Five Medical Cases for the Nightingale Prize.
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West Bank,
Lochhead.

I have to thank Dr. E. Matthew for permission to make use of four of these cases, and Prof. E. Bramwell for the fifth—namely, the second diabetic case.
Case 1.

L.S. Male. Aet. 45 years.

Occupation - Law-Clerk.

Admitted - 9:32.

Discharged -

Complaint: 1. Shortness of breath on exertion - 9 months.
           2. Swelling of feet and ankles - 3 months.
           3. Palpitation - 2 years.

History: For two years patient has had intermittent attacks of palpitation, about every two months. They seem to be brought on by over-exertion, but passed off when he rested. About nine months ago he began to be breathless on exertion. If he went up-stairs too quickly he got very breathless. This has got progressively worse, so that now if he goes too quickly along the road he becomes breathless. Lately he has been taking things very slowly.

For the past three months he has had swelling of the feet and ankles towards the end of the day, but passing off after a night's rest in bed. This also has got progressively worse, and for the past two or three weeks his feet have been purplish in colour.

He has also had a hard dry cough for the past two years, worse during the winter.

Previous Health: He has had excellent health up to the present, except for influenzal attacks now and again. When he was about six years old he remembers being able to peel the skin off his hands, but did not have a sore throat.

Habits: Was a very heavy smoker for years, averaging twenty to thirty cigarettes per day. He drank a good deal of beer up to the New Year, but no spirits. Since then he has not taken any alcohol.

Family History: Is not married. Father died of senile decay, aet. 80. Mother died of acute phthisis with peritonitis, aet about 46.

Home Conditions: Lives in lodgings. Room is cold and damp. The food is not too good.

EXAMINATION.
When patient was admitted he was very cyanosed, his face having a peculiar grey appearance, and his lips, ears and fingers being of a blue colour. After being a day in bed he lost the grey colour, his complexion becoming a pale pasty colour. He has a worried, anxious expression. He is quite intelligent and answers questions readily. He is quite well developed.

**Cardio-Vascular System:**

- **Pulse**: Is regular in time and force, 80/min., expansion is good. Wall is not palpable. Blood pressure - 122/85.
- **Heart**: Apex beat is in 5th., outside the mid-clavicular.
  - Right border is at right sternal margin.
  - Upper border is at 3rd. rib.
  - Sounds are closed in all areas. First sound in the mitral area is somewhat shortened.

**Respiratory System:**

- **Subjective symptoms as in history.**
- **The chest is barrel-shaped. On deep inspiration it tends to be drawn up bodily instead of expanding in an anterior-posterior direction. Expansion is therefore not good, but is equal on both sides.**
- **There is bulging of the apices.**
- **Vocal fremitus is present and equal, but decreased.**
- **The percussion note is hyper-resonant over both upper lobes.**
- **The areas of superficial cardiac dulness and hepatic dulness are decreased. There is absolute dulness at the back on the right side up to the fifth rib, and on the right side up to the 8th. rib.**
- **The breath-sounds are vesicular. There is prolongation of expiration on both sides, most marked over the right lung. Vocal resonance is present over both upper lobes, but is diminished in intensity. The breath sounds are diminished below the fifth rib and absent below the 7th rib on the right side. On the left side they are diminished below the eighth rib and are accompanied by crepitations on both inspiration and expiration.**

**Alimentary System:**

- **He has had acid eructations with heart-burn and flatulence**
for eighteen months, relieved by taking powders. He is not constipated. Abdomen - Is definitely and generally swollen, with bulging in the flanks. There is no tenderness or rigidity anywhere. There is shifting dulness in the flanks and a fluid thrill is obtained. Liver - is enlarged and palpable below the costal margin. It is somewhat tender. There is marked pitting of the skin at the back on pressure.

Urine System:

He has noticed that his urine has been highly coloured for the past two or three weeks.

Urine - Quantity was 20ozs. on admission, increasing to 40ozs. on 12:3:32. Sp.Gr. 1025, acid in reaction. A trace of albumen present using the salicyl-sulphonic acid test. No other abnormal constituents. Micro. - triple phosphate crystals present.

Nervous System:

All reflexes present and normal.
Pupillary reflexes normal.
No sensory disturbance.

Special Examinations:

Wasserman reaction - negative.

X-ray examination: report stated that there was a right-sided encysted pleural effusion, with a degree of collapse over the lower lobe of right lung.

Bacteriological report on fluid from pleural cavity: Shows a very large number of large cells, of endothelial cell type, with deeply staining nucleus and basophil cytoplasm. There are other cells of a similar type, not staining so deeply. Some of these cells are dividing, while many are in sheets containing two or more cells. There are also some lymphocytes and red blood cells. No tubercle bacilli or other organisms were found, and no growth was obtained on culture.

DIAGNOSIS.

There are two symptoms present in this case, viz. breathlessness on exertion and oedema of the feet and ankles, which together are diagnostic of one condition - heart failure. The other symptoms and signs, palpitations, crepitations at the bases and pleural effusion,
merely serving to confirm this diagnosis. This however is only a p\text{-}oxi-
mate diagnosis, for there are numerous conditions which may cause heart-
failure, and \textit{in} one has to find out what condition is causing it in this case.

Heart-failure may be due to any one of numerous conditions in-
volving either, 1. cardio-vascular, 2. respiratory, or 3. renal systems. The absence of hypertrophy of the left ventricle, the normal blood-pres-
sure, and the absence of urinary signs and symptoms definitely excludes \textit{chronic interstitial nephritis}, i.e. only renal condition which causes congestive cardiac failure. The absence of the hypertrophied left ventricle and high blood-pressure also excludes arterial disease, and there was no sign of the presence of pernicious anaemia. We are thus left to consider a. the heart itself, and b. the respiratory system.

In the heart itself there are two structures to be considered, the myocardium and the endocardium. The endocardium, i.e. the valves, can be definitely excluded in this case as the sounds were closed in all areas. We are thus left with the myocardium, and there can be little a condition of chronic myocarditis existed here, and that this has led directly to the failure of the heart. Chronic myocarditis may be due to a. poisons, either chemical, metabolic, or bacterial in origin, or b. some conditions in the lungs which causes a chronic obstruction to the flow of blood through the pulmonary circulation over a considerable period.

In this case both factors were at work, and we have to decide which is chiefly to blame. He had been a very heavy beer drinker for some considerable time, and alcohol, especially in the form of beer, has a definite toxic effect on the heart muscle, and is a well-known cause of myocarditis. In the respiratory system well-marked signs of emphysema were found, the diagnosis of this condition being based on the barrel-shaped chest, hyper-resonant note all over both lungs, and ves-
icular breath-sounds with prolonged expiration. Emphysema is a classic cause of congestive heart-failure, by causing chronic myocarditis. There can be little doubt that it was a factor here, and in support of this there was the peculiar deep cyanosis which is a feature of emphysema and which was present in this case.

The diagnosis therefore is one of congestive heart-failure, fol-
ollowing a chronic myocarditis, due to a poisoning by alcohol, and b. the effects of emphysema.

With regard to the pleural effusion, the report on the fluid was rather alarming, suggesting as it did the possibility of a neoplasm. The diagnosis lay between a passive effusion due to the heart-failure, tuberculous pleurisy, and an effusion due to a neoplasm of the endothelial lining of the pleura. In the first case the fluid should contain only a few endothelial cells, which should not be in clumps and should show no signs of mitosis. In tuberculous pleurisy the cell content of the fluid is very similar to this, except that the cells should show no mitosis. The absence of tubercle bacilli on culture, and the absence of any sign of tuberculosis in the lung does not invalidate this diagnosis, but to make the diagnosis certain one way or the other, guinea-pig inoculation should have been carried out. The large numbers of endothelial cells, their presence in clumps, and the mitotic activity displayed are all strongly in favour of a neoplasm arising primarily in the endothelium of the pleura.

The diagnosis must therefore be uncertain, being either tuberculosis or neoplasm. Only future developments will enable one to decide which it is.

TREATMENT.

Patient was put to bed on admission, propped up in a semi-upright position with pillows, and not allowed to get up.

He was put on a milk diet at first. On the fifth day he was promoted to a light dry diet, a copy of which is appended.

Early morning. Orange juice -100 c.c. Sugar - 10 gms.
Breakfast. Tea - 150 c.c. with milk - 60 c.c. One egg lightly boiled.
Crisp toast - 1 thin slice.
Butter - 10 gms. (one good pat).
Jelly, marmalade, or honey - a teaspoon.

11 a.m. Orange juice - 100 c.c. Sugar - 10 gms.
Dinner. Fish (not fried) medium helping, or chicken or tripe or rabbit. Cream or soufflee can be made from the rabbit, chicken or fish.
Butter - 10 gms.

A tablespoonful vegetable, put through a sieve, cauli-
flower, spinach or carrot mashed. No cabbage, sprouts, peas, beans or
lentils.

Curds or custard made with milk 100 c.c. Stewed apples - 3 table-
spoonfuls.

Tea. Tea - 150 c.c. milk - 60 c.c.
Egg - lightly boiled.
Crisp toast - 1 thin slice.
Butter - 10 gm. (on good pat).
Jelly, marmalade, or honey - one teaspoon.

Supper. Benger's food - milk 200 c.c., dry Benger's 15 gm., or the
same amount of milk pudding if desired.

No salt is used in cooking or served on tray. No water or extra
fluid is given. Butter need not be salt free. Bread need not be salt-
free as so little is given. Sugar can be given as desired.

This diet gives a total calorie value of 1400 Cals., made up as
follows: Cho. 153 gms. (627.3 Cals.). Prot. 46 gms. (425 Cals.). Fats -
67 gms. (275 Cals.).

It contains 930 cc of fluid, and is salt poor.

Drugs: He received Blue pill, gr.iv, on admission, and again on 3rd.,
6th., and 17th. days.

PROGRESS NOTES.

With rest in bed and diet, the oedema subsided, and the dysp-
noea became much less, the resps. falling from 28-32/min. to 22/min. The
pulse, which was 126/min. on admission though quite regular, fell to
84/min. within a few days, and stayed there.

The physical signs of pleural effusion became more marked, and
after X-ray examination the right pleural cavity was tapped on the 8th.
day, oxs. viii of almost clear fluid being withdrawn. After aspiration
patient's condition improved. A second X-ray examination showed a very
small quantity of residual fluid in the right costo-phrenic sinus. His
temperature remained normal.

His condition improved steadily, and on discharge he was feeling
well, and there were no abnormal physical signs whatsoever.

Discharged - 3. 4. 32.

Admitted -

Died -

Complaint: 1. Breathlessness on exertion. 14 months.
2. Swelling of ankles and legs. 6 months.
3. Swelling and discomfort in abdomen. 3 months.

History: When 16 years old patient had rheumatic fever, and was told that her heart was affected. It never gave her any trouble however, except when she exerted herself considerably, when she would have an attack of palpitation; and she was able to lead a busy life for many years.

About fourteen months ago patient began to be troubled with slight breathlessness on exertion, and since that time she has become progressively more breathless, so that now she can scarcely walk even a short distance, such as the length of the ward. She also became easily tired, and six months ago she noticed her ankles beginning to swell especially at night.

She first noticed the swelling of her abdomen three months ago, since when it has become progressively larger. It has caused her a dull aching feeling in the left side. She is much troubled with flatulence, and her bowels, which used to be regular, have become increasingly constipated.

