A. H. KITCHIN.

CASE I

Name: James Alexander.

Age: 46

Address: 65, Howdenhall Road, Liberton.

Occupation: Gardener.

Date of Admission: 20.2.47.

COMPLAINT: Breathlessness on exertion - 7 days.
Puffiness of face - 6 days.

HISTORY:

This man is a gardener by occupation. He states that he was in perfect health until six days prior to admission, when as he was walking home from his work through the snow he became aware of undue shortness of breath. On reaching home, however, he felt quite well, slept satisfactorily, and returned to his normal heavy outdoor work the next day. That evening he had a similar experience and in addition his wife noticed that his face appeared slightly swollen and puffy. His dyspnoea was more marked but was related only to exertion. He also had a sense of discomfort, not amounting to pain, in his chest; a feeling of constriction or tightness. The next day he did half a day's work without any unwonted symptoms but his face remained swollen and after work in addition to the symptoms already noted he found his ankles slightly swollen. He retired to bed early, feeling tired, and the next day his face and ankles were grossly swollen. When his doctor came/

came to see him he was feeling better after the rest in bed and the symptoms were less obvious. On the doctor's second visit on Wednesday, however, he considered it advisable to hospitalise the patient, who was consequently admitted to the Royal Infirmary the next day.

The patient stresses that up till the Saturday he continued to do heavy work without any difficulty. The work he was engaged on was mainly shovelling snow, but on questioning it appears that a few days previously he had cut down a Douglas fir and had accidentally soiled his face with some of the plentiful sap from the tree.

Mr Alexander was in the Fire Service during the war without any absence through illness. Apart from childish ailments, including scarlet fever, mumps and whooping cough, he has had no significant illnesses. He never had rheumatic fever. He has had occasional attacks of impetigo of the face superimposed on a congenital ichthyosis of which there is a family history.

On admission the patient was afebrile.- He had had no headaches and no urinary symptoms. He had had a rather severe head cold three weeks before, but no sore throat. He was not subject to sore throats. He had had no digestive upset, and his usual bowel habit of 2-3 motions per day was unchanged. The shots were of normal colour and consistence though rather bulky. He smokes 40 cigarettes per week and takes no alcohol.

His father died aet. 76.

Mother, 76, alive and well.

4 brothers and sisters alive and well.

1 brother died, aet. 29, from heart disease.

EXAMINATION:

The patient was a small man of stocky build and good muscular development. His face was obviously puffy though of good colour, oedema being well marked around the eyes. His skin was of a rough and scaly texture though there were no eruptions. He was not pale or cyanosed or jaundiced. Temperature 98°F.

Cardiovascular System:

Pulse rate normal (76). Rhythm regular in the main with occasional extrasystoles. Good pulse pressure; vessel wall impalpable.

Blood pressure 150/85.

The jugular veins were congested and there was oedema of the face and legs. No capillary pulsation.

Precordium - Apex beat visible and located in 6th space in the anterior axillary line. There appeared to be some indrawing of the chest wall overlying the apex with systole. Visible carotid pulsation was present in the neck.

On palpation the forceful apex beat was confirmed. No thrills were felt.

The area of deep cardiac dullness was increased both to the left and right of the sternum.

The heart sounds were loud. A short rough mitral systolic murmur was present propagated internally towards the sternum. No diastolic murmur was present. The heart sounds in other areas were loud but closed and no murmurs were detected.

The radial pulses on both sides were equal and the pulses in the lower limb were present.

Respiratory/

Respiratory System:

The chest was symmetrical but slightly increased in relative antero-posterior diameter and did not move well on respiration. The chest expansion was poor. Dullness to percussion was present at both lung bases posteriorly, where the breath sounds and vocal resonance were diminished. Over other areas the breath sounds were vesicular.

The nose and pharynx appeared healthy.

Alimentary System:

No symptoms were present. The abdomen was of normal contour with no dilated veins. Ascites was not present.

The liver edge was palpable one finger's breadth below the costal margin. The spleen was not palpable.

Renal System:

The kidneys were not palpable. No frequency or dysuria. Urine examination - no blood, casts, albumen or sugar.

Nervous System:

Cranial nerves - no lesion detected.

Motor function - no weakness or paralysis or wasting present.

Reflexes - all present and normal.

No sensory loss found.

SPECIAL INVESTIGATIONS:

W.B.C. 4,600.

Haemoglobin 106%

Circulation time 14"

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

Pathological:

22.2.47: Pleural fluid: Some large mononuclear cells, polymorphs, lymphocytes/

lymphocytes and R.B.Cs. No tubercle bacilli or other organisms.

