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by

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The selection of a few cases from all those seen on medical wards was difficult. It was thought desirable that the cases be related to one another, but this was almost impossible in practice. These reported here have in common only the chest as their source of symptoms. Otherwise the choice of cases has been governed by their great interest to me personally.

Case No. 1.

Mrs. S., from Edinburgh, a 61 year-old housewife, who was happily married and who lived in a two-roomed tenement flat.

HISTORY. This lady presented with intermittent weakness in both legs. The attacks had begun three years previously and had been frequent since. Usually they were brought on by walking and disappeared after a few minutes rest. Mrs. S. had never actually fallen and claimed that she had no difficulty climbing stairs.

These episodes of leg weakness were accompanied by parasthesiae in the right leg and a peculiar fluttering sensation over the left eye. There was no loss of vision but focusing was sometimes difficult. The attacks did not come on when Mrs. S. was with someone.

Light-headedness was frequent and she had severe headaches, almost every day, and always in the left frontal region. There was often a tingling sensation in the left arm, especially after exercising the arms. All of these symptoms were precipitated by excitement or fear. Mrs. S. had never lost consciousness. She had no diplopia, vertigo, or tinnitus. No nausea or vomiting. Swallowing, speech and hearing were good. No other symptoms could be obtained.

Mrs. S. did not smoke or drink. She had had no children. Her father died in diabetic coma. Her brother and sister had both suffered from tuberculosis.

EXAMINATION. A rather obese lady who seemed fairly intelligent and sensible. No cyanosis, anaemia, oedema, finger-clubbing, or lymph gland enlargement.

Cardiovascular system.

Pulse : 80/min. Regular in time and force; good volume; vessel wall palpable; no radial pulse detected on left; all other pulses present; femorals both strong and equal in time with right radial.

Apex-beat not palpable even after exercise.

Blood pressure : 130/90 on the right, but unobtainable on the left.

Jugular venous pressure not increased.

Heart : no thrills; first and second sounds normal and heard all over; systolic bruit in aortic area and in right side of neck.

Nervous system.

Despite a thorough examination, the only abnormality detected here/

here was an increase in the left ankle and knee jerks.

No other physical signs were elicited.

DISCUSSION OF FINDINGS.

The history is a rather confusing one at first sight, suggesting some kind of neurological lesion or the need for psychiatric help for this lady. Examination was of little help but as it indicated the presence of a vascular lesion tended to favour the former as the cause of symptoms. The absence of a radial artery was not necessarily significant, but the fact that a measurement of blood pressure could not be obtained from the same arm suggested that an aberrant artery was not the answer and that this finding was relevant.

INVESTIGATIONS.

1. Haematology : Hb. = 95%; W.B.C. = 9,000 per cmm.; E.S.R. = 4 mm./hr. i.e. normal.
2. W.R. and Kahn : negative, indicating that syphilis was probably not involved.
3. Chest X-ray : nothing abnormal.
4. Skull X-ray : no evidence of intracranial lesion.
5. Cervical spine X-ray : no bony abnormalities, no undue narrowing of disc spaces.
6. Arch aortogram : after injection of the radio-opaque dye a good demonstration of the right innominate, right common carotid, right vertebral, and left common carotid arteries was obtained, but there was no filling of the left sub-clavian. When the dye had almost disappeared from the other large vessels a little did appear in the left sub-clavian and very slow filling took place. The blood did not come from the direction of the origin of the artery but from its branches. Reversed flow was clearly seen in the vertebral and other branches.

DIAGNOSIS.

This is an example of the sub-clavian steal syndrome, which is a very rare condition.

As was shown by the aortogram the left sub-clavian was blocked at or near its origin. It was, therefore, from the anastomoses between the branches of the sub-clavian and the branches of the other arteries that the left arm received its blood supply. The case of the rather bizarre neurological symptoms was obvious. When Mrs. S. was relaxed and at rest the anastomoses were more than adequate to furnish the arm with blood. When, however, the arm was exercised or Mrs. S. became emotionally upset, the anastomotic channels had to supply more blood and were taxed to the limit. One such channel was via the vertebral artery taking blood from the Circle of Willis. When much blood was drained from this there was relative ischaemia of the brain giving rise to the neurological signs.