She has had a cough with a thick sputum for the past eighteen months.

Previous Health: Rheumatic fever when aet. 16 years. Whooping-cough eighteen months ago. Influenza several times.

Family History: Husband died, aet. 32, pulmonary tuberculosis.

Two sons alive and well.

Home conditions - good.

EXAMINATION.

Cardio-vascular System.

Pulse - 120/min., totally irregular in time and force, wall not palpable. Blood pressure, 125/85, the average of several readings of sphygmomanometer.

Heart - Right border is $\frac{1}{2}$" from the right sternal margin.

Upper border at the second rib.
Apex beat palpated in 5th. space, 1” outside the midclavicular line.

Auscultation. A loud blowing systolic murmur is heard in the mitral area, propagated through to the axilla and to the scapula behind. When the rate had slowed to 90/min. some observers thought they heard a mid-diastolic murmur. A short, softer systolic murmur was heard at the tricuspid area. The second sound was closed in all areas, but in the pulmonary area it was markedly accentuated and reduplicated.

Haemopoietic System.

The spleen was very much enlarged, forming a large tumour mass occupying the whole of the left side of the abdomen. It extended over to the umbilicus, and downwards almost to the symphysis pubis. There were no enlarged glands.

Blood Count.  R.B.Cs.  2,980,000 per cmm.

Haemoglobin  -  60%.  Colour index - 1.

W.B.Cs.  4,100 per cmm.

Differential count: lymphocytes  -  41%.

polymorphs  -  59%.

A film showed the typical blood-picture of a secondary anaemia, with anisocytosis, poikilocytosis, and polychromasia.

Blood fragility test - normal.

Splenatic puncture. Red cells as in blood film.

White cells - 85% mononuclears.

Respiratory System.

Subjective symptoms as in history.

Respirations - 22/min.

Sputum - average amount, muco-purulent in character. Numerous and varied types of organisms, pus cells, and epithelial cells containing a brown pigment.

Inspection - X Ray Chest is somewhat emphysematous.

Percussion - Note is increased all over chest, except at the bases, where it is somewhat impaired.

Auscultation - Breath-sounds vesicular, with some prolongation of expiration and a few rhonchi. There are some crepitations at the bases.

Alimentary System.

Subjective symptoms as in history.

Tongue - clean and moist. Teeth - all false.
Abdomen - Left side obscured by the enlarged spleen. Liver palpable one inch below the costal margin. No pulsation. Slight tenderness.

**Urinary System.**

No subjective phenomena.

Kidneys not palpable. No tenderness in kidney angles.

Urine. sp.gr. 1020. Acid. No abnormal constituents.

**Nervous System.** - Nil to note.

**DIAGNOSIS.**

Mrs. T. presents two symptoms which are absolutely diagnostic of one condition, namely heart failure of the congestive type. These symptoms are progressive breathlessness on exertion, and swelling of the feet and ankles; the other symptoms, palpitation, irregular pulse, and crepitations at the bases, merely help to confirm the diagnosis. There are, however, numerous conditions which precede and ultimately cause heart-failure, and we must now decide what the condition is in this case.

The failure may be due to some condition in a. the cardiovascular system; b. respiratory system; or c. renal system. The absence of hypertrophy of the left ventricle, the normal blood-pressure, and the normal urine, definitely excludes any renal condition. The absence of the first two signs also rules out arterial disease. There is thus left for consideration the heart itself and the respiratory system. In this case the respiratory symptoms and signs were not at all marked, and in any case they had only developed after symptoms of heart-failure had already manifested themselves.

In the heart itself there are two structures to be considered, the myocardium and the endocardium. When the endocardium is diseased the valves are involved, and either incompetence or stenosis, or both, develop, and murmurs are heard on auscultation. In this case there was at least a systolic mitral murmur, identified by its point of maximum intensity, and its direction of propagation; and there was a possible diastolic murmur in the mitral area, diagnosed by being limited to an area at and just internal to the apex beat. The first murmur indicates an incompetence of the mitral valve, while the latter,
If present, is diagnostic of a mitral stenosis, when fibrillation is present. The total irregularity of the pulse, and the fact that the rate at the apex was faster than at the radial pulse, is diagnostic of auricular fibrillation. Auricular fibrillation may not always be so easy of diagnosis, however. If the pulse is slow, about 75/min., but wholly irregular, the patient may be in the transition stage from normal rhythm to the abnormal rhythm of heart-block. To distinguish between them the patient should be asked if he has been under treatment with digitalis. A pulse showing numerous extra-systoles without any block may be very difficult to distinguish from one of fibrillation.

In the latter case, one would have to watch the result of exhibiting digitalis; or have a electro-cardiogram tracing done, when the distinction will at once be clear.

It is probable that there were changes in the myocardium also, of the nature of a chronic myocarditis, due to the old-standing mitral incompetence, and possibly to a rheumatic infection of the myocardium.

The lung symptoms and signs were due to congestion in the pulmonary circulation, following on the heart-failure.

The enlarged spleen could not be explained by the heart-failure, though the suggestion was made that it might be due to a series of infarcts. The smoothness, size, and uniform enlargement of the organ, however, ruled that out. A variety of conditions may produce enlargement of the spleen, in a woman of this age who shows no evidence of syphilis or tuberculosis, and has not been abroad, namely: 1. The leukaemias, myelogenous and lymphatic; b. splenic anaemia; c. haemolytic jaundice; d. lymphadenoma; e. sarcoma; f. pernicious anaemia rarely; g. mitral disease. Aspleen of the size found in this case would at once suggest one of the leukaemias, especially the myelogenous type, but an examination of the white cells of the blood disposed of this diagnosis, confirmation being obtained by the splenic puncture. In lymphadenoma there are generally signs of enlarged glands elsewhere in the body, and there is an eosinophilia, which was not present here. Sarcoma is very rare and is usually secondary.

The diagnosis therefore lay between splenic anaemia and haemolytic jaundice, with the remote possibility of pernicious anaemia. The condition was finally diagnosed as splenic anaemia, the
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Mrs. J's Temperature Chart

DEATH
Showing the effect of Diginutin on the pulse-rate in Case 2.
diagnosis being based on the following points: 1. Total absence of jaundice which is present in haemolytic jaundice. 2. The blood-count which revealed a secondary type of anaemia (microcytic), with a leucopenia and relative lymphocytosis. 3. Normal fragility of the red cells.

The third and terminal condition present was undoubtedly erysipelas. The spreading red blush with the definite raised margin, and the remittent type of fever, are quite characteristic of the condition. Where the infection came from is difficult to say, but probably was obtained from some other patient who was infected or who acted as a carrier.

Treatment and Progress Notes.

On admission, patient was put to bed and given diginutin, min. xv. 4-hourly from 8 a.m. till 12 p.m. The pulse was 120/min. on admission and totally irregular. She was put on a light dry diet, identical with the one detailed in Case 1.

The following day the pulse-rate had fallen to 100/min. in the morning, and the diginutin was reduced to mins. x.t.i.d. On the following day it had again fallen to 84/min., and the diginutin was again reduced to min. v.b.i.d., and this dose was maintained throughout. The accompanying figure shows the relation between pulse-rate and the amount of digitalis administered.

She was occasionally troubled with insomnia, and for this received adalin gr. x. during the night. This is a hypnotic of the dulphonal group.

She improved as regards her cardiac condition, but was still troubled with the symptoms caused by the enlarged spleen. Operation was considered, when the diagnosis had been settled, but she did not feel inclined for one, and it was thought that her general condition did not warrant it. There was no oedema, though the urinary output remained low, averaging 30-35 ozs. per diem. This would be due in part at least to the dry diet.

Nearly three weeks after admission, on 28. 3. 32, it was noticed that her face was a brightly flushed, and her cheeks of a higher colour. The temperature, which had hitherto been perfectly normal, rose abruptly to 102 F. She was given pyramidon, gr. x., a
diagnosis being based on the following points: 1. Total absence of jaundice which is present in haemolytic jaundice. 2. The blood-count which revealed a secondary type of anaemia (microcytic), with a leucopenia and relative lymphocytosis. 3. Normal fragility of the red cells.

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which is an anti-pyretic and analgesic of the phenacetin group; quinine salicylate, gr. ii 4-hourly, 8 a.m.-12 p.m.; and a smart purge, - calomel gr ii, followed by a Seidlitz powder.

The temperature still remained high and remittent in character (see chart), and there was an increase in the white blood cells to 9000/cmm. Within a day or two a definite margin to the redness could be made out. It spread down the front of her neck onto her chest, and then towards the left axilla; and also over the top of her head and down the back of her neck. Treatment in the form of 4-hourly Magnes. sulphate soaks, and painting with iodine, was instituted immediately, but it proved of no avail. Her face became very swollen, and was almost unrecognisable. Insomnia was marked during this febrile, and she received adalin gr. x. every night. Before the end, an abscess formed on the right eyelid and was discharging pus.

The patient gradually became collapsed, her face became very cyanosed, and she died on 4. 4. 32.

3. 3. 32. Throat swab. No B. diphtheria or other organisms were found in the direct smear. In culture there were growths of staph. albus and of haemolytic and non-haemolytic streptococci. Pus from the abscess showed growths of diplo-streptococci were obtained which may be either of the pneumococcal or streptococcal groups. (Bacteriological report),
DISCUSSION.

These two cases are very good examples of heart-failure with a regular and with an irregular pulse; and both are due to quite different causes.

Heart failure may be defined as "the condition in which the heart is unable to maintain an efficient circulation when called upon to meet the efforts necessary to the daily life of the individual." It will thus be seen that the essential criterion is the manner in which the heart performs the work it is called upon to do, and if this is done efficiently and without any embarrassment to the individual in his daily round, heart failure does not exist, no matter what other signs may be found in the heart on physical examination. This is most admirably illustrated in Case 2, who was able to lead a fairly strenuous life, in spite of having an incompetent and possibly stenosed mitral valve.

Cases of heart failure can be divided into two distinct groups, namely those in which exhibit the characteristic symptoms belonging to the anginal group, and those in whom the failure manifests itself first by breathlessness on exertion, and swelling of the ankles, and ultimately proceeds to a profound engorgement of the venous system. This second group might be classified as cases of congestive heart failure, and it is to this group that the two cases under discussion belong. The former group is associated with disease of the aortic valves and coronary arteries, due frequently to syphilis. The second group is associated with many and diverse conditions, both in the cardio-vascular system itself, and outside it.

Consideration of Symptoms. Both the cases under discussion present two symptoms in common, namely increasing breathlessness on exertion and oedema of the lower extremities beginning at the feet and ankles. These two symptoms, when present together, are pathognomonic of congestive heart failure, and therefore are of considerable importance. Everyone tends to become breathless on exertion at some time or another, when the exertion has been fairly strenuous, and no remark is passed on it, much less is it suggested that the person has cardiac failure. We may take it therefore that there is a physiological limit to the amount of work the heart can perform, this limit varying with the condition
of training of the individual, as is shown by the fact that the same individual can perform much more strenuous work if he improves his physical condition. This dyspnoea on exertion in a normal person, however, must be recognised as due not entirely to the heart, but also to the mechanical and functional efficiency of the muscles of the body generally. A person in whom this efficiency is low will require more oxygen, and therefore tend to become breathless more quickly than an athlete in whom it is high. Inasmuch, however, as the heart itself is a muscular organ, it may be inferred that its condition is on a level with the rest of the body musculature, and therefore that the amount of dyspnoea present will give a fair indication of what that condition is.

