high pressure it was
Clifford Allbutt was the first to show that high blood pressure can arise without any need for chronic renal disease to be present. That high blood pressure is a constant in certain forms of chronic nephritis is undisputed but it is thought that it is rather the kidney disease which necessitates a higher pressure than the heightened pressure causing the renal disease which is the main process.

We will discuss here rather that type of heightened pressure which is primary in itself and which eventually gives rise to many symptoms. It has been called by a variety of names and in a very confusing manner is referred to as essential hypertension, arteriosclerosis, and hyperpiesis. The disease is spread over many years and has been divided into three stages.

First. The stage when the blood pressure is raised but there is no sign of cardiovascular involvement.

Second. This high pressure has brought on changes in the vessels and the heart.

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The First stage.

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Professor Barger has extracted substances from meat which had a very marked pressor effect. Others have shown that certain substances which can be obtained from meats when these undergo putrefaction are absorbed but in a healthy person are excreted. In people with high pressure it was
that these toxic amines were not excreted. They had a very marked pressor effect when injected. These facts are in agreement with clinical observation. It is fairly common to find a person who leads a sedentary life and who eats too much may develop high pressure.

The cause has been sought amongst the endocrines, it having been noticed that hyperpiesa often comes on in women about the time of the menopause. Also it is frequently stated that the adrenal contains more cholesterol than usual. Aschoff bases his theory on the fact that there is thus more cholesterol in the circulation, this is imbibed by the intimal cells which degenerate and by causing arterial disease heightens the blood pressure. He points out that during the War when the amount of lipoid material available in food was very limited the incidence of hyperpiesa in Germany at least was greatly decreased. Coming from such an authority as Aschoff one has to pay some attention to this theory but it entirely fails to explain the cause of the increase in the circulating cholesterol above its normal 140-180 mgm.%. Is it that there is a primary disease of the adrenal which is the ultimate cause of hyperpiesis?

Obesity and thyreoid have been indicted as factors in high blood pressure.

Occasionally it has been noted that the removal of septic teeth or of unhealthy tonsils have resulted in a lowering in blood pressure and thus sepsis has been described as a cause.

As in most other diseases diathesis has been called on to explain why some should have hyperpiesis and others none. Osler was the first to point out that some people seem to be born into the world with bad arteries. They serve alright for the early years of life but soon they tire and give out and high pressure results. Osler's statements on 'congenital bad tubing' and similar statements savour very much of the physiologist who when he is trying to explain some difficult chemical change which occurs in a particular organ explains it on some theory
of vital function! For instance I read the following statement in one volume which was dealing with the causation of high pressure 'not only may the material be faulty but there may be an inborn tendency to make faulty use of it'. This is the type of statement which to my mind leads absolutely nowhere. If one argues in that vein surely it would not be stretching the point to say that there is a special inborn disposition to each and every disease that man is heir to.

It is however the case that high pressure is commonly found in hard-working hard living people - the tired business man of the daily press. Worry and responsibility do seem to have an effect in raising the pressure and a synonym for the term high blood pressure is the Statemans disease.

At this early stage of the disease the clinical features will be few. In the routine examination of a man between the ages of forty and fifty the blood pressure will be found to be 200/115 or so, the first sound will be loud and the second heart sound short and snapping but there will be no enlargement of the heart.

Second Stage. The long continued high pressure has its effect on the left ventricle. This chamber of the heart together with the large arteries must increase their muscular tissue to keep up the volume of the blood. They thus hypertrophy - an overwork hypertrophy - and the apex beat can be seen to have increased in force and extent and the tonicity in the larger vessels cause them to be felt very easily. Clinically at this stage the left border of the heart is increased in size, the apex beat has become more diffuse and heaving and the second heart sound is now possibly reduplicated. The patient still feels in good health and carries on his daily work. He may have attacks of headache or giddiness but they do not worry him and the condition passes into the next stage.
The Third Stage. This stage is characterised by the onset of certain changes in the arteries. In the aorta, cerebral and coronary arteries the development of atheroma occurs. Although some say that atheroma and hyperpiesis do not necessarily occur together it is generally believed that atheroma follows on the sustained high pressure. Aschoff believes that it is a primary increased cholesterol content that causes the atheromatous change and then the heightened pressure.