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The tingling of the left arm is more likely to have been due to ischaemia of the arm itself rather than to ischaemia of the appropriate region of the brain.

TREATMENT : Three forms of treatment are available:-

1. To do nothing. This seemed undesirable as the patient found her symptoms very distressing, as the nature of the blockage would remain in doubt, and as permanent brain damage would occur.

2. To tie off the vertebral artery. This would be a smaller operation, with less risk to the patient, than 3. The disadvantages were that tying the vertebral is a difficult procedure, that the brain would remain deprived of the blood from that vertebral, that the arm would be more ischaemic, and that the true nature of the lesion would remain in doubt.

3. To explore the region of the left sub-clavian and attempt to restore the flow between the aorta and the arm. This has none of the above disadvantages but calls for a major operation on a woman of 61 years.

Mrs. S. was transferred to a Thoracic Surgical Unit where the origin of the left sub-clavian was explored. A large mass of organised thrombus was found extending from just within the aorta to the origin of the internal mammary artery. This was removed and flow was then normal, with a good radial pulse on the left.

There were no post-operative complications.

PROGNOSIS.

It is impossible to be sure whether thrombosis or embolism was the original cause of the blockage. In view of the additional findings at operation of much atheroma and a slight kink in the artery, the former seems the most likely explanation. No source of emboli was evident. Given that this was a primary thrombus, it would not be surprising if thrombosis occurred again. If this did happen there would be the same choice of three types of treatment. If a major operation were still feasible then the insertion of a prosthesis in place of the affected length of sub-clavian would seem desirable rather than a simple removal of thrombus.

If there is no re-thrombosis then there should be a permanent disappearance of symptoms.

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Case No. 2.

Mr. E. from Edinburgh, a 71 year-old retired miner who lived with wife in a council house.

HISTORY: This/

HISTORY : This man's only complaint was of changes of his finger-nails over about two months. He felt very well.

On direct questioning Mr. E. admitted having had some breathlessness for ten years. He had occasional cough on waking in the morning and a little spit, but had had no haemoptysis. There was no history of lassitude, anorexia, or loss of weight. There were no symptoms referable to the cardiovascular, alimentary, urinary, or nervous systems.

Mr. E. had been a miner for 52 years. He had had no serious illnesses or operations. He smoked 15-20 cigarettes per day and had done since the age of 13. There was no history of T.B.

EXAMINATION : A large pleasant man who looked very well. There was no cyanosis, anaemia, oedema, or lymph gland enlargement. Very distinct finger-clubbing was present.

Cardio-vascular system.

Pulse : 70/min. Regular in time and force, good volume, wall palpable.

Apex-Beat : not displaced.

No venous engorgement.

Blood pressure : 120/85.

Heart : no thrills, first and second sounds heard all over, no murmurs.

Respiratory system.

No abnormality could be detected. No abnormal signs were found in any other system.

DISCUSSION OF FINDINGS.

Obviously in this case the history and examination give little indication of the presence of any disease. A degree of breathlessness is not uncommon in a man of this age and was not of sufficient degree to make Mr. E. consider himself abnormal. Similarly, a little cough and spit, while not normal, is not uncommon nor of much significance in a smoker and ex-miner of this age.

The one positive sign is the gross finger-clubbing. This is frequently found in patients with certain types of respiratory disease, notably bronchial carcinoma and chronic suppuration such as in lung abscess, empyema, and bronchiectasis. It does not as a rule occur in chronic bronchitis or emphysema unless there is accompanying pulmonary suppuration, and not in pulmonary tuberculosis except in advanced cases. Certain other non-respiratory conditions can, less often, cause clubbing - sub-acute bacterial endocarditis, congenital cyanotic heart disease, and rarely Crohn's disease, malabsorption syndrome, and hepatic cirrhosis. Finally, and very rarely, clubbing can be found in healthy people as a familial trait.