When a heart begins to fail, however, this dyspnoea becomes a prominent feature. At first it manifests itself in the more strenuous pursuits in which the individual indulges, and which previously had caused him no distress whatever. It is a progressive phenomenon and gradually becomes more and more marked, until, as we have seen in these two cases, even a short walk on the level serves to make them breathless. The reason for this breathlessness is hard to find, and I do not think that a rational and complete explanation has yet been given. In dealing with this question, MacKenzie merely says, "The centre for respiration in the medulla is influenced by its blood-supply and its nervous connection with the periphery. A free blood-supply is necessary to supply oxygen and remove the carbonic acid; and an insufficient interchange of these gases is betrayed by an increased activity of the centre, thus giving rise to breathlessness." Why this dyspnoea should develop with the onset of heart-failure is not clear. It may be due to one or more of a number of factors, namely, an increased sensitivity and therefore increased activity of the respiratory centre; and increased need for oxygen by the muscles, including the myocardium; or a pulmonary condition which prevents the free interchange of gases between the blood and the inspired air; or a change in the blood chemistry which acts upon the respiratory centre; or lastly an insufficient amount of blood being thrown into the systemic circulation.

The oedema is also of considerable interest. Both the cases complained of swelling of the feet and ankles when they were up and going about, i.e. in the most dependent parts of their bodies. After a
few hours in bed, however, neither showed any oedema of these parts, the fluid instead having gravitated to what were now the most dependent parts, namely the back and sacral region, and the bases of the lungs. In Case 1 there was marked oedema of the subcutaneous tissues of the back, and in both cases there were signs of fluid in the lungs, as evidenced by the crepitations heard at the bases. These later, indeed, are often the very first sign of impending heart-failure.

The causes of cardiac oedema may be put as follows:

1. The venous pressure, in severe cases of congestive heart-failure, is raised from the normal level of 10-15 cms. water in the median basilic vein to 30-35 cms. water. The capillary pressure must be correspondingly raised, and this tends to filter fluid out into the tissue spaces. This cannot be the sole factor, because a rise of venous pressure is not necessarily associated with oedema unless malnutrition is present as well.

2. With a failing circulation, there is an inadequate blood-supply to the organs. Imperfect metabolism may occur in the tissue cells, and metabolites, instead of being rapidly removed, accumulate in the tissue spaces, the local osmotic pressure rises, and retention of fluid follows.

3. The raised venous pressure and metabolites dilate the capillaries and their permeability is increased; protein escapes into the tissue spaces in excessive amounts, and this tends to disturb the water balance between the blood and the tissue cells.

4. There may be swelling of the tissue cells themselves, as the increased hydorgen-ion concentration of the colloids enables them to take up more water by imbibition.

5. The oedema fluid is possibly formed all over the body, and gravitates down to accumulate and become obvious in the dependent parts.

Oedema may occur in various other conditions besides heart-failure and the distribution may vary with the cause; but probably the causes are much the same.

Pathology of the Cases.

In Case 1. the absence of any murmurs almost certainly excludes the endocardium from consideration, and attention is focussed at once upon the myocardium. The completely normal temperature throughout his stay in hospital is practically conclusive evidence of the absence of
active bacterial infection, and therefore suggests that the lesion is in
the nature of a chronic degenerative or inflammatory process in the
myocardium. The Wassermann reaction being negative, it is unlikely to be
syphilitic in origin, and as there was no history of rheumatism in any
of its forms, it is unlikely to be a chronic myocarditis following on
an acute rheumatic infection of the myocardium at an earlier period. One
small point however may be worthy of some consideration. He stated that
as a child, he remembered the skin of his hands peeling off. This was
unaccompanied, so far as he remembered by any other symptoms; but in
children this is commonly seen after a scarlatinal infection, which may
be so mild as to escape notice entirely. Scarlet fever is a common ante-
cedent to diseases of the heart, either endocarditis or myocarditis, all
being due to a haemolytic streptococcus. There is the possibility here
that he suffered from a mild scarlatinal infection, in which his myocar-
dium was involved to a degree sufficient to weaken it somewhat, and
render it more susceptible to strain or infection later.

In discussing the diagnosis of this case, two conditions were
regarded as being implicated in the production of the heart failure,
namely, the marked history of alcoholism, and the presence of definite
emphysema of the lungs. Taking the latter first, the essential point is
the
an obstruction to a flow of blood through the lung-capillaries, owing
to a thinning and breaking of many of the alveolar walls, and a consequ-
ent obliteration of the capillaries. The emphysema might be due to a
chronic cough, e.g. bronchitis; or it may be due to a congenital weak-
ness of the alveolar walls, owing to a lack of elastic tissue. This
causes them to distend more readily than those in anormal person, and re
remain distended, the distension being primarily caused byn expiratory
efforts with the glottis closed. Or according to Freund's theory, the
primary condition is a disease of the costal cartilages, a chronic hyper
plasia with premature ossification, which brings about a state of rigid
dilatation of the chest to which the emphysema is secondary.

The obliteration of a considerable portion of the pulmonary
capillary bed results in a raising of the pulmonary blood pressure, with
consequent strain being thrown on the right ventricle. This first dilate
and then hypertrophies, and then when the limit of hypertrophy is reach-
and the strain is still increasing, dilation again occurs, which this
time is progressive and results ultimately in failure of the right side.

In this case there was no definite **evidence** of enlargement of
the right side; but that does not mean that there was none, because
the emphysematous lung overlaps the right side and gives out a resonant
note on percussion.

The cough which he had had for two was probably due to the
emphysema, but may have been the cause of it. There was, however,
no adequate cause for it in the lung apart from the emphysema. The
suggested malignant condition in the pleura would hardly account for it.
The cause of the emphysema in this case is rather hard to find. Apart
from this cough there was no definite cause for it, and it appeared of
considerably longer duration than two years. This is in favour of
Freund's theory, that the emphysema was secondary to a developmental
abnormality in the chest.

The effects of the chronic alcoholism would be of a degener-
avative nature rather than a chronic inflammation. Two degenerative
changes are produced by alcohol, namely fatty degeneration of the mus-
cles themselves, the contractile elements of the fibres being
replaced by globules of fat, and fatty infiltration, in which the peri-
cardial fat invades the muscle, the fat cells growing in between the
muscle fibres and producing pressure atrophy of these fibres. The effect
of these degenerative changes is to weaken the heart muscle,
which is now incapable of the work which it formerly performed. The
ventricles dilate, especially the left ventricle which has the most
work to do, and this causes a damming up of the blood-stream at the
centre. First of all the pulmonary circulation becomes congested, and
then this spreads backwards until it involves the whole venous side,
with consequent engorgement and stagnation, and the production of a
condition of congestive heart-failure.

**Case II.** offers a most interesting contrast to the other.
The endocardium was undoubtedly affected, the murmurs that were present
being organic in origin, and indicating that the valves were damaged.
In this case there was a difference of opinion as to whether or not a
diastolic mitral murmur was present. None was heard before digitalisa-
tion, but when the heart rate had slowed to 80/min., a murmur, mid-diastolic in time, was thought by some to be heard in the mitral, limited to an area at and just internal to the apex beat. Personally, I did not hear it at all. Now if the only murmur present was a mitral systolic the condition was one of mitral incompetence; whereas if both systolic and diastolic murmurs were present, it was a mitral stenosis, a vastly different matter. A very interesting question is raised by this difference of opinion, namely does a person suffering from mitral stenosis live to a greater age than sixty years, and it is a very difficult question about which to be dogmatic. It is, however, a very unusual occurrence, as most of the cases do not see fifty years, the condition being a progressive one and one which early leads to heart-failure. The question, however, as to whether or not stenosis of the mitral valve was present was never settled, as no post-mortem examination was carried out.

The etiology of the case is obvious, for when she was sixteen years old she suffered from rheumatic fever. Rheumatism, in any one of its numerous forms, is regarded as the etiological factor par excellence of disease of the mitral valves. The mechanism is as follows:

The toxin produced by the haemolytic streptococcus to which the rheumatic conditions are due, damages the endothelial lining of the valve cusps, the damage taking place at the line of closure of the cusps, and minute beady swellings appear. Gross infection of this damaged part by the streptococci may follow, with the production of sub-acute bacterial endocarditis and death of the patient within a year. The damaged valves may recover, however, with a thickening and shortening of the valve-cusps due to the formation of fibrous tissue. If this thickening and shortening is sufficiently marked, incompetence of the valve results. The valve in any case is weakened, and in any subsequent infection is liable to become involved again, with further shortening and thickening of the cusps. The latter may this time become adherent at their free edges - narrowing or stenosis of the opening being the result. This may go on till only a very small opening is left, forming the button-hole type of mitral stenosis. The valve cusps may also become adherent to one another without having become sufficiently shortened to produce mitral incompetence, and stenosis is present alone.
The rheumatic virus is quite liable to attack the myocardium as well, and this is what possibly what was present in this case, a chronic myocarditis being present in addition to the endocarditis. In addition however there would be a certain amount of senile myocarditis present, and indeed this may have been fairly extensive.

Production of Murmurs. The systolic mitral murmur, indicating mitral insufficiency, was produced by the blood being forced through the partly open mitral valve during ventricular systole. In a mitral stenosis with normal rhythm, a pre-systolic murmur is present, due to the forcing of the blood through the stenosed orifice from auricle to ventricle during auricular systole. When auricular fibrillation sets in, however, the presystolic murmur at once disappears, as the auricle is no longer contracting; but a diastolic murmur takes its place, being produced by the suction of the expanding ventricle during ventricular diastole, drawing blood through the narrowed mitral valve.

Production of Symptoms. The incompetence, and possible stenosis, of the mitral valve would cause an engorgement of the left auricle with regurgitating blood, and it would first dilate to accommodate the increased contents, and then hypertrophy in an attempt to overcome the obstruction. The auricles, however, have not much capacity for hypertrophy, and ultimately it began to dilate again. This caused a stagnation of the blood in the pulmonary circulation, and through an increasing strain on the right side of the heart, first on the ventricle, and then on the auricle. The would first hypertrophy and when the limit of its power had been reached, dilation set in with consequent failure. The dilatation caused a stretching of the ring round the tricuspid valve, and relative incompetence of the valve resulted, with the production of the systolic murmur heard in the tricuspid area. The incompetence of this valve in turn led to engorgement of the right auricle, which finally became greatly dilated, as was shown by the right border being found 4" to the right of the sternum. Stasis in the venous side of the systemic circulation followed, with the production of the enlarged liver and oedema.

The engorgement of the pulmonary circulation causes a transudation
of fluid from the capillaries into the alveoli, which gives rise to the crepitations heard at the bases of the lungs, in both cases. In addition, in Case ii. there was a cough with a thick expectoration. The cough was due to the engorgement, with a greater or less amount of bronchitis; the expectoration containing numerous epithelial cells laden with a brown pigment - heart-failure cells.

**Auricular Fibrillation.** This was diagnosed in Case ii. by the totally irregular character of the pulse, and the response of the heart to impossible digitalis. How long this had been present it is difficult to say, but the history states that whenever she exerted herself unduly she would have an attack of palpitation. This may have been a simple increase in the heart rate, but on the other hand they were possibly paroxysmal attacks of auricular fibrillation.