In the internal organs there is a characteristic fibrosis of the arterioles. Again some believe that it is this thickening or spasm of the arterioles that is the cause of the high pressure. Nevertheless in the final stages such a change does occur and is responsible for many of the clinical signs at this time. It occurs in the brain and the kidney where it gives rise to the 'arterio-sclerotic kidney'. It also occurs in the pancreas where it is said to cause the glycosuria of old age and also in the skeletal muscles.

Clinically.

In the brain many conditions are possible. The patient may be carried off by a massive haemorrhage in the region of the corpus striatum by the rupture of the lenticulo-striate artery. He may have transient attacks of paralysis causing impairment in the lower limbs and in speech which is said to be due to minute haemorrhages. He may have peculiar attacks in which he loses consciousness but is still capable of carrying on his work - this is the so-called cerebral angiospasm. The classical example is that of the minister who gives his sermon with his brain in an attack of angiospasm and on recovering imagines that he has not yet delivered it and treats his congregation to another and identically similar sermon.

Headache, giddiness and tinnitus are all also met with. If the patient escapes a cerebral death his heart may begin to fail and
an intense heart failure sets in with very marked oedema. Angina pectoris may occur as may the arrhythmias.
The effect on the kidney is to cause obliteration of kidney units. These disappear one by one till the kidney reserve disappears and definite renal insufficiency appears. It is held that a patient with high blood pressure does not die of renal disease of vascular origin but that a superimposed inflammatory condition may lead to the uraemia which is a fairly common way for the arterio-sclerotic to die.
In the skeletal musculature a peculiar condition may arise called intermittent claudication. The patient finds that on walking a certain distance he is suddenly seized by an intense pain in the muscles of the calf of either both or one leg. He has to wait some little time till the pain passes off and till he can thus continue his walk. I have seen two cases of hyperpiesis who presented themselves with this condition as their only symptom.
The whole subject is singularly confused and it is with reluctance that one chose a case of raised blood pressure to discuss. The importance of its early recognition in the first stage is of great importance with regard to its treatment, and it is because one has been impressed with the fact that a great deal can be done for a patient with high blood pressure that this case was chosen.
Possibly the most important question to decide is whether the raised blood pressure precedes the arterial changes or whether the opposite is the case. I have not read two single articles which are in complete agreement on this point. Most pathologists describe an atheromatous change affecting the aorta, cerebrals and the coronaries. Also a medial degeneration affecting the smaller arteries such as the radial and the brachial which they state is the cause of the pipe stem arteries felt by the
clinician. And lastly but not by any means unanimously a type of degeneration called diffuse hyperplastic sclerosis. This change may affect all the parenchymatous organs but is especially seen in the kidney. It consists in a subendothelial thickening of the intima which may lead to an eventual obliteration of the vessel. It is this change that is always associated with raised blood pressure but again the most recent publications will not commit themselves on the point of which comes first—the increased pressure or the arterial changes.

I hesitate to discuss the relationship of kidney changes to increased blood pressure. Can you have an entirely healthy kidney as is evidenced by excretion tests with a raised blood pressure or are all cases of increased pressure the first effects of a condition known as chronic interstitial nephritis. I will discuss this point further in a case of uraemia.

Turning now to the case of Crombie, what stage of increased pressure is he at? Is his disease primarily renal or is it to be found in his arteries? By a consideration of his history we see that he is probably in his early third stage.

1) His superficial arteries are considerably thickened. The arteries in his retina show the typical copper wire appearance and are compressing his veins there.

2) His blood pressure is considerably raised and on admission was about 180/130. One would not expect an aura to be present in every case how-

3) The typical symptoms of giddiness and so forth are and have been present for some considerable time.

4) His left ventricle is considerably enlarged and is showing numerous extrasystoles which some say are of great prognostic importance.

5) An examination of his blood excludes any syphilitic basis and shows-
that at present there is no gross interference with renal function. His urea nitrogen is 11mgm% and his creatinine is also normal 2mgm%. These combined with the fact that his excretion of urea was very satisfactory being over 1.5% in the first specimen and over 2% in the second specimen in McLean's urea excretion test show that to all intents and purposes his kidneys are as healthy as one could wish.