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The mechanism of production of clubbing has yet to be elucidated. One suggestion is that an unknown substance is released from the damaged lung tissue (when the clubbing is respiratory in origin). This substance is said to have vaso-dilator properties and to act in such a way that its effects are only apparent at the fingers although it also affects vessels elsewhere. This remains very much a theory.

INVESTIGATIONS.

Chest X-ray. There was a distinct irregular right hilar opacity which was very suggestive of carcinoma. The remainder of the right lung and the whole of the left showed no abnormality.

Bronchoscopy. Larynx, trachea, main carina, and all the left bronchi were normal in appearance. A vascular friable, nodular mass of tissue sprouted from the right upper orifice, and a biopsy of this was taken. The other right bronchi looked normal.

Biopsy report : A poorly differentiated squamous cell carcinoma.

DIAGNOSIS.

This is that of bronchial carcinoma. The disease has been discovered early in this case. Diagnosis can often be made in this way, i.e. by radiological examination, bronchoscopy, and biopsy, when the tumour is in the hilar region (as in 75% of cases) or when metastasis to hilar lymph glands has occurred.

Sputum examination can be very helpful in early cases - both 'central' and 'peripheral' - but is not yet a practicable proposition as a routine investigation. The technique of the examination is simple but the sure recognition of the cancer cells requires much experience. Experts can find the cells in 70% of cases.

TREATMENT.

Surgical removal of the tumour is the one treatment which holds out any hope of cure. The alternatives are deep X-ray therapy and cytotoxic drugs. The former is of considerable palliative value and may prolong life but only very rarely has it been shown to cure a definite case. It is not a milder or more conservative substitute for surgery. A full course produces about the same physical disturbance as pneumonectomy, and damages a considerable amount of healthy lung no matter how skilfully the X-rays are administered. Cytotoxic drugs are much less effective than radiotherapy, and their considerable side-effects, especially bone marrow depression, result in their use in very few cases.

CONTRA-INDICATIONS TO SURGERY.

These are numerous:

1. Distant metastasis as evidenced by lump, pain, tenderness, or neurological complications.
2. Mediastinal obstruction with swelling of neck veins and face.
3. Parcoast syndrome.
4. Recurrent laryngeal palsy with voice changes.
5. Phrenic/

5. Phrenic paralysis giving paradoxical movement.
6. Oesophageal involvement giving dysphagia.
7. Blood-stained pleural effusion indicating pleural spread.
8. Erosion of vertebrae as seen on X-ray.
9. Bronchoscopy findings of tracheal or carinal involvement.

The question of fitness for pneumonectomy is decided on clinical rather than on bronchospirometric grounds. This is done by observing the patient's performance at climbing stairs. The best measure of the latter is the Forced Expiratory Volume, which should be greater than 1.0. Further help can be obtained at operation by seeing how well the patient's colour is maintained on 20% oxygen after the intra-bronchial anaesthetic balloon has blocked off the affected lung. It must be borne in mind that a patient with a collapsed or otherwise functionless lung will be less dyspnoeic after the pulmonary vessels leading to it are ligated and their blood diverted to aerated lung.

A past history of coronary occlusion makes the operation more dangerous, as does chronic bronchitis or emphysema. None of these, however, forbids surgery. A considerable degree of hypertension is usually well tolerated. Any congestive cardiac failure calls for rejection, however.

In general age is no bar, but those over seventy have to be in very good condition before they are acceptable. Below this age lung resection has a very low mortality. In borderline cases efforts should be made to remove the tumour by lobectomy rather than pneumonectomy, even although the results for the former are less good.

Forbidding radiological appearances do not justify rejecting a patient because the degree of malignancy cannot be assessed from an X-ray. Even invasion of the chest wall, providing the thoracic outlet and vertebrae are not involved, does not contra-indicate to surgery.