**Causes.** The two conditions in which fibrillation of the auricles most frequently occurs are 1. mitral stenosis; and 2. senile myocarditis. It also occurs quite frequently in hyperthyroidism, in the acute fevers - pneumonia and typhoid, and rheumatic myocarditis. So frequently did it occur in mitral stenosis that the older physicians called it the pulsus irregularis perpetuus of mitral stenosis. In this case the cause might be one of several. If mitral stenosis were present, it was probably due to it. If however mitral incompetence alone was present, the etiological factor would be more likely to be senile myocarditis or rheumatic myocarditis, or both.

**Nature.** Lewis defines it as "a condition in which the auricles fail to contract en masse, the muscle activity consisting only of fibrillating twitchings; the normal and regular impulses transmitted to the ventricle are absent, while rapid and irregular impulses produced in the auricle replace them and produce gross irregularity of the heart's action." The first person to recognise this type of irregularity as a separate and definite entity was Sir James MacKenzie in 1890, by means of the polygraph. He considered, at first, that the auricles and ventricles contracted simultaneously, as he could find no evidence of auricular activity in the polygraph tracings. The stimulus he considered to arise in the A-V node, and he called it "nodal rhythm." Cushny (1900) was the first to suggest that this type of irregularity might be due to fibrillation.
of the auricles. With the advent of the electrocardiograph more accurate observations were made regarding the contractions of the chambers of the heart, and in 1909 Lewis found that the oscillations produced during ventricular diastole by the fibrillating auricle in the electrocardiogram of a dog in which auricular fibrillation had been experimentally produced, corresponded with those seen in the electrocardiogram of MacKenzie's nodal rhythm, and so established the fact that the latter was really due to fibrillation of the auricles. Lewis has pursued his enquiries into the nature of this condition, and his views as to the origin and method of production are generally accepted as being correct. (see infra.)

The pathology of the condition is indefinite. Dilatation or hypertrophy of the auricles is frequent; and microscopically there is a more or less intense grade of subacute or chronic inflammatory change of the myocardium, progressing to fibrosis, and seen most conspicuously in the auricles. The characteristic picture is a diffuse fibrosis, accompanied by leucocytic infiltration, and atrophy of the neighbouring muscle cells. Similar lesions, however, are found in cases where fibrillation has never occurred, and hearts which have shown this disorder may not present any of these lesions.

When fibrillating, the auricle is beating about 450 times per minute, but no co-ordinate contractions are visible. A fine irregular tremulousness on the surface of the auricles is all that can be seen. The understanding of the nature of the condition is due to the conception of a "circus movement" which can be shown to exist in a fibrillating auricle. If a normal auricle is stimulated at one point, the waves of contraction spread out equally in all directions till they meet at a point on the other side. While contracting, the muscle is completely refractory to further stimuli, and so when the waves of excitation reach the other point, they are faced with unresponsive tissue and can go no further. As the rate of excitation increases, however, a partially refractory state of the auricular muscle develops, in which certain parts of the muscle become responsive again, while others are still refractory when each stimulus falls on the muscle. If then, with a high rate of stimulus formation, a stimulus sets out from the S.A. node
node over the auricles, it will be met in one place with the muscle in
the refractory state, while in some other direction it is responsive,
so that the wave tends to travel in the latter direction; when it
reaches the place that was formerly refractory, it now finds it re-
ponsive and travels on, going round and round the auricles, the refractor-
ry phase passing off at any one place just before the wave of excita-
tion arrives. This is known as the "circus movement."

To establish a circus movement two conditions must be present:
a. An area of refractoriness must be present to prevent the passage of
the impulse in one direction. b. The originally refractory area must
be recovered when the excitatory process reaches it for the second time.
Conditions favouring such a movement are: a. a large ring, i.e. dilated
auricles; b. slow rate of conduction; c. short refractory period.

In auricular fibrillation most of the auricle at any moment
is in a state of complete or partial refractoriness, and the wave, there-
fore, takes a coarsely sinuous path, which may vary from circuit to
circuit, looking for responsive tissue. Impulses do not reach the out-
lying parts of the auricle readily, being obstructed by refractory
areas, and therefore the A.V. node is stimulated at irregular intervals.
In spite of this however the stimuli are reaching the node too rapidly
and the A.V. bundle cannot transmit them all. The ventricular rate,
therefore, is much less than that of the auricles, while the beats are
completely irregular, and bear no constant relationship to one another.
The ventricular beats also may be large or small, depending upon how
much blood is present in the ventricle at the beginning of each systole
The smaller ones may be unable to open the aortic valves, and so no
blood passes into the aorta, and the pulse misses a beat at the wrist.
The rate at the apex is thus found to be faster than the rate at the
radial pulse. This rapid and irregular beating of the heart greatly
interferes with the output per minute which falls to less than four
litres (the normal being 7-8 litres).

Palpitation. This was present in both the cases. Both patients de-
scribed it as a violent beating of the heart, coming on generally after
exertion. This description, however, gives no clear idea of what exactly
was present, and it may have been one of several different things.
It may simply have been an increased heart rate, not rising above 170/min., in which the heart's action is perfectly normal and the rhythm regular, except for an occasional extra-systole. The other causes have been classed together, owing to the heart's contraction not starting at the normal place (MacKenzie.) This class includes paroxysmal tachycardia, auricular fibrillation, and auricular flutter. In these the heart-rate may reach 300/min., and the rhythm is frequently irregular. It is probable that both cases belonged to the first type, but Case ii. may have been an example of paroxysmal fibrillation.

**Treatment.** The first essential in treating any morbid condition is to have a clear idea of what is to be treated, and the treatment of heart-failure is no exception. As Sir James MacKenzie says, "This is all the more necessary if we desire to treat the patient on a rational system, for it will be found that the careful analysis of the symptoms in each case, and of the circumstances that have led to heart-failure, very so much that no two patients present the same conditions, and therefore each patient has to be treated with reference to his special requirements." This dictum is most aptly illustrated in these two cases.

**Principles.** We must bear in mind always that failure of the heart is due always to an exhaustion of its reserve force, and that the guiding principle is to restore this reserve force, if not to normal, at least to a sufficient degree as to enable it to perform its duties fairly efficiently. To this end we must make a thorough investigation into the cause of the failure, and also we must have a shrewd idea of the value of the various symptoms, subjective as well as objective. If we can remove the cause of the failure, so much the better; but not often this is possible, and one must be content to strengthen the muscle, and then let it work under conditions which are not likely to cause it undue strain. On the other hand we must avoid going too far, refrain from and restricting the patient's activities more than is necessary. A lot of unnecessary inconvenience and trouble will thereby be avoided.

**Remedial Measures.** When the heart begins to fail the first essential is to give it rest. As this cannot be effected directly, the body must be rested, in order to give the heart the minimum amount of work todo. This is done by first of all restricting the patient's activities; but
if the failure is too far advanced for that, as it was in the two cases under consideration, the patient must be put to bed. It is best that at first rest in bed should be complete and absolute, the patient not getting up at all, and having as little excitement and nervous fatigue as possible. Such patients are frequently somewhat dyspnoeic even at rest, and they find great relief in reclining in the bed, propped up in a semi-upright position by pillows or a bed-rest. Such measures were adopted in both our cases, and though in neither was the dyspnoea marked, they obtained great relief.

Diet. this is a question of considerable importance. In resting the patient it is advisable to rest every part of him, including his digestive processes, and for this reason the patient must avoid overfeeding. Nature, of course, provides for this by reducing the individual's appetite while he is in bed, and it can often be left to the patient himself to choose his own diet, subject to certain general directions. The discovery of the means of measuring the basal metabolic rate has made the practice of dietetics a much more exact a science than it ever pretended to be before; and we can now lay down a diet, the calorific value of which is known, with the assurance that what we are giving is neither too much or too little.

The diet should be a light one, avoiding an excess of proteins on account of its action in increasing the metabolism, and giving a sufficiency of vitamins. The diet mapped out in the two cases is a typical one, and one that gives very little trouble in its preparation. It will be noticed that its calorific value is only 1400 Cals.; but although this is somewhat lower than the B.M.R. of a normal healthy adult (1700 Cals.), we must remember that we are not dealing with healthy people, and that the B.M.R. of an ill person is undoubtedly lower than that of a healthy one.

The absence of salt in the dietary, and the restriction of fluids, are measures taken to relieve the oedema. The absence of salt diminishes the concentration of salts in the body, as there is always salt excretion taking place and none is being ingested to take its place. This lessens the ability of the body tissues to attract fluid, this normally being necessary to hold the salts in solution. The modus operandi of the restriction of fluids is obvious. Either the
salt or the fluids can be increased at will, according to the progress of the patient.

**Exercise.** The body generally is always benefited by exercise, and the heart is no exception to this rule. The good effect upon it arises not only from the exercise of its inherent function, but also that the more energetic action causes the whole organ to be flushed by a large supply of blood. It can only be allowed, of course, when the heart has regained a fair proportion of its former strength. Therefore when the patient is has been in bed long enough to obtain this result, he is allowed up for so long each day, the time being gradually increased, and the amount of exercise taken also graduated. When he is discharged he should be able to undertake most things that do not involve any strain.

**Drugs.** There are many drugs in use for the condition of heart-failure but their number is a fair indication of their efficacy. These two cases are a very good illustration of the use of drugs in cardiac failure. Case i. received no drugs at all, except for an occasional dose of mercury to clear his bowels. None were required, the main thing being to secure rest in bed and a suitable diet.

In Case ii. the presence of auricular fibrillation was a clear indication for the use of digitalis in some form; diginutin, a proprietary preparation, was used.

Digitalis is mentioned as a drug as early as 1250 A.D., and was used to a limited extent thereafter for various diseases. In 1785, Withering published the results of ten years observations on cases of dropsy, and advised it chiefly as a diuretic, but also recognised its beneficial action on the heart. During the 19th. century little progress was made with regard to its use and actions in heart diseases, and it was not until 1900 that MacKenzie, after patient investigations, discovered its specific action in auricular fibrillation, or as he called it at the time, "nodal rhythm."

The success with which the drug is used varies in different cases. The best results are obtained in acute fibrillation supervening on an old mitral lesion of rheumatic origin, in which there were no previous signs of failure. But it is useful in many other cases of longer standing. It often fails when active endocarditis is present.
It frequently has little effect on old-standing cases with widespread changes in heart, kidneys, and vessels, with a moderate pulse, but may relieve the symptoms produced by exertion in such cases.

**Action.** Digitalis does not cure auricular fibrillation, for the auricles continue to fibrillate. Indeed, it generally accelerates the rate of the circus movements in the auricles, this effect being ascribed to an increased activity of the Vagus which results in a shortening of the refractory period of the auricular muscle. Digitalis also has a direct action on the auricular fibres, tending to lengthen the refractory period, and transmission interval, and therefore slow the rate of fibrillation. Generally, however, the vagus factor prevails, and the rate is increased.

As the pulse-rate falls, however, it is obvious that some blockage to the passage of stimuli over the auriculo-ventricular bundle is set up; or that the excitability of the ventricle is so reduced, that it will respond only to a small proportion of the impulses which it receives. The block may be partial or complete, according to the amount of digitalis administered; the more that is given the more complete it is. Finally it does become complete, and the ventricle beats with its own rhythm, perfectly regularly and about 40-50 times per minute.