6) His periods of unconsciousness are very difficult to explain. A most careful physical examination of his nervous system fails to reveal any organic change present. Our history as to his precise condition in his period of unconsciousness is not good. Nobody can give us any information as to his exact condition when it occurred and as we have previously mentioned he was recovering consciousness when he came into the hospital. In view of the physical findings I suppose the most likely cause for his loss of consciousness was that of cerebral angiospasm. A sudden spasm of his arteries, possibly those small vessels which are affected in the diffuse hyperplastic sclerosis we have mentioned caused the unconsciousness to occur. This would also account for the complete absence of physical signs after he had recovered consciousness. I suppose that with a history of an injury to the head as Crombie had a year or two ago one should consider the possibility of traumatic epilepsy. However the old man states that he felt nothing wrong with him before he lost consciousness, no tinglings or peculiar sensations in his fingers or toes. One would not expect an aura to be present in every case however. Taking the case as a whole however I think that the probability is against that of an epilepsy.

Treatment.

Let us first consider how we would have treated Crombie if we had seen him some years ago. Suppose we had examined him for some insurance pur-
ose, or had been examining him for entrance into one of the services. We found that his blood pressure was raised, not much, but sufficiently raised especially in the diastolic pressure for us to be able to know that here was a man who was a candidate for its more serious effects.

Our first duty would be to go over his whole mode of life. What is his work? Is it to strenuous? Would it be advisable for him to give up his hard manual labouring and take up some job that was easier? This is easy to say but in the majority of cases is much more difficult to carry out. In a better class patient such as a professional man one would be able more easily to control his life. The aim is to control his habits and to pitch his rate of living at a much lower level than he has been accustomed to. His work then must be not too strenuous and must be regular. His habits of eating and sleeping must be regular. Eight hour sleep should be aimed at. His meals must be taken at regular hours and he must avoid a heavy meal at night. It is useful especially in female patients at the menopause to teach them to relax for an hour or so after their meals. One should look for any points of focal sepsis bearing in mind that in some cases a blood pressure has returned to normal after the removal of bad teeth or the draining of a septic sinus.

Exercise must be taken in moderation and no patient should be prohibited from his occasional round of golf. One must not allow him to become too sluggish and fat as it is well known that obesity often leads to an increased blood pressure. In more severe cases especially in older people massage may often be employed with some degree of success.

Turning now to his diet. Again it is useless here to impose all sorts of fancy restrictions. Naturally if the patient is obese one would treat by careful dieting cutting the diet down to about 1000C per day till the requisite level had been reached. It has been suggested
that the alkaline ash diets now used in hydraemic types of nephritis may be of service. Allen's salt free diet has now been entirely given up. Fluids are best given in large quantities rather than with-held. A daily action of the bowels must be ensured either naturally or by a nightly cascara evacuant or a morning saline.

Thus in the early stages we would confine ourselves to regulating the patient's life as regards work and worries. We would advise him on his food, telling him to eat an ordinary mixed diet in moderate quantities and avoiding to much meats and condiments. We would encourage him to drink plenty fluids but to rigidly give up alcohol except perhaps for an occasional diluted whisky and also asking him not to smoke too much.

But supposing we had met him in the second stage when the pressure was raised a little further and the apex beat was now passing out into his sixth interspace and he had a few attacks of giddiness etc. He would still be carrying on his work in an active and energetic fashion. We would advise him to go to bed for a week or a fortnight as it is surprising how a short time in bed will bring down the pressure. We would then carry out all the investigations and treatment detailed above and would probably consider the administration of a drug to see if it was possible to lower the pressure in that way. There are a great number of drugs available and it is difficult to know what one is to be taken. I will give a brief list of the more commonly used drugs.

1) Potassium Iodide is an old and proved favourite.

2) 'Rhodan' - sodium sulphocyanate. This drug was first introduced by Pauli in 1904 but was later forgotten. Westphal in 1924 reintroduced it but gave it very small doses. grii thrice daily is said to have a good depressor effect.

3) The nitrite series are used but of course their action is fairly
rapid. Erythrol tetranitrate, and mannitol hexanitrate have more prolon-
ged action. Bismuth subnitrate was introduced in the belief that the
flora of the large gut would reduce it to the nitrite and so a more pro-
longed action would be obtained.

4) Depressants and sedatives are also used. grilii-v of potassium bromide
is a favourite remedy. Luminal in $\frac{1}{2}$-i grain doses is sometimes used but
its effect passes off after three weeks. Theominal is a trade prepara-
tion which contains theobromine andluminal and has obtained some popular-
ity.

5) The caffeine and calcium series of drugs are occasionally used.