All these factors were considered in Mr. E's. case. Although 71, he was in good physical condition with none of the signs of inoperability. His F.E.V. was 2.2 and he did not become over-breathless on climbing stairs. A decision for operation was made and Mr. E. was transferred to a Thoracic Surgery Unit. Here a right upper lobectomy was successfully carried out.

Three weeks after operation, Mr. E. was discharged feeling well and with only a little increase in breathlessness.

PROGNOSIS. Any patient who has a lobectomy for this condition has a 50 - 50 chance of being alive after 3 years. Mr. E. was investigated and treated early so that his prognosis should be better than most. On the other hand the carcinoma was undifferentiated, and these have a poorer prognosis. Spread may well have occurred beyond the excised area. Conversely the whole tumour may have been removed and a permanent cure effected. Only time will tell.

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Case No. 3.

Mrs. M. from Tranent, aged 28, a happily married woman with no children.

HISTORY.

This lady's presenting complaint was moderate breathlessness. As a child and teenager she had been very healthy but over the last two years had noticed a steadily increasing breathlessness. Her feet were always cold, but there were no tinglings, cramps, or weakness in the legs. The right ankle became swollen on occasions. She had no headaches or dizziness, no chest pain or tightness, and no cough or spit. Her appetite was poor, her weight steady, and her bowels regular. No other symptoms were obtained.

There was nothing of note in the past history. Mrs. M. did not smoke or drink.

EXAMINATION : A pale, thin, worried young woman who was not dyspnoeic sitting up in bed. There was no cyanosis, anaemia, oedema, finger-clubbing or lymph gland enlargement.

Cardiovascular system.

Pulse : 80/min. Regular in time and force, good volume, vessel wall not palpable. Femoral and other lower limb pulses were weak. Femorals were behind the radials in time.

Apex-beat was displaced laterally 1".

No venous engorgement.

Arterial pulsations could be felt over the scapula, and a systolic murmur could be heard over this.

Heart : no praecordial thrills; the second sound was accentuated in the pulmonary area; a loud systolic murmur was present over the base of the heart.

Blood pressure : 150/90 in both arms; 90/30 both legs.

No abnormality could be detected in the respiratory, alimentary, urinary, or nervous systems.

DIAGNOSIS : The signs elicited and to a lesser extent the history are the classical features of aortic coarctation. Further confirmation was obtained from a straight antero-posterior chest X-ray. This showed notching of the lower edges of the ribs, a small aortic knuckle, and left ventricular enlargement. This latter finding was also evident on electrocardiography.

TREATMENT : The only possible treatment is surgical correction of the deformity. The upper age limit for this is usually regarded as 30, because of the arterio-sclerotic changes resulting from many years of hypertension in the upper part of the body. These changes made the aortic wall unsuitable for suture, and by this time the changes in the terminal arterioles have become irreversible so that the blood pressure does not come down even after operation. In addition these changes make the collateral circulation a more formidable surgical obstacle.

Mrs. M./

Mrs. M. was very near this upper age limit.

On the other hand there is little doubt that the prognosis in untreated cases is very poor. Abbott has found the average age at death to be 32. Kiefenstein found that 61% of patients died before the age of 40, and 90% before the age of 50.

A decision for operation was made in Mrs. M's. case because:-

1. She was below the upper age limit.
2. The upper part of her body was not greatly hypertensive, so that arteriosclerotic changes would not be too far advanced.
3. There was no evidence of co-existing cardiac abnormalities, such as aortic regurgitation or congestive failure which are contra-indications to surgery.
4. There was no evidence of difference between the sides in respect of pulses or blood pressure. This would have indicated left sub-clavian or left common carotid involvement.
5. Her condition was deteriorating.
6. The prognosis untreated was so poor, whereas the operative mortality was only 7%, the results being good in those surviving.