The diminished rate of the ventricle allows more complete filling of the ventricular cavity, and therefore more stretching of the muscle-fibres. Also it allows the latter to recuperate to a greater extent between each beat. Both these factors tend to strengthen the beat of the ventricle, while digitalis also has a direct strengthening effect on the muscle itself.

The blocking of the impulses passing through the bundle of His is due to two factors, namely, a. an inhibitory action on the A.V. node through the Vagus; and b. a direct action on the conducting fibres of the A.V. bundle. The relative importance of these two factors varies with the amount of digitalis given, and with the length of time during which it is given, the direct action superseding the indirect inhibitory action.

The result of digitalis therapy is to slow the ventricular
rate very markedly, and may result in complete heart-block with the setting up of an idio-ventricular rhythm. A new form of irregularity may appear in some cases, when the heart is greatly slowed, this taking the form of coupled beats - pulsus bigeminus. The first beat is a normal ventricular beat due to a stimulus coming from the A.V. node. The second is an extra-systole arising from some focus in the ventricle and probably due to the tendency of digitalis to increase the irritability of the heart-muscle. Either The appearance of either complete heart-block or coupled beats is an indication that digitalis has been pushed far enough, if not too far, and that it is time that it was stopped.

The point to which the pulse may be slowed varies in different cases. The best are usually obtained with a rate of 70-80/min., but sometimes the results are best with a rate of 50-60/min. Each case must receive individual attention.

The question of the maintenance dose of digitalis to be administered once the pulse-rate has been reduced to the optimum, is of great importance. This also is found to vary, some cases requiring regular treatment with small doses, while others are able to discontinue the use of the drug once the heart rate is satisfactory. In the latter group, however, the heart is liable to return at any time to its former condition, if subjected to extra strain. In this case the maintenance dose arrived at was min.v. b.i.d.

Sleeplessness is often a feature in these cases, as it was here, and a mild hypnotic may be required. In this a proprietary preparation of the Sulphonal group was used - adalin - in grs. X doses.

The other conditions present in Case ii. cannot be regarded as having any direct connection with the heart condition.

The anaemia showed one rather curious feature, namely, the high colour index; in a secondary anaemia this is usually definitely low, about .6, whereas in this case it was 1. The reason for this is obscure, for the blood-picture showed a typical microcytic anaemia.

The causal factor in the production of splenic anaemia is not known, but the cure is definite and gives very good results, viz. general removal of the spleen. In this case, however, the condition was not
one in which operation was advisable. The gastro-intestinal symptoms were produced by the pressure of the enlarged spleen on the bowel.

The third condition, erysipelas, was, of the nature of an intercurrent infection, and death was probably due directly to this. It was an exceedingly good example of erysipelas migrans, the infection beginning on the face and spreading over the head and down the neck onto the chest and arm. The abscess formation showed that the patient was in a very poor condition to withstand the infection. The remedies used were those which are advocated at the present time, but are not at all specific.

Prognosis. In Case i. the prognosis is fairly good, if the lung condition is not what the bacteriological findings suggest it might be, viz. malignancy. He will have to be very careful not to over-exert himself, and will require to pay attention to his diet and home conditions, and avoid all causes of anxiety. In addition, alcohol for him must be taboo. If however the lung condition is malignant the outlook is very grave, and he will have, at most, two or three years to live. If it is tuberculous, the prognosis is materially improved, although it is by no means bright. The absence of any physical signs in the lungs themselves indicates that there is probably some slight apical lesion which has spread outwards to involve the pleura. If this is the case, the condition may clear up spontaneously, as it frequently does. On the other hand it may spread, and result in pulmonary phthisis, and or miliary tuberculosis, in both conditions the outlook is poor. His weakened heart, also, will not help him to withstand any serious disease.

In Case ii. the outlook at first was poor, owing to the degree of heart-failure and the presence of auricular fibrillation, and the age of the patient. She would, and undoubtedly did, improve under treatment but would probably have relapsed quickly. With the advent of the erysipelas, however, the prognosis became almost hopeless, as her heart was too weak to withstand the extra strain to which it was subjected, and the toxins which were circulating in the blood.
Mrs. R.  
Act. 42 years.  
House-wife.

Admitted -  
Discharged -

Complaint:  
1. Excessive thirst. )
2. Loss of weight. )  
3. Weakness. )  

Duration - six months.

History: About six months ago patient gradually became more and more thirsty. She drank a lot of lemonade and ate many oranges in an endeavour to assuage her thirst. At the same time she began to lose weight and this has been progressive. She had, however, a very good appetite and could eat anything. She says that after taking food she is troubled with flatulence and discomfort in the epigastrium. There is no actual pain or vomiting. She passes a large quantity of pale urine. She has never had any itching. All this time she was getting progressively weaker and less able for her work. Her feet get very cold at times. There has been no swelling of the ankles or breathlessness on exertion.

On the day before admission she felt very drowsy in the morning and stayed in bed all day. The drowsiness gradually got worse until she lost consciousness completely. She vomited during the day. Her doctor was then called in. He gave her an injection of 80 units of insulin and sent her into hospital on the following day.

Previous Health: Had scarlet fever when young and also measles. Her health has been good since marriage.

Family history - nil to note.

Home conditions good.

EXAMINATION.

On admission patient was unconscious. She had marked air-hunger, her respirations being deep and sighing. There was a smell of acetone in her breath. During the night she appeared to be in great pain, and lay moaning in bed. No tenderness could be found on examination of the abdomen and there was nothing else to account for her apparent pain.

Temp. on admission - 97 F.
EXAMINATION.

Patient is of average intelligence. She is of average development, but she is very thin and has obviously lost a fair amount of weight. She is cheerful and takes plenty of interest in her surroundings.

Height - 4ft. 11½ins. Weight - 6st. 5lb.

Cardio-Vascular System.

No subjective phenomena.

Pulse regular, 100/min., wave normal, B.P. 98/70, no thickening of the wall.

Heart - Apex beat in 5th interspace inside the midclavicular line. Right border at mid-sternum. Upper border behind 3rd. left rib. Sounds closed in all areas. First sound in the mitral area tends to be short and sharp.

Genito-Urinary System.

Subjective - Passes an excessive quantity of pale urine.

Urine - 62ozs. per diem, acid, sp. gr. 1030, sugar , 7.1gr/oz on admission, acetone , no other abnormal constituents. Nil to note microscopically.

No other signs referable to the urinary tract.

Patient was menstruating on admission, and continued to do so for twelve days.

Respiratory System.

No subjective phenomena.

Movements good. Vocal fremitus good.

Percussion note resonant all over.

Breath-sounds are vesicular, rather harsh in character. No accompaniments. Vocal resonance good.

Alimentary System.

Subjective phenomena as in history.

Tongue clean and moist. Teeth fair.

Abdominal examination revealed nothing of note.

Nervous System.

No sensory disturbances. All reflexes present and normal.

For some days after admission patient was unable to see to read or write even with glasses, though previously she had quite good sight. This gradually improved however, and three weeks after admission...
she was able to read and write without glasses.

**Other systems - Nil to note.**

**Special Examinations:**

28.1.32. Pasting Blood Sugar, 175 mgms%.

30.1.32. Fasting Blood Sugar, 125 mgms%.

7. 3.32. " " " 400 mgms%.

12. 3.32. " " " 200 mgms%.

8. 3.32. White blood count, 12,200 per cmm.

**Treatment.**

On admission, patient was put straight to bed, between blankets and surrounded with hot-water bottles. She was given insulin - 80 units the first day in four doses at 5-hourly intervals, and covered with glucose, 1.5 gms. per unit, given intra-venously. She recovered consciousness six hours after admission, but treatment was continued, and on the following day she received 70 units, in four doses, covered with glucose as before. On the third day after admission, she was able to take a fluid B.15 diet and thereafter improved steadily.

**Diet Chart.**

<table>
<thead>
<tr>
<th>Day</th>
<th>Diet</th>
<th>Calories</th>
<th>Urine - Sugar</th>
<th>Acetone</th>
<th>Insulin</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st.</td>
<td>-</td>
<td>480</td>
<td>--</td>
<td>--</td>
<td>units 20-3-hourly.</td>
</tr>
<tr>
<td>2nd.</td>
<td>-</td>
<td>360</td>
<td>G.F.A.</td>
<td>tr.</td>
<td>units 20-4-hourly;</td>
</tr>
<tr>
<td>3rd.</td>
<td>B.15.fluid. 1509</td>
<td>1:75</td>
<td>tr.</td>
<td>U.20 -6a.am. 10 -6p.m</td>
<td></td>
</tr>
<tr>
<td>4th.</td>
<td>B.15.ordy. 1518</td>
<td>1:1</td>
<td>--</td>
<td>--</td>
<td>units 30</td>
</tr>
<tr>
<td>5th.</td>
<td>&quot;</td>
<td>&quot;</td>
<td>&quot;</td>
<td>tr.</td>
<td>U.50 in 3 doses.</td>
</tr>
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<td>6th.</td>
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</table>

Discharged out, much improved. On 6.-25b diet and 20 units insulin daily.
6. 3. 32. Menstrual period began. Temperature rose abruptly to 102.5 F, pulse 120/min. White blood count - 12,200. Fasting blood sugar - 400mg%. By the following morning the temperature had fallen as quickly as it had risen. Bowels kept open with aloin pill 2-3 times weekly.

Weight on admission - 6st. 5lb.

" " discharge. 6st, 7½ lb.
CASE 2.

Admitted - 21. 4. 32. General labourer.
Discharged - 18. 5. 32.

Complaint: 1. Excessive thirst. } 8 weeks.
2. Polyuria.

History: Five weeks ago patient began to feel excessively thirsty, and tried to quench his thirst by drinking large quantities of water, and eating oranges. He also passed considerably more urine than normally - this necessitating his rising 3-4 times at night. Since then the thirst and other symptoms have increased. He also began to lose weight, and has become considerably thinner. For the past two weeks he has felt rather tired, and less able for his work. There has been no pruritus. He has always had a good appetite, but he has been eating more than usual during the past week five weeks.

Patient has been troubled with boils on the back of his neck for the past five or six years. They have never been very bad, but as soon as one crop cleared up, another crop formed.

He has been shortsighted for a long time, but his sight has grown worse during the past fortnight.

Previous History: Influenza once or twice. Nil else to note.

Family History. Mother died - cause unknown.
Father and rest of family - a. and w.
No history of diabetes in the family.

EXAMINATION.

Patient is a thin man, with an anxious expression on his face, He is a little unsteady on his feet. Is intelligent and answers readily. 5ft. 5in. Height - 7st. 9½lbs. Weight

Urinary System 
Subjective symptoms, thirst and polyuria as in history.
Urine. Acid. 1030. Sugar --. Acetone --. Nil else to note. Quantity - 70 ozs. per diem.

Alimentary System.
Subjective phenomena as in history.
Tongue - moist and furred. Teeth - two septic stumps in lower jaw. Rest in poor condition.
Breath contains a trace of acetone.

**Abdomen.** Moves fairly freely on respiration. Very little subcutaneous fatty tissue.

Some tenderness just to the right of, and above, the umbilicus. Liver and spleen not enlarged.

**Circulatory System.**


Heart. Apex beat in fifth space, inside the nipple line.

No enlargement on percussion.

Sounds closed and pure in all areas.

**Haemopoietic System.**

No enlarged glands. Spleen not enlarged.

Blood Wassermann reaction negative.

**Respiratory System.**

No subjective phenomena.

Resps. 20/min.