6) Acetyl-choline and its derivatives are used. They are especially indi-
cated in cases of intermittent claudication and I have seen some very
good results follow its use.

7) Organo-therapy has of course been tried with moderate success.

In the last stages as Crombie is in now all one can do is
to treat symptoms. He will now have to stay in bed for the greater part
of the day. Potassium iodide and bromide should be exhibited. The rest
of the treatment will be on lines indicated above.

Prognosis.

As his kidney is at present functioning quite well we fear no renal
cause of death.

His heart shows the presence of many extra-systoles and as I have said
this is taken by some as an indication of heart failure.

The immediate prognosis is always precarious as a cerebral haemorrhage
might carry him off at any moment. His heart may fail at any time now
and accordingly his expectation of life is not long. With careful atten-
tion he may last a year or more.

Referred from Dr. McAdoo, Portobello.

Admitted 28th. April 1931.

Complaint. Weakness at the back of the knee, Trailing of the left toes, both of three months duration. Bad eyesight, of three months duration.

History of the present illness.

For the past year or so he has been working at the Taller water works. He lives in a hut with the other labourers and has to walk about two miles to the actual place of work. He comes home at the week ends to his home in Edinburgh.

About six months ago he noticed that he seemed to be wearing the toes of his boots away very rapidly but did not take particular notice of this. Soon however he found that his legs were becoming weaker and that he was definitely trailing the toes of his left foot. On arising in the morning he felt quite weak. This state of affairs only began to worry him seriously about three months ago. He went to his doctor who gave him 'something to rub the legs with'. This unfortunately did not do him much good At this time also he found that his eyesight was getting worse. Two years ago he had got glasses for reading but he now found that even when he wore these that he could not see far.

His family persuaded him to go to an optician and get his glasses changed which he did. However this optician 'looked into his eye with something' and told him that it was no use him getting glasses and said he should go and see Dr. Traquair.

About three days before admission, he had been working up to this time, he went to the Eye department at the Infirmary and was seen by Dr. Traquair. As a result of this examination a letter was sent to Dr. McAdoo who referred him to ward 23.
Previous Health and History.
Patient went to Vancouver in 1907 and took part in the Klondyke gold rush in 1909. At this time he contracted pleurisy and was in hospital for a week or two. During the course of treatment at this hospital he received two burns over the stomach with 'some electrical thing!'. He returned back to Scotland in 1911. Since that time he has been in regular employment. During the war he served by working in a Carlisle mill.

He has had no serious illness other than the pleurisy. He is not sure what illnesses he had as a child but thinks that he had scarlet fever.

Personal Habits.
In his younger and rougher days he was a hard drinker. Since he has been married he only drinks at the week ends and then only partakes of beer. He smokes about an ounce of warlock tobacco a week but formerly about three and a half ounces.

He has always enjoyed his food and has partaken generously of a mixed diet. Since he has been out at the water works he has been eating only tea and bread etc. with hardly any meat.

Family History.
Two living children. Both healthy. One child died of Diphtheria.
Wife perfectly healthy. Has had no miscarriages.
His mother died of old age at 76. His father died at the age of 46.
EXAMINATION.

Patient is a spare man just turning grey. His memory for the lesser events of life is not good. His speech is somewhat slurring. For an out of door blacksmith his complexion is rather pale. He has no marked anaemia however.

Gastro-intestinal system.
The tongue is only moderately clean. His upper teeth are all false but the few remaining teeth in the lower jaw are very septic.
The examination of the abdomen is entirely negative. The liver and spleen are not enlarged. There is nothing palpable.

Nervous System.
The pupils respond to light and accommodation. They are regular and of equal size. His sight is markedly impaired especially in the left eye. He cannot say how many fingers one is holding up at a distance of five feet, but at a distance of four feet he is able to with his right eye. His peripheral vision is also impaired. He cannot read newspaper print and can only, with some difficulty, read the headlines.

On dilating the pupils with homatropine the following points are made out.
1) The arteries have a definite silver wire appearance, i.e. the whole thickness can be easily seen
2) The veins are very tortuous and dip in and out of the vision.
3) There are numerous small haemorrhages many of the typical flame shape.
4) There are also numerous white glistening areas of 'albuminuric retinitis.'
The movements of the eyeball are entirely unimpaired.
The strength and the sensation of the upper limbs are normal. One is struck however with the fact that although he is a blacksmith his mus-
cular development is not very great. The lower limbs are somewhat wasted. There is no impairment of either motor or sensory power. The knee and ankle jerks are present and are not exaggerated. The Babinski response is negative. At one time I was able to obtain ankle clonus on the left side but I have not been able to repeat this.