The co-arctation was excised and end-to-end anastomosis carried out. There was good recovery and transfer back to the medical ward was possible in nine days. Two days later Mrs. M. felt very unwell and had a temperature of 104°F. Staphylococci were cultured from the blood. Lincomycin was prescribed. X-ray of the 12th post-operative day showed an aneurysm near the suture site. Three days later the temperature was normal and Mrs. M. was feeling much better. One week later Mrs. M. went back to the surgical ward and the following day underwent operation for repair of the aneurysm. This was discovered to be a false aneurysm and when the clot was removed the aorta began to bleed. The aorta was, therefore, cross-clamped. Immediately after the heart stopped. All efforts to restart it were unsuccessful.

DISCUSSION.

It is impossible to give a definite reason for the cardiac stand-still. Undoubtedly the immediate cause was the cross-clamping of the aorta, but other factors were involved as cross-clamping is only rarely followed by asystole and death.

The heart had always been under strain because of the coarctation and its reserve power was much impaired by this on admission, as evidenced by the increasing dyspnoea and the displaced apex beat. Further strains were imposed on it by the first operation, which included cross-clamping for some time, by the infection and associated tachycardia, and then by the second thoracotomy. In addition the vaso-motor centre would be depressed by the anaesthetic. Perhaps, therefore, it was not too surprising that, when on top of all these stresses, the aorta was finally cross-clamped, causing a sudden great increase in the resistance against which it had

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it had to work, that this final insult was too great and the heart was found to have passed the limit of its endurance.

Case No. 4.

Mr. B., a 29 year old married policeman from Edinburgh.

HISTORY.

This young man was perfectly well on the day before admission. During the night, however, he felt shivery, and woke in the morning with severe breathlessness and right-sided chest pain. The pain was sharp and constant, and was aggravated by coughing or attempts at deep inspiration. There was no cough or spit and no haemoptysis. These symptoms continued without respite up to the time of admission.

There was no history of trauma. Mr. B. had had four similar, though less severe, episodes in the past. None of these had been treated. The first was at the age of 15 and the last about one year previously. No history of respiratory disease, except colds, was obtained and there was no personal or family history of tuberculosis.

Both Mr. B's. parents were alive and well. None of his family had ever had similar attacks.

EXAMINATION.

A tall, muscular, fit-looking man. No cyanosis, anaemia, or oedema. No finger-clubbing. No lymph gland enlargement. Temperature 98°F.

Cardiovascular system.

Pulse : 110/minute. Regular in time and force, good volume, wall not palpable.

Apex beat in 5th space and mid-clavicular line.

No venous engorgement.

Blood pressure 130/90.

Heart : no thrills, first and second sounds normal, no murmurs.

Respiratory system.

Moderately dyspnoeic sitting in bed.

Diminished movement on right.

Trachea central.

Vocal fremitus absent on right.

Tympanitic percussion note on right, normal on left.

Breath sounds absent on right. No accompaniments.

Vocal resonance absent on right.

Coin sign negative.

DIAGNOSIS.

The history and clinical signs are those of the classical picture of spontaneous pneumothorax. The dramatic onset of pain and breathlessness also suggests pulmonary infarction but this would have produced a greater degree of shock, perhaps cardiac failure, and certainly not the distinctive lung signs elicited.

Radiological/

Radiological examination revealed the virtually complete collapse of the right lung, with air in the pleural cavity. The mediastinum was central and the left lung field clear. No effusion was present. The diagnosis of pneumothorax was confirmed.

AETIOLOGY.

The causes of this condition are:-

1. Rupture of a sub-pleural emphysematous bulla.
2. Rupture of a sub-pleural tuberculous focus.
3. Certain other conditions in rare instances - lung abscess, pulmonary infarction, bronchial carcinoma, and bronchopneumonia (especially in children).
4. Idiopathic. This is a large and important group made up of persons in whom no other evidence of pulmonary disease can be discovered. It is usually assumed that such cases are due to the rupture of small blebs or bullae.

The cause in this case was unlikely to be tuberculous for several reasons. The history of previous similar episodes does not suggest T.B.; Mr. B. felt very well the day before the attack; he had no fever, a normal E.S.R., and his sputum did not reveal acid fast bacilli on microscopic examination; pneumothorax as a manifestation of T.B. is found only in cases with obvious clinical and radiological signs of the disease.