Chest. Inspection - well developed. Expansion is good.

Palpation - movements good and equal on both sides.

Vocal fremitus is normal.

Percussion note is resonant all over chest.

Auscultation - Breath sounds are vesicular. No accompaniments. V. R. normal.

**Nervous System.**

No coma. No tremor.

Superficial reflexes present and normal.

Deep reflexes present and normal.

Organic reflexes - normal.

No sensory abnormalities. Pupils react normally.

**Special Examinations.**

Blood Sugar test meal.-

8 a.m. - 222 mgms. %  9 a.m. - 400 mgms. %

10 a.m. - 364 mgms. %  11 a.m. - 285 mgms. %

**TREATMENT.**
TREATMENT.

Patient was put to bed on admission, and kept on ordinary diet for the first two days.

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<tr>
<td>16th.</td>
<td>C17.</td>
<td>-ve. tr.</td>
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Progress Notes.

Temperature remained normal throughout.

Pulse on admission 90/min. - reduced to 74/min. on 24th.

Patient felt very much better after a few days on a diet. Appetite remained good. Polyuria still present.

Weight - 7st. 9½lb. on admission.

7st. 11½lb. 12 days after admission.
7st. 10½lb. 19 days after admission.
7st. 12 lb. 26 days after admission.

Allowed up on the 21st. day. No ill effects. Feeling much better.

Discharged on the 27th day much improved, on a 2600Calorie diet.
DIABETIC DIETS.


**Breakfast:**
- Bacon 60gms. (2oz.)
- Tomato or other 5% vegetable 100gms. (3½oz.)
- Oatcake 30gms. (1oz.) - or porridge, 3 tablespoons.
- Butter from ration.
- Milk from ration. Tea or coffee.

**Dinner:**
- 5% vegetable 200gms. (7oz.). Butter from ration.
- Lean meat 60gms. (2oz.).
- Orange or other 10% fruit 100gms. (3½ozs.). peeled weight.
- Oatcake 37gms. (1½ozs.). Butter from ration.
- 5% vegetable 100gms. (3½ozs.).
- 1 egg.
- Milk from ration. Tea.

**Tea:**
- Oatcake 30gms. (1½ozs.). Butter from ration.
- 5% vegetable 160gms. (3½ozs.).
- 1 egg.
- Milk from ration. Tea or coffee.

**Supper:**
- Oatcake 30gms. (1½ozs.). Butter from ration.
- 5% vegetable 160gms. (3½ozs.).
- 1 egg.
- Milk from ration. Tea or coffee.

**Rations for day** - Milk 90gms. (3ozs.). Butter - 50gms.
- Clear soup, bovril or oxo, not more than 2 cups daily.
- Water, 4 glasses daily.

CHO 0.56. Prot. 0.54. Fat 1.40. Calories 1700. Total "G" 101. G:F:A. ::1::1.5

**Breakfast:**
- Bacon 45gms. 1 egg. Butter from ration to fry.
- Tomato or other 5% vegetable. 100gms.
- Oatcake 15gms.
- Milk and cream from ration. Tea or coffee.

**Dinner:**
- 5% veg. 200gms. Butter from ration.
- Lean meat 45gms.
- Orange or other 10% fruit 100gms. peeled weight.
- Oatcake 15gms. Butter from ration.
- 5% veg. 100gms.
- 1 egg.
- Milk and cream from ration. Tea or coffee.

**Tea:**
- Repeat as for Tea.

**Supper:**
- Repeat as for Tea.

**Rations for day** - Milk 100gms. Cream (thin) 75gms.
- Butter or fat of meat 82gms.
- Fluids as above.

CHO 0.63. Prot. 0.56. Fat 1.59. Calories 1907. Total "G" 111. G:F:A. ::1::1.5

**Breakfast:**
- Bacon 45gms. 1 egg. Butter from ration to fry.
- Tomato or other 5% veg. 100gms.
- Oatcake 23gms.
- Milk and cream from ration. Tea or coffee.

**Dinner:**
- 5% veg. 200gms. Butter from ration.
- Lean meat 45gms.
- Orange or other 10% fruit 100gms.
- Oatcake 22gms. Butter from ration.
- 5% veg. 100gms.
- 1 egg.
- Milk and cream from ration. Tea or coffee.

**Tea:**
- Repeat as for tea.

**Supper:**
- Oatcake 15gms. Butter from ration.
- Rest as for tea.

**Rations for day** - Milk and cream as for C17. Butter or fat of meat - 103gms.
- Fluids as above.

CHO 0.31. Prot. 0.62. Fat 1.75. Calories 2107. Total "G" 124. G:F:A. ::1::1.5

C. - 21.

Breakfast: Porridge 3 tablespoons or oatcake 30gms.
Cream from ration.
Bacon 45gms. 1 egg. Butter from ration to fry.
Tomato or other 5% veg. 100gms.
Milk from ration. Tea or coffee.

Dinner: 5% veg. 200gms. Butter from rations.
Lean meat 60gms.
Orange or other 10% fruit 100gms, peeled weight.
Cocktail 22gms. Butter from ration.
5% veg. 100gms.
1 egg.
Milk and cream from ration. Tea or coffee.

Tea: Same as for tea.

Supper: Rations for day - Milk 100gms. Cream (than) - 75gms.
Butter or fat of meat 118gms.
Clear soup, oxo or bovril not more than two cups daily.
Water four glasses daily.


Breakfast: Porridge 3 tablespoons or oatcake 30gms.
Cream from ration.
Bacon 60gms. 1 egg. Butter from ration to fry.
Tomato or other 5% veg. 100gms.
Milk from ration. Tea or coffee.

Dinner: 5% veg. 200gms. Butter from ration.
Lean meat 60gms.
Orange or other 10% fruit 100gms, peeled weight.
Cocktail 30gms. Butter from ration.
5% veg. 100gms.
1 egg.
Milk and cream from ration. Tea.

Tea: Same as for tea.


Breakfast: Porridge 3 tablespoons, or oatcake 30gms.
Cream from ration.
Bacon 60gms. 1 egg. Butter from ration to fry.
Tomato or other 5% veg. 100gms.
Milk from ration. Tea or coffee.

Dinner: 5% veg. 200gms. Butter from ration.
Lean meat 60gms.
Cocktail 15gms.
Orange or other 10% fruit 100gms peeled weight.
Cocktail 30gms. Butter from ration.
5% veg. 100gms.
1 egg.
Milk and cream from ration.

Supper: As for Tea.

Rations for day - Milk and cream as above. Butter or fat of meat - 120gms. Olive oil - 22gms. Fluids as above.
C. - 28.

### Breakfast:
- Porridge four tablespoons or oatcake 40gms.
- Cream from ration.
- Bacon 60gms. 1 egg. Butter from ration to fry.
- Tomato or other 3% veg. 100gms.
- Milk from ration. Tea or coffee.

### Dinner:
- 5% veg. 200 gms. BUTTER from ration.
- Oatcake 15gms.
- Orange or other 10% fruit 100gms. peeled weight.
- Olive oil from ration.

### Tea:
- Oatcake 45gms. BUTTER from ration.
- 5% veg 100gms.
- 1 egg.
- Milk and cream from ration.
- Olive oil from ration.

### Supper:
- Same as for Tea, without olive oil, and only 35gms, oatcake.

---

Rations for day - as in C. - 25, but with 45gms olive oil.

<table>
<thead>
<tr>
<th>3% vegetables (take the full quantity)</th>
<th>10% vegetables</th>
<th>15% fruit</th>
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<tbody>
<tr>
<td>Lettuce</td>
<td>Veg. marrow</td>
<td>Water cress</td>
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<td>Spinach</td>
<td>Asparagus</td>
<td>Seakale</td>
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<tr>
<td>Celery</td>
<td>Cucumber</td>
<td>Eggplant</td>
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<tr>
<td>French beans (young)</td>
<td>Rhubarb cooked with a pinch of baking soda</td>
<td>Leeks</td>
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<td>Radishes</td>
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<td>Broccoli.</td>
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<td>Tomatoes</td>
<td>Endive</td>
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<td>Brussels sprouts</td>
<td>Mushroom</td>
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<td>Cauliflower</td>
<td>Cabbage</td>
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6% vegetables (take half the quantity)

<table>
<thead>
<tr>
<th>Carrots</th>
<th>Turnip</th>
<th>Onions</th>
<th>Beetroot</th>
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<table>
<thead>
<tr>
<th>2½% fruit</th>
<th>10% fruit</th>
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<tr>
<td>Watermelon.</td>
<td>Cranberries.</td>
<td>Apple</td>
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<tr>
<td>Lemon</td>
<td>Orange</td>
<td>Pear</td>
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<td>Strawberries</td>
<td>Fresh peach</td>
<td>Fresh cherries</td>
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<tr>
<td>Grapefruit</td>
<td>&quot; pineapple</td>
<td>&quot; raspberries.</td>
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<td></td>
<td>&quot; gooseberries</td>
<td>&quot; apricots</td>
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<td></td>
<td>&quot; blackberries</td>
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132gms. (4½ ozs.) 100gms. (3½ ozs.) 66gms. (2½ ozs.)

Admitted - 6.3.32.

Discharged -

Complaint:  1. Septic thumb - 7 days.

2. Feeling drowsy and tired - 3 days.

3. Vomiting - 2 days.

History:  Patient was in R.I.E. six years ago with diabetes mellitus. She has been treated dietetically since, and has never had insulin. Her urine is always sugar free unless she breaks her diet, or is suffering from some intercurrent infection. On the whole she has kept well to her diet; and has kept strictly to it for the last six months.

A week ago she developed a septic thumb. The abscess was opened five days ago and pus has been discharging since. She has been feeling very drowsy and tired for the last three days, and has vomited practically everything she took.

Family History: Father and mother died of old age. Husband alive and well. Has had four children, two of whom are alive and well; the other two died twenty-four hours after birth, cause unknown. Number of pregnancies four; number of full-time labours four.

Home conditions: good.

Abstract from Previous History.  October, 1925.

Admitted complaining of a. Bad taste in mouth; b. loss of weight; c. general weakness; d. polyuria. Duration - 3 years.

Three years before she developed a whitlow of the thumb, and went to her doctor, who found sugar in her urine. She began to lose weight and was put on a special diet, but did not adhere to it. She does not have undue thirst. She is not constipated and does suffer from pruritus vulvae. She has had no previous illnesses.

She was treated dietetically, and sent out on C 22 diet and no insulin, after three and a half months in hospital. Much improved.

EXAMINATION.

On admission patient was in a profound coma. There was marked air-hunger. The limbs were flaccid, blood pressure low, and intra-ocular tension/
tension was diminished. Her breath smelt strongly of acetone.

On recovery. She is above the average in intelligence. Her muscular development is poor. There is marked loss of subcutaneous fat.

Height - 5' 2½". Weight

There is no obvious morbid appearance. No anaemia.

**Urinary System**

Subjective symptoms as in history.

Kidneys - not palpable. No tenderness in kidneys angles.


No other abnormal constituents.

**Alimentary System**

Appetite is generally good. No marked thirst. Vomiting for the past few days after food.

Tongue - slightly furred, moist, and tremulous.

Teeth - artificial denture.

Nothing abnormal found in abdomen. Liver and spleen not enlarged.

**Cardio-Vascular System**

Pulse. 100/min. Regular. Sharp up-stroke, poorly sustained, quick down-stroke. B.P. 92/64.