Genito-urinary system.
Recently he has had to rise at night to pass water. Never has any pain or discomfort on passing urine.

The Urine.
Amount. 1500c.c.
Acid in reaction.
Of low specific gravity.
Albumen is ++
No sugar etc.
On centrifuging many granular casts are to be found.

The respiratory system.
On inspection the chest does not move much on respiration. Palpation confirms this and shows no decrease in vocal resonance. On percussion there is increased resonance over the apices and the borders. The breath sounds are vesicular and expiration is prolonged. Some crepitations are to be heard over the bases of the lungs.
The most noticeable thing however is the fact that there is a definite periodicity in his breathing. He takes about sixteen respirations at a time. They rise in a crescendo fashion and then die away again. There is then a period of apnoea of forty to fifty seconds before the initial respirations start again. Patient is entirely unconscious of this, he has never felt breathless at all.

The cardiovascular system.
The apex beat lies in the sixth interspace 5½" from the midline. In
character it is diffuse and heaving. The upper border lies behind the third rib and the right border about $\frac{3}{4}$" to the right of the sternum. In the mitral area the first sound is prolonged and booming an character In the aortic area the second sound is short and sharp and is reduplicat ed in the pulmonary area.

The pulse. The rate is eighty per minute. It is regular in rate and rhythm. The upstroke is sudden, the pulse is well sustained and the downstroke is slow.

The arteries.
The radial and brachial, also the dorsalis pedis arteries are all very tortuous and thickened.

For the three weeks after his admission he was put on a light diet consisting of milk, stewed fruit, toast, potatoes, vegetables, a little fruit and brown and butter. As she always duty for his meals and was I'm afraid a little character. We have been in hospital at the small amount of his appetite. He was also given two grains of potassium iodide daily and the morning before every motion of his bowels by giving him a little of fish bones.

His condition remained the same to the next three weeks, he came down to a clinical lecture one day and was upstairs at the experience although he could not see all the operations he was able to present. He was allowed up daily for the three weeks of which he greatly appreciated as he was able to see some scenes in the ward. He had "pneumonia on one side of the lung in the lungs" on the other side conversation which in the end was not successfully aspirating.

A urea concentration test done at this period showed that he was able to concentrate urea to the extent of 1.5% in some samples. The urine still
Course of patient while in hospital.

Patient was admitted on the 28th. April and his condition on examination has already been detailed. His blood pressure was fairly constant in the region of 220/130. His pulse was about 70-80 and his temperature 97°.

Waldie was a likeable fellow and was always very ready to let one examine him in any way. He liked talking about his old experiences in Canada but was often somewhat difficult to understand as his speech was a trifle slurring and thick. His one grudge in life was that he was unable to read because of the state of his eyes and I must say I felt rather miserable when he asked me each day if I thought they would soon be better. Naturally one told him it would be a matter of time and that he would just have to be patient.

For the three weeks after his admission he was put on a light diet consisting of milk, stewed fruit, chicken, potatoes, vegetables, a little fruit and bread and butter. He was always ready for his meals and was I'm afraid a trifle chagrined when he first came into hospital at the small amount of his repast! He was also given ten grains of potassium iodide daily and the nursing staff ensured a daily action of his bowels by giving him jalap at short intervals.

His condition remained the same for these three weeks, he came down to a clinical lecture one day and was highly amused at the experience although he could not see all the gentlemen he was told were present. He was allowed up daily for intervals of about one hour which he greatly appreciated as he was able to talk to some other men in the ward. (He had a pneumonia on one side of him and a gangrene of the lung on the other so conversation while in bed was not particularly inspiring.) A urea concentration test done at this period showed that he was able to concentrate urea to the extent of L. 6% in each specimen. The urine still
On the eighteenth of May about three weeks after admission he was allowed up for two hours. When I had seen him in the morning he had been quite his usual self and had been very pleased as he could see the roofs of the opposite medical pavilion for the first time. When I arrived up at the ward on the nineteenth I was told that towards the evening of the previous day he had become somewhat queer and was starting to get noisy and try and get out of bed against sister's orders. As he was getting rather unmanageable he was given a sixth of morphine and a fiftieth of atropine at midnight. This tided him over the night but when I saw him he was very confused and was trying to get out of bed. It was decided to lumbar puncture him and about twenty c.c.s were drawn off. He was also venaected twice on 19th and a total of one and a half pints removed.