There was no evidence of emphysema in the lungs, no history of bronchitis or asthma, and no breathlessness between attacks. The presence of emphysematous bullae was most unlikely, therefore.

The less common causes can be dismissed because of the lack of history, symptoms, or signs of any antecedent condition. Pneumothorax in such conditions is usually a late development and not a presenting complaint. In a man of this age none of these causes is likely, especially as the attacks have come over a period of fourteen years.

Mr. B. would, therefore, appear to fall into the group of apparently healthy persons who develop this condition idiopathically.

CAUSATION OF IDIOPATHIC SPONTANEOUS PNEUMOTHORAX.

This occurs most frequently in young adults between the ages of 15 - 25. A high proportion of such cases become chronic, the lung failing to re-expand within a few weeks. In addition many recur - as many as 20% within a year.

In unilateral cases the cause is often a small bleb or bunch of blebs related to an apical adhesion or old scarring. In other unilateral cases and in most bilateral ones, especially if recurrent, a sub-clinical generalised emphysema is present. Pathologically this is indistinguishable from the chronic degenerative emphysema common in the aged, in which there is a general loss of pulmonary elastic tissue. In the young this condition is clinically a clear-cut entity found in otherwise healthy adults, and may well be due to a congenital deficiency of elastic fibres in the lung. Some support for this view is given by

the fact that the affected lung, if seen early in the disease, either at thoracoscopy or thoracotomy, shows small blebs scattered over its surface. These are quite unrelated to scars or adhesions; and are fed by minute air bubbles 'seething up between the alveolar lobules in a way that strongly suggests interstitial defect'. At this stage no bullae are to be seen but these do develop later, usually at the apices initially.

TREATMENT.

The aim is to bring about re-expansion of the collapsed lung. When the pneumothorax is small and the patient not dyspnoeic, no active treatment is required. The patient is re-assured and kept under observation. The air in this case is gradually re-absorbed and the lung re-expands over a period of a few weeks.

Active measures are needed in more severe cases and therefore in Mr. B's. case. Immediate and complete re-expansion can usually be obtained by inserting a rubber catheter into the pleural cavity and connecting it to a water-seal drainage system, the catheter then being left in place for five or six days on continuous suction. If expansion does not take place then either a communication remains between the pleural cavity and the air passages or the lung is held down by thickened pleura.

In such chronic cases and also in recurrent ones surgical treatment is required. At operation any ruptured bullae are tied off and the parietal pleura is stripped off. The lung is then re-inflated to fill the hemithorax. If successful such operation is a permanent cure.

The only other alternative, not often used, is the induction of a chemical pleurisy, followed by an attempt to withdraw the air, in the hope that the inflamed visceral and parietal layers of pleura would then adhere. This method had little to commend it. It is highly unreliable, very prone to recurrence, and more painful and disabling to the patient, than thoracotomy. It also leaves the true nature of the lesion in doubt.

In Mr. B's. case the lung re-expanded well on drainage and suction. Two weeks later he felt very well and the lung was fully expanded, the drainage having stopped on the fifth day. He was, therefore, discharged.

PROGNOSIS.

This is not good. Recurrence is almost inevitable. Chronicity is likely on the next occasion and operation will be required. In fact, as this is a generalised affection of the lung, bilateral pleurectomy will be required before Mr. B. can be assured that the attacks will not happen again.

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Case No. 5.

Mrs. T., a 29 year old housewife, happily married with two children.

HISTORY.

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HISTORY.

This lady was first admitted to the ward in 1957, having suffered from breathlessness for two years. This had been considerably worse during her first pregnancy in 1956, and on this earlier admission she was again pregnant. All the classical symptoms and signs of mitral stenosis were then present.

Mrs. T. was transferred to a Thoracic Surgical Unit where a mitral valvotomy was performed. At operation no regurgitation was found and partial finger rupture and complete anterior and posterior commissurotomy with a transventricular dilator were carried out. After this Mrs. T. was considerably improved and later had a straightforward confinement.