Heart. Apex beat in 5th, left interspace, internal to the mid-clavicular line. Right border is at the right sternal margin.

Sounds closed in all areas, no murmurs. First mitral sound rather short.

**Haemopoietic System.**

Leucocytosis of /$.5$.0.

**Special Examinations.**

<table>
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<tr>
<th>Date</th>
<th>Blood Sugar (7 a.m. spec.)</th>
<th>CO combining power</th>
<th>Date</th>
<th>Blood Sugar (7 a.m. spec.)</th>
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</thead>
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<td>6. 3. 32;</td>
<td>667 mgms.%</td>
<td>12.30 p.m.</td>
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<td>444 mgms.%</td>
<td>1.30 p.m.</td>
<td>52.5 vols. %</td>
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<td>24. 3. 32.;</td>
<td>200 mgms.%</td>
<td>2.30 p.m.</td>
<td>59 vols. %</td>
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<tr>
<td>25. 4. 32.</td>
<td>(12.30 p.m. spec.) - 167 mgms. %</td>
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<td>65 vols. %</td>
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<td></td>
<td>(1.30 p.m. spec.) - 150 mgms. %</td>
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<td>52.5 vols. %</td>
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<td></td>
<td>(2.30 p.m. spec.) - 200 mgms. %</td>
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<td>59 vols. %</td>
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</table>
Special Examinations.

Blood cholesterol - 120 mgms.%.

Progress Notes.

With glucose and insulin patient was brought out of her comatose condition. For details see the treatment sheet. Thereafter she showed a swinging temperature, (see chart), due to a succession of subcutaneous abscesses in addition to the whitlow of the right thumb. These were successively in the back, left infra-mammary region, and left arm. They were opened in turn under local anaesthesia. The infecting organism was found to be staphylococcus aureus. Clinically she was drowsy and disinterested, though her urine was free of acetone. Finally she developed erysipelas, and was sent to the Fever Hospital along with detailed diet sheets.

She returned from the City Hospital on 15. 4. 32 recovered of the erysipelas. She developed several more abscesses and it was decided to transfer her to the surgical side of the House to have them opened. This was done, and she was operated on on 24. 5. 32, under G. and L. anaesthesia, and the abscesses opened. Since then she has remained on the surgical side and has improved somewhat.
The diagnosis is rarely difficult, as one easily determine if there is sugar in the urine by applying the appropriate tests. Of these Fehling's test is the easiest and most widely used, and gives perfectly reliable qualitative results. Alkapton reduces Fehling's sol. but is optically active and does not ferment. Other reducing sugars, e.g. laevulose, lactose and pentose, and the breakdown products of various drugs may be possible sources of error. As regards the former, the phenyl-hydrazine test is very useful in distinguishing between them. Finally it should be remembered that Fehling's sol. is unstable if kept for any length of time, and is reduced spontaneously on heating. One should therefore always heat the urine and reagent separately and add one to the other when boiling.

The presence of ketone bodies is diagnostic of diabetes when sugar is also present.

It is when glucose only is found in the urine that mistakes are apt to occur. Other causes of glycosuria are a. excitement or emotion, due to the action of adrenaline on the glycogen stores of the liver. This form of glycosuria is very transient. b. Alimentary glycosuria is produced by the ingestion of a large amount of glucose. The liver is unable to deal with it all and the overflow is excreted. This also is transitory. c. Renal - due to a lowering of the renal threshold for glucose. This may be normal or be produced by the drug phloridzin. The determination of the fasting blood-sugar level is of importance in distinguishing between these forms of glycosuria. It is raised in diabetes mellitus, a reading higher than 120mgms% being regarded as meaning diabetes. In the other forms of glycosuria, the fasting blood sugar is 80mgms% or less. A sugar tolerance test meal is of value as was carried out in Case iii.

The individual symptoms, namely loss of weight, thirst and polyuria may lead to some trouble if the patient only complains of one of them; but careful questioning in cases of moderate severity will almost always elicit these three symptoms. The presence of a healthy appetite in spite of the loss of weight is a strong guide to the diagnosis.

Early cases often provide great difficulty in diagnosis, but in doubtful cases an estimation of the fasting blood sugar, with results
as above, and the sugar tolerance is sufficient to settle the diagnosis.

**Diabetic Coma.**

Two of these cases arrived in hospital in an unconscious condition, and one could therefore get no help from them as regards their history and previous symptoms. Fortunately they were both accompanied by a note from their family physicians, which pointed to a provisional diagnosis of diabetic coma. The smell of acetone in the breath, and the typical air-hunger, made the diagnosis fairly certain; and this was clinched by a diagnosis from an examination of the urine in each case, when it was found to be loaded with sugar and acetone. The reaction to insulin and the fasting blood sugar figure merely helped to strengthen the diagnosis.

A patient may however arrive in hospital, or be met with in practice, in an unconscious condition, and one may have no guide history. One has therefore to consider the various possibilities, and they may be listed as follows: 1. diabetic coma, 2. uraemic coma, 3. eclamptic coma, 4. alcoholic coma, 5. Adams-Stokes syndrome, 6. cerebral apoplexy, 7. epilepsy. The following points should be attended to before arriving at a diagnosis:

1. Smell the breath and notice the type of breathing, whether sighing or stertorous.
2. Take the blood-pressure, and the pulse rate.
3. Examine the urine - chemically and microscopically.
4. Examine the limbs as regards flaccidity or rigidity, and the condition of the reflexes.
5. The state of the pupils should be noted and the temperature taken.
Osler defines the condition as a disease of metabolism, especially of the carbohydrates, in which the normal utilization of carbohydrate is impaired, with increase in sugar content of blood, and consequent glycosuria. There is a tendency to subsequent disturbance of the fat metabolism with resulting ketosis.

The three cases chosen are all typical examples of diabetes mellitus. It is a disease which has been known to the medical profession for many centuries, Araeetus being the first to call it by this name. He described it as a wonderful affection "melting down the flesh and limbs into urine." In 1776, Dobson found sugar in the urine; and in 1789, Rollo wrote an admirable account of it and recommended a meat diet. It is, however, from Bernard's discovery of the glycogenic function of the liver that the modern conception of the disease has developed.

The etiology of the condition is obscure, and none of the cases described was any definite point discovered that would suggest the cause, except possibly in Case iii. She suffered originally from local sepsis about the hands, and it was when she went to her doctor to have a whitlow attended to that the diabetic condition was discovered. In passing, it may be noted that this illustrates an important practical point in the treatment of such cases of sepsis; one should always test their urine for sugar. What relation this sepsis had to the diabetes it is difficult to say. It may however have been that some septic condition, not necessarily this whitlow, that was at the root of the trouble, a focus being present in the body from which toxins and organisms entered the blood, circulated and finally settled in the pancreas. Such an explanation is suggested in Case ii also from the condition of the teeth, a definite pyorrhoea being present. The septic focus theory has become rather discredited recently, Owino probably to its being invoked to explain most of the diseases to which man is heir; but in diabetes at any rate no other more satisfactory explanation has been given.

It is generally said that obesity is, or has been, a prominent feature of the diabetic, but it cannot be said that any of these cases illustrate this point. Heredity also is said to play a large part in the etiology, but none of these cases confirmed this either, there
being no family history of diabetes in any of them.

The immediate cause of the disease is an insufficiency of the secretion of the islets of Langerhans in the pancreas. Mering and Minkowski were the first to show, in 1889, that when they removed the pancreas of a dog, it developed severe diabetes. In 1900, Schultze tied the pancreatic duct, and this led to atrophy of the acinar tissue, but not to diabetes, showing that the primary fault, whatever it was, did not lie in that part of the gland. In 1916, Sharpey-Schafer suggested that the maintenance of a normal carbohydrate metabolism depended on the formation, by the islets of Langerhans, of a hormone, which he named Insulin; and that a deficiency of absence of the hormone produced diabetes.

Pathology. Macroscopically, there are no changes in the pancreas, nor are there any constant microscopic changes except for a change in the islet cells. There may be a chronic interstitial fibrosis, suggesting a chronic inflammatory process, but frequently there is nothing. The islet cells have been shown to be of two kinds, classed according to the staining reactions of their granules, namely, a-cells containing oxyphil granules, and b-cells containing basophil granules. It is in these b-cells that the most constant changes are seen in diabetes, the cells becoming swollen and the granules disappearing (Allen). It is therefore suggested that these cells form the insulin. Lane found that if dogs, in which most of the pancreas had been removed, and which were on the threshold of diabetes and had a low carbohydrate tolerance, were given too much carbohydrate, they developed diabetes, and the characteristic changes were found in the b-cells. By reducing the carbohydrate and carefully dieting them, it was sometimes possible to get the b-cells to resume their function, so that the animal returned to the former threshold state. He based his system of dieting on this pain of allowing the islets as much rest as possible, and giving them every chance to recover (see later).

Clinical Manifestations. The most prominent symptoms, as was well illustrated in this series, are thirst, polyuria, and loss of weight. These taken together are almost diagnostic, but frequently one symptom only may be complained of, and it requires a little more care to diagnose the condition. The explanation of these three symptoms is simple.
The thirst is naturally due to the polyuria, as the more fluid that is excreted the more is required to take its place; and also a larger amount of fluid is required in the body to hold the sugar in solution. The large amount of urine that is passed is necessary in order to hold the sugar that is being excreted in solution. While the loss of weight is due to the rapid mobilisation and wasting of the glycogen of the body tissues, and also to the excess metabolism of body fats that occurs in order to provide the body with the energy that is normally supplied by the carbohydrate metabolism.

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Itching of the skin generally and the pudenda in particular was not present in any of these cases, but it is a fairly frequent symptom. The pudendal itching is due to the irritation of the sugar laden urine, while the general itching is probably due to the excess of glucose in the blood causing irritation of the subcutaneous tissues.

A good appetite is a feature of the majority of the cases, and in some there is a marked increase in the appetite. This is associated with the rapid loss of carbohydrate from the body.

Glycosuria. The diagnostic sign however is glycosuria. This may occur in a number of other conditions, as has been pointed out already, but the vast majority of cases are diabetes. The urine normally always contains traces of reducing substances, corresponding to .03 to .1% glucose, though whether they are glucose or not has not so far been demonstrated. This amount will not reduce the ordinary reagents, but amounts of .2% glucose and upwards do, and the condition is then termed glycosuria. The sugar present in the urine of diabetics is invariably glucose, as can be shown by the phenyl-hydrazine test, in which glucosazone crystals are formed. Its presence is usually demonstrated by its making use of its reducing action, various reagents being employed, e.g. Fehling's and Benedict's. The amount present in the urine can be estimated by these reagents also, Benedict's reagent being perhaps the one most commonly employed. This latter determination has, however, tended to fall out of use of late, owing to the ease with the blood-sugar content can be estimated. The high specific gravity of the diabetic urine is due to the presence of the glucose in solution.

Glycosuria depends on two things: 1. The sugar content of the blood and 2. the renal threshold. Anything that increases the first to a level...
Fig. 1. Normal Blood Sugar Curve.

Fig. 2. Blood Sugar Curve in Diabetes (Case 2).
above the renal threshold, or reduces the latter below the blood-sugar level, causes glycosuria. It is the former condition that is present in diabetes mellitus, i.e. a hyperglycaemia.