He was slightly improved by this. His condition was now quite changed. He was irritable and somewhat noisy, his pulse was slowly beginning to creep up as was his temperature. It was noticed that his hands and fingers were showing twitchings especially when he was under the influence of opiates. Although he was now passing water incontinently it was only with great difficulty that a movement of the bowels was obtained.

At times he was remarkably alive to the situation. For instance on the 24th May when I was taking off some blood he rolled up the sleeve of his sleeping jacket for me, and said that he felt much better. He recognised his wife at all times when she came up to be at his bedside.

As soon as his condition had become pronounced he was given ammonium chloride in fifteen grain doses thrice daily and was given plenty fluids, which he was always ready for.

Beyond this nothing much else could be done. Repeated lumbar puncture was not done as the fluid was not under tension at all. He was kept well under opiates and latterly one hundredth grain doses of hyoscine were given.
This state of affairs lasted a surprisingly long time for a whole ten
days in fact.
Towards the end his condition was pitiable, he was entirely incontinent
and unconscious he showed marked Cheyne-Stokes breathing and many twitch-
ing movements of the hands were present. By the 29th May the pulse had
risen to 120 and the temperature to 101°. The blood pressure began to
fall and at the end was 130/110. He died on the last day of the month
after having put up a gallant fight against the inexorable onslaught of
the fell uraemia for almost a fortnight. How often it is that uraemia
claims as her victims those who have worked and played hard at a time
when they might yet enjoy a few years of rest and pleasure before they
are called away.

The mild and transient nervous phenomena which he exhib-
ted were most probably due to some small cortical haemorrhages or possibly
due to some degree of arterio-capillary fibrosis. These changes were not
marked and while in hospital all nervous phenomena disappeared.

I would like to take up two questions in this discussion, firstly the
production of chronic interstitial nephritis and secondly the question
of uraemia.

Chronic interstitial nephritis is an end result of two distinct disease
processes, it may be primarily renal or may be primarily due to vascular
disease.

A modern view which is gaining ground among clinicians is that Bright's
disease in all its forms may be looked on as a consecutive nephritis.
The disease naturally leads on from its first form of acute inflammation
to its end result of chronic interstitial nephritis. Naturally one imme-
diately replies to such a hypothesis that you can produce in a very short
time several cases of chronic nephritis which have never suffered from
Discussion.
The diagnosis in this case was comparatively simple. As soon as attention had been drawn to the eyes and these had been examined with the ophthalmoscope the diagnosis was almost made. An examination of the urine revealed the presence of numerous casts and much albumen. The raised blood pressure and the hypertrophied left ventricle helped in the diagnosis. A McLean's urea concentration showed that the urine was only concentrated 1.6% in each specimen. The examination of the blood in the early weeks showed no marked changes. The presence of albumen although not usually accepted as a sign in chronic interstitial nephritis often occurs and may be due to an acute or a chronic degeneration in the kidney tubules.

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The disease naturally leads on from its first form of acute inflammation to its end result of chronic interstitial nephritis. Naturally one immediately replies to such a hypothesis that you can produce in a very short time several cases of chronic nephritis which have never suffered from
an acute attack. But taking an analogy, the appendix is often removed in a state of fibrosis which can only be the result of previous acute infection yet the patient has never complained of an acute attack of pain in the right iliac fossa. A duodenal ulcer may actually perforate before the patient realises that anything is amiss with his alimentary canal. Naturally absolute proof is difficult but there is a classical case of a female who was observed over a period of twenty eight years. She at first developed an acute nephritis and the albumen never entirely disappeared for many years. During this time she had typical hyaemic nephritis with casts, albumen oedema and anaemia. After some years this passed off and she began to pass large quantities of water of low specific gravity, her blood pressure began to rise and she died in uraemia. This is in example of what is believed to happen in most cases of Brights disease.