This improvement remained until six months before this admission, when she began to feel very breathless on exertion once more. This steadily became worse and it became obvious that re-stenosis had occurred. A second valvotomy was performed. After complete posterior and partial anterior commissurotomy at this operation, a strong regurgitant jet was felt at the posterior end of the valve. The exact cause of this was uncertain, but it was thought likely that there had been detachment of the aortic cusp at the posterior end. In addition the valve was left with some stenosis because of the failure to divide the anterior commissure completely.

Recovery from this second valvotomy was satisfactory but on her return to the medical ward Mrs. T. was little improved and, in fact, the dyspnoea was a little worse. After ten days at home Mrs. T. had to be re-admitted. She was able to walk only ten yards on the flat and was continually breathless. Palpitations were frequent and paroxysmal nocturnal dyspnoea severe. She had a cough, producing white frothy sputum.

EXAMINATION.

A thin, pale, worried woman, slightly dyspnoeic sitting up in bed. No cyanosis, anaemia, or oedema.

Cardiovascular system.

Pulse : 100/minute. Regular in time and force; poor volume; wall not palpable.

Apex beat : forceful and displaced 1" laterally.

Blood pressure : 115/60.

Heart : No thrills; first sound was diminished; pansystolic murmur heard in the mitral area. This was a loud blowing murmur transmitted into the axilla and into the back near the scapula. It was also heard down the left sternal edge. A mid-diastolic rumble could be heard in the mitral area also.

Respiratory system.

There were a few inspiratory rhonchi and basal crepitations on both sides, but no other abnormality.

No abnormal signs were found in the alimentary or nervous systems.

INVESTIGATIONS.

Chest X-ray. The cardiac outline was unchanged from those taken before the last operation. The left ventricle was not markedly enlarged. Some congestion of pulmonary vessels was seen.

E.C.G. This showed left atrial hypertrophy and a little left ventricular hypertrophy.

DISCUSSION OF FINDINGS.

The symptoms and signs in this unfortunate woman were consistent with the production of major incompetence at her second mitral valvotomy. There was no evidence of any respiratory infection. The dyspnoea was so severe that life had become quite intolerable for Mrs. T. The prognosis untreated was extremely poor. Only one course was open - further surgical intervention to repair the damaged valve. Transfer to the Thoracic Surgical Unit was again carried out.

OPERATION.

This was performed under cardio-pulmonary by-pass. An attempt was made to suture the torn aortic cusp but this left a very stenosed valve which could not be dilated. The whole mitral valve was, therefore, resected and a Starr Edwards valve introduced and stitched into position.

Recovery from the operation was satisfactory and uneventful. Anti-coagulants were begun on the 3rd day. Just how long these must be continued for is still a matter of some debate, but for several years at least.

By the time of discharge Mrs. T. was greatly improved from her pre-operative state. Any real evaluation of the effect of the operation must, of course, be made at a later date.

PROGNOSIS.

Seven or eight years ago the prognosis for a case of this severity would have been almost nil. Now with replacement by an artificial valve, Mrs. T. has been subjected to one of the very recent advances in this field.

Her prognosis is impossible to forecast even approximately. Given that there are no immediate complications she should have a considerable mechanical improvement of blood flow in the heart, and there should be a corresponding improvement in symptoms. Some was certainly apparent on discharge.

Because insertion of an artificial valve is such a recent venture, the possible complications are only now being discovered. There may be a tendency for thrombosis to occur at the valve, hence the long-term anti-coagulant therapy. The complications of this therapy itself are not inconsiderable. Although the materials used in the construction of the valve are designed to prevent it, a low-grade inflammatory reaction to the valve's presence by the surrounding tissues is not impossible. Infection may be pre-disposed to by the presence of the valve.

How/

How long the valve itself will last has not been discovered, although testing in animals gives it a theoretical life of 70 years. One drawback already found in the human is a tendency for the valve to shrink, especially the ball.