**Hyperglycaemia.** A persistent rise in the blood-sugar level is the earliest indication of diabetes. Normally the only sugar in the blood is glucose, and it varies amount from 80 to 180 mgms.%. It is an equilibrium mixture of the two stable forms, a-glucose and b-glucose.

Even in a normal person however the blood-sugar level is not constant, for it varies with the amount of carbohydrate ingested, as is shown by the blood-sugar curve. To determine this, the fasting blood-sugar is determined and then 50-100 gms. glucose dissolved in water, is taken by mouth. 5cc. of blood is drawn off at intervals of one-half or one hour, and the sugar content estimated. From these estimations a curve can be constructed, as in fig.1. There are various methods in use for determining the blood sugar content, notably those devised by Benedict, and Folin and Wu. It is the former, a colorimetric method, that was used in the estimations made in this series of cases, the sugar content of venous blood being estimated. Recently, however, Herbert and Bourne have pointed out that there may be quite an appreciable difference in the sugar content of arterial and venous blood, amounting in some cases to as much as 80mgms.%. It is the sugar content of arterial blood that is of importance, since it is arterial blood that passes through the renal glomeruli. It will be seen therefore that such a difference may cause mistakes, particularly in the diagnosis between renal and pancreatic diabetes. They describe a method, which is a modification of the Folin-Wü method, using capillary blood, as they have found that arterial blood does not lose any appreciable amount of glucose in passing through the skin.

It will be seen from the curve in fig.1, that one-half hour after the ingestion of glucose the blood-sugar rises to 140-160 mgms.%- owing to some of the absorbed sugar passing through the liver into the systemic circulation. During the next hour the blood-sugar falls steadily to its previous level, or it may even fall below normal owing to an excess secretion of insulin. In an early case of diabetes, the fasting blood-sugar is higher, the initial rise is greater and it does not disappear so rapidly, the initial high level continuing for an hour or
more and falling only slowly. Fig. 2, which is the blood-sugar curve from Case ii, illustrates this very well, though it is a moderately severe case as is shown by the high Fasting blood-sugar. In advanced cases of diabetes the renal threshold for sugar, normally 180 mgms.%, appears to be raised. This is well illustrated in Case iii, in whom a specimen of urine, taken one hour after the blood-sugar level was determined and found to be 200mgms.%, was sugar free.

If any complications occur in the course of the disease, even a simple rise in temperature, the blood-sugar level tends to rise at once and coma may supervene. This point is well illustrated in Cases i and iii in the former. A rise of temperature to 102°F caused a rise in the blood-sugar from about 150mgms.% to 400mgms.%, while in the latter a septic thumb sent her into coma.

**Metabolism in Diabetes.** Clinical experience has shown that it is essential, when administering insulin, to secure a proper balance between the amount of insulin administered and carbohydrate ingested, as otherwise glycosuria or hypoglycaemia will result. In the normal functioning of the pancreas therefore, since neither of these effects ensues, there must be some means whereby the amount of insulin liberated from the islet tissue corresponds with the amount of carbohydrate to be dealt with. In diabetes however the insulin is absent, with resulting hyperglycaemia and glycosuria. But this also occurs in starving animals from whom the pancreas has been removed, and who are not getting any carbohydrate to excrete. The glucose which they are excreting therefore must be endogenous in origin, and comes either from the glycogen stores in the body, or from a breaking down and conversion of other body substances, namely the proteins and possibly the fats. It will thus be seen that in the absence of insulin there are two processes at work, a. failure to store glucose as glycogen; and b. faulty metabolism of the glycogen already in the body, it being broken down to glucose but no further. This latter point is confirmed by a study of the Respiratory Quotient in diabetes, which is lowered, and in severe cases to that of fat (0.7).

Protein metabolism is also seriously interfered with, much of the protein being broken down transformed into glucose and excreted as such. This is effected in the liver, hypoglycaemia resulting in a starved
diabetic animal if the liver is removed.

The metabolism of fat is also seriously disorganized. The R.Q. in a severe case indicates that only fat metabolism is occurring; but this is not a normal metabolism. Normally the stored fats, when required for use, are desaturated, i.e. a double bond is put in in the middle of the fatty acid carbon chain, two or more H atoms being removed, this occurring in the liver. Then the fatty acid molecule undergoes oxidation, this occurring by the oxidation of the carboxyl group and the a-carbon atom, while the b-carbon atom is oxidised to a carboxyl group. This the well-known a-oxidation of Knoop, Dakin, and Embden. Normally, fatty acids can be oxidised in this way spontaneously, as it were, to a four carbon chain - butyric acid; but after this stage carbohydrate is required for their proper oxidation to H2O and CO2. In diabetes, however, there is no carbohydrate for them, and so the fat catabolism stops at this point with the formation of ketone-bodies, namely b-oxybutyric acid, diacetic acid and acetone. These are excreted in the urine of diabetic patients, and are known as the acetone bodies. Acetone was present in all three cases in this series.

These substances are acid and therefore tend to reduce the alkali-reserve of the blood. If they become too abundant a condition of "acidemia", or more correctly "reduced alkali reserve" appears, with resulting diabetic coma.

Diabetic Coma. This condition was present in two of the cases in the series when admitted to hospital. In Case iii the immediate cause was undoubtedly the sepsis in the finger, for as we have seen, sepsis is a potent factor in raising the blood-sugar level with consequent increasing failure of carbohydrate metabolism. In Mrs. R's. case, the cause is not so obvious, though it is quite liable to occur in advanced cases without any precipitating factor. There is one point however that may be of importance. It is well known that, in diabetic women, during menstruation larger doses of insulin are required to keep the blood sugar down, owing to the antagonistic action of the ovarian hormone of menstruation. Now when Mrs. R. was admitted in coma, she was menstruating, and continued to do so for some days thereafter. Further, on the first day of her next menstrual period, she developed a temperature and the blood-sugar suddenly rose, which may
or may not have been connected with her menstruation. It is reasonable to suggest therefore that the precipitating factor in her case was the onset of menstruation.

In both cases the urine was loaded with sugar and acetone and the latter could be smelt in their breath.

Diabetic coma is a medical emergency, and one that is to be met with anywhere. It behoves every medical man, therefore, to have a sound knowledge of the treatment of such a case. The only cure for diabetic coma is insulin, and as Joslin says, "Insulin cures coma unless the patient is moribund." The description of the treatment given in the two cases is typical and needs no repetition. The pint of importance in giving insulin is to have it covered with a sufficiency of glucose, 1.5-2gms. per unit, in order to avoid hypoglycaemia. Once the patient is brought out of coma and the urine is sugar and acetone free, the treatment is the same as for an ordinary case of diabetes.

**Treatment of Diabetes.** The principles of the treatment are:

1. To rest the pancreas by giving less food for a time.

2. To give foodstuffs in proportions that make the chemical work of the body as easy as possible, so that all that is taken in is properly oxidised and not wasted as sugar or ketone bodies with harm to the tissues. Favourable proportions are carb. 1 : protein 1.5 : fat 3. Carbohydrate may be higher with advantage as regards the risk of ketosis but not lower. The diet should contain not less than 4-1 gm. protein for each kilogram body weight and not much more.

3. To make sure that these results are obtained by showing that the urine is free from sugar and diacetic acid and that the blood-sugar is at or near normal.

4. If these objects are not obtained by diet alone, to use insulin without further delay.

Treatment may thus be dietetic alone or dietetic with insulin.

There are two methods of dieting:

1. **Fasting Method.** Allen found that fasting reduced the blood-sugar to or near normal, with disappearance of sugar from the urine, and also that it reduced ketosis rapidly. He therefore advocated
starvation for a period as a preliminary to dietetic treatment. If
the diet is then increased, step by step, these results may be main-
tained, and a suitable ultimately found that keeps the symptoms in
check and yet lets the patient lead an active life. This method is
falling out of favour as it tends to waste time.

2. Basal Ration Method. This is the method employed in this series.
The Basal ration is calculated on a basis of 25Cals. per kgm. body
weight, this giving the average Basal metabolic rate. For a person of
70 kgms, this is therefore about 1700Cals per day. A diet of this
value is given and the patient kept on it for a few days, until the
urine is sugar and acetone free, giving insulin if required. Once the
urine is clear the ration is increased step by step, usually 100-200
Cals at a time, insulin being employed if necessary to keep the urine
free, until a diet is reached of a sufficient calorific value as to
enable the person to perform his ordinary duties. This varies from
person to person, as is seen in Cases i and ii. The former was sent
out on a 2087Cals diet, while the latter, a man and therefore requiring
more energy, was sent out on 2600Cals. The length of time required for
also stabilization varies from case to case, depending on the severity of
the case and the amount of insulin administered. Mrs. R., who came in
coma took fifteen days to become sugar free; and was then getting
30 units of insulin daily; while Case ii. took 14 days and was getting
15 units daily. Case iii took 10 days and was getting 50 units.

The diet sheets appended are typical samples of the diets
used in these cases. They illustrate very well the graduated rise in
the calorific value of the successive diets, which is obtained chiefly
by increasing the fat and protein content. The guiding principle in
making up such a diet is to get a proper balance between the ketogenic
and anti-ketogenic foods, i.e. between the fats, and the carbohydrates
and proteins. It is found that this is most accurately obtained when
the fat content (F) of the diet does not exceed twice the carbohydrate
(2C) and half the protein (P/2). \[ F = 2C - \frac{P}{2} \]

In calculating the glucose content of such a diet it must be
remembered that the body obtains glucose from fats and proteins as
well as carbohydrates. Such being the case, it is found that all the
carbohydrate, 60% of the protein, and 10% of the fat must be regarded as equivalent to glucose. The glucose value of each diet is appended at the top of each diet.

Insulin Therapy. Many patients do not need insulin. As Hutchison puts it: The test is:—Can enough food for the daily needs be taken, without sugar appearing in the urine, and without an unduly high fasting blood-sugar, say about 170-180 mgms. %? If not insulin should be used. If therefore on the basal ration, preceded if advised by a fast for one day or a low diet, sugars appears, or if after getting the urine sugar free and on raising the diet, sugar again appears in the urine or the blood-sugar rises, then give insulin.

Complications Frequently arise in diabetics in the form of various infections, and these have to be treated also. This is well illustrated in Case iii, who in addition to suffering from abscesses, was also afflicted with erysipelas. In the latter condition, strict attention to the diet and insulin is sufficient. In the case of abscesses, however, if they become large, as they did here, surgical intervention is required. In such a case anaesthesia is necessary. In Caseiii, operation was performed on 25. 4. 32 and the blood sugar figures were determined before nitrous oxide and after the operation. The anaesthetic used was gas and oxygen. The first two figures are the figures before operation, when the urine was sugar free. She received 20 units of insulin before operation. The third reading gives the content immediately after operation, the urine being again sugar-free. This indicated the value of this type of anaesthesia for such operations in diabetics, for although a rise did take place it was not very marked. The anaesthesia was maintained for about 15 minutes.

Prognosis. In the first two cases, the prognosis is quite good provided that they follow the instructions they have received and stick to their and insulin. If they do not do so however, they will just relapse, and with each relapse will become worse until finally they become so weakened, that they cannot resist any infection with which they may come in contact, and die of ti. Both however were fairly intelligent, and so there is some hope that they will da, as they are told.
Case iii however presents a different picture altogether. She undoubtedly has a septicaemic or pyaemic condition, as is shown by the abscesses appearing in such widely differing regions. She will therefore go downhill rapidly, and the end cannot now be long delayed.