One believes then that nephritis starts as an acute process but that this may not be observed in all cases. The next stage is that which one calls chronic parenchymatous or hyaemic nephritis. The process may stop at the stage of acute nephritis but does not stop once the second stage has resulted. The majority of patients showing this stage markedly do not usually pass to the third stage of this consecutive nephritis namely chronic interstitial nephritis. Thus we see that most nephritics who reach the final stage will not have shown the second stage to any marked degree. Naturally one does not believe that the cause of the original disease continues right through the whole course of the disease. A rheumatic infection of the heart affects the heart valves but it is the end result of the healing processes that cause the majority of valvular lesions which lead to so much disability.

The etiology of this type of nephritis is obscure and it arises under
many conditions. Possibly the commonest cause of acute nephritis is a tonsillitis or a sore throat. That form arising during the end of the second week in scarlet fever is also common. Many other factors are possible but it is evident that the streptococcus or its toxins appear to be largest single factor in the causation of nephritis. This then is the type of nephritis that is due to a primary renal condition. The other type of chronic nephritis is that which is due to a primary arterial condition. In a previous case we showed how in the third stage of hypertension there were many vascular changes in the various organs and that the vascular change was one of a diffuse hyperplastic sclerosis. The kidney is the commonest organ to be affected by this change and the end result is known to pathologists as the arterio-sclerotic kidney but to clinicians is more or less a variety of chronic interstitial nephritis. It is possible to describe different forms of this arterio-sclerotic kidney but that really serves no useful clinical purpose. A chronic interstitial nephritis is due than to either the end result of a progressive and consecutive nephritis or it may be found in the course of an ill understood and badly defined condition of raised blood pressure with arterial changes. Which type had Waldie? Was his renal condition primarily renal or was it a condition occurring in the course of hyperpiesa? The fact that his blood pressure was raised and his left ventricle was enlarged does not help us at all as they are affected in both types of the disease. His hard life both as a prospector and as a blacksmith would naturally expose him to conditions when he might be attacked by an acute nephritis but on the other hand we have no proof that hypertension has not existed for many years. The fact that he had albumen in his urine is possibly of
It is well known that mixed types of nephritis occur, and in our conception of a consecutive nephritis this is quite a possibility. It is obvious that the passage of the condition from one stage to another need not be quite clean cut and that mixed forms are to be expected. On the other hand a more or less acute infection or affection of the remaining functioning kidney tissue in an arteriosclerotic kidney is quite possible. We cannot say therefore on our evidence clinically whether or not the chronic stage is that form due to a consecutive nephritis or to primary renal disease and the microscope is the only solution to our question.

Let us now turn to a brief discussion on uraemia. The condition of uraemia may occur in practically all of the more serious affections of the kidney, and is usually called a toxaemia (Osler). The condition is readily recognised clinically but is pathology and its chemistry are ill understood. Clinically uraemia is most often ushered in by coma of slow or rapid onset. This coma is often preceded by headache and a mild delirium and this is exactly how Waldie went into uraemia. Later vomiting may occur but more commonly muscular twitchings are seen especially in the extensor muscles of the wrist. The breathing is typically Cheyne-Stokes in type and has a peculiar hissing character towards the end. Diarrhoea and intractable vomiting are also symptoms. Occasional nervous symptoms are observed especially amaurosis, hemiplegias and monoplegias. Convulsions are common towards the end but we did not observe this in the case we are studying. A pseudo-uraemia occurs in acute nephritis but true uraemia is always fatal. An attack may be warded off for some time but sooner or later it comes back and the patient dies. It is as certain to kill a patient as an inoperable carcinoma.
Before turning to the chemical problems of uraemia let us first say a few words on tests for renal function. In routine clinical work it is usual to test for renal function by its ability to excrete urea. About 15g. of urea are given suitable diluted and flavoured. The urine passed at the end of one hour and at the second hour are collected and the percentage of urea in each specimen estimated in a ureometer. In young healthy people the concentration of urea should be above 2% in the second specimen and anything below 2% in this specimen is evidence of damaged kidney function. Another very simple test is to give the patient a good meal and to compare the specific gravity of the urine before and after the meal. In health the urine after the meal should be more concentrated than before it but in kidney disease the urine remains of a low specific gravity both before and after. The blood is tested by the biochemist for different substances and different wards choose different substances to act as their criterion. It may be the non-protein nitrogen, the urea or the urea nitrogen. The creatinine is always asked for and in many cases the carbon dioxide combining power. These substances show characteristic changes in nephritis. Probably the first function to be lost is that of the kidney to concentrate the urine. After this it loses its ability to concentrate urea. Shortly after the blood will show evidences of nitrogen retention by the fact that the non-protein nitrogen and the urea nitrogen are increased. Still later the blood creatinine begins to rise and later in the end stage the carbon dioxide combining power falls. I can find nowhere a satisfactory discussion of the problems of uraemia as a whole. Most articles seem to pin their faith on one theory alone and it is difficult to estimate how correct they are. It is now proven that urea retention is not the cause of uraemia although...
the estimation of blood urea is very helpful in estimating the probability of uraemia in any given case. The facts that exclude the urea theory are as follows:

1) Urea in large doses does not cause uraemia experimentally.
2) Certain conditions such as cystic kidney may exist with marked nitrogenous retention for years without any uraemia occurring.
3) Uraemia has been observed to occur with no nitrogen retention.

Many other chemical substances have been invoked as the cause of uraemia but an argument against them all is that they do not show changes in all cases of uraemia. Creatinine has been suggested but a person can die of uraemia without a very marked increase above the normal.

Calcium is a substance that deserves more attention. Unfortunately only one estimation was done on Waldie and that showed a normal amount. We might have had changes later on however. It is the commonest finding that the calcium may be lowered to 6mg% or less in uraemia and it is natural that the convulsive phenomena of this condition should be compared to those of parathyreoid tetany and spasmodiphilia etc. Unfortunately these latter conditions show a condition of alkalosis and not acidosis which is usual in uraemia.

By an acidosis we mean a lowering of the normal alkali reserve of the blood and we know that this can occur in either of three ways. There may be an overproduction of acid in the body which takes up most of the available base with a consequent condition of acidosis. Acidosis may occur as the result of excessive loss of base as is typically in the diarrhoea of children. Lastly acidosis may result by a failure of the kidneys to eliminate acid substances either as acid radicles or as true acids. The normal carbon dioxide combining power of the blood lies between 53-80 vols%.

Anything below 53 is called acidosis and anything above 80 is called an alkalosis.
James Waldie, Died 31st May.

- Urea nitrogen in mgms%
- Creatinine in mgms%
- CO2 combining power in vols.%
It is thus stated that in chronic interstitial nephritis the carbon di-
oxide combing power is always lowered below 53vols%. That is uraemia is always associated with an acidosis. Although many authorities believe that uraemia and acidosis are one and the same thing this is not alto-
gether proved. The symptoms of an acidosis are peculiar deep respiration, a drowsiness deepening into coma, headache, vomiting and occasionally some nervous disturbances. A condition not unlike uraemia.

In the case of Waldie we have almost proof that a state of alkalosis can exist with clinical uraemia, in fact we have both an alkalosis and an acidosis during the course of the uraemia. On the nineteenth May when uraemic symptoms appeared the carbon dioxide in combing power showed that there was an alkalosis. Being very surprised at the result I took the trouble to see the biochemists. I told them that the patient was in uraemia with Cheyne-Stoke respirations, coma and twitchings. They replied equally firmly that he could not possibly have uraemia and that I must go back and change my diagnosis. My diagnosis an incidentally that of the physicians of the ward was quite unshaken, here was as definite a case of uraemia as ever there was. However a glance at the chart provided will show that the alkalosis gradually was replaced by an acidosis although a temporary rise may have been caused by the administration of ammonium chloride and he eventually died in coma with acidosis. He received no chloride till the CO2 was 70vols%.

I have been at some pains to enquire into this and it strikes me that this case definitely disproves the theory that uraemia is merely a manifesta-
tion of acidosis. I know of no other case cited in which this has occurred. Possibly this case might be worth mention in higher spheres if uraemia with alkalosis has not been recorded before.

Uraemia then still is a mystery to us and I must say the more I read
the condition the more convinced I am that the real solution has not yet been reached.
A further glance at the chart will show how the urea nitrogen and the creatinine slowly rose till death occurred.
The treatment of the condition is practically hopeless. The bowels must be kept continually open and cleared either with high colonic lavage or by drastic purgatives. Venesection must be repeated if necessary and the cerebro-spinal fluid should be frequently drawn off if there is any suspicion of oedema. Large quantities of morphia and hyoscine may be necessary to control the nervous symptoms. The treatment is in most cases entirely ineffective.
The man who elucidates the problem of uraemia and who evolves some method of keeping this terrible manner of death at bay will have performed not the least of services to mankind.