Biochemical:

21.2.47: Pleural fluid: total protein 1.18 gm.%

CO_2 combining power: 64.

Serum albumen - 3.75 gm.%

Serum globulin - 2.5 gm.%

Blood urea nitrogen - 13 mg.%

22.2.47: B.M.R. - 9%

Blood cholesterol = 191 mg.%

4.3.47: B.M.R. + 21%

13.3.47: B.M.R. ± 5%

X-Ray:

22.2.47: Chest - Enlargement of heart. Aortic knuckle unusually small. Pulmonary vessels enlarged in both lung roots. Evidence of effusion in right costophrenic angle.

27.2.47: Chest - Some diminution in opacity of right lower lung field. Pleural effusion cleared. Heart shadow enlarged in all diameters, mainly right ventricle and pulmonary vessels. Aorta small.

Screen examination - No diminution in pulsation at any point: rather increased over right border and middle segment of left border. Pulmonary vessels also pulsatile.

12.3.47: Chest - Some diminution in size of heart and pulmonary vessels.

Electrocardiogram:

Normal rhythm. R. 60. Occasional ventricular extrasystoles. P.R. 0.16. P waves of very low voltage. Vent. complexes broadened to 0.12 with slurring of 5 in lead I - Wilson type R. bundle branch block./

block.

Chest lead from point over L. ventricle grossly aberrant. Serial chest leads - late activation of R. ventricle.

Treatment and Progress:

With complete rest in bed facial and ankle oedema disappeared in about a week and at the same time the signs of right pleural effusion disappeared. The weight fell from 10 st. 3 lbs. to 8 st. 8 lbs. in the first two weeks, and thereafter remained steady. The venous engorgement coincidently returned to normal. The blood pressure remained constant at about 140/85 throughout the patient's stay. The frequency of extrasystoles tended latterly to increase. The cardiac enlargement remained substantially unaltered until two weeks after admission and thereafter diminished until on discharge the apex beat was in the 5th space and only a finger's breadth outside the midclavicular line. During the second two weeks thyroid gr. I b.i.d. was administered.

Three weeks after admission the patient was allowed up. He was discharged at the end of the fourth week feeling well and with no symptoms. His bradycardia was still present, however, and associated with many extrasystoles. The E.C.G. abnormalities (right bundle branch block) persisted.

DISCUSSION:

This case is of interest because it presented all the clinical features of a mild congestive heart failure occurring in a man aged 46 with no previous history of ill health, and in whom none of the major aetiological factors of heart disease could be convincingly demonstrated. As remarkable was the rapid recovery of apparently normal health as a result of four weeks rest in bed. The principal features of the case on admission were progressive effort dyspnoea, oedema distributed to the face and legs, and clinical evidence of a grossly dilated, slow, active heart with murmur, a rough mitral systolic, arrhythmia of extrasystolic type, and marked systemic venous engorgement. A small right pleural effusion was present. Further investigation revealed a slightly reduced serum albumen (3.75), a variable B.M.R. (-9 to +21), blood cholesterol level of 191, B.S.R. 12, enlarged pulmonary conus and right bundle branch block with severe myocardial damage. There was also a history of a head cold three weeks before admission, a normal bowel habit of two to three stools per day, and a brother having died of heart disease at the age of 29.

In differential diagnosis of this condition one is faced with the possibility of a primarily renal or primarily cardiac origin. An acute nephritis might well be suggested by the distribution of oedema, the onset with malaise and cardiac embarrassment, the mild hypertension on admission, and the history of upper respiratory infection. On the other hand the urinary examination, chemical and microscopic, was entirely negative. There was no sustained hypertension as one would expect in chronic glomerulo-nephritis, and no albuminuria or nitrogen retention. In the absence of any evidence of impaired

renal/

renal function the hypothesis of a renal origin must be abandoned.

Of the causes of gross cardiac enlargement those affecting the left side of the heart particularly, chronic hypertension, coronary artery disease, and syphilitic heart disease, seem to be unlikely factors in this case. There was no evidence of a primary or essential hypertension, and none of one secondary to renal disease. The mode of onset, too, with mild dyspnoea and marked venous engorgement is not suggestive of systemic hypertension failure. There was, however, radiological evidence of pulmonary hypertension. Coronary disease, possibly in the form of a "silent" coronary thrombosis, might be compatible with the rather vague sense of constriction in the chest which the patient experienced, and with the electrocardiographic report, but the patient's age, in the absence of any sign of degenerative arterial disease, aortitis, aortic valve disease, or syphilitic serology, makes this unlikely. The occurrence of coronary embolism as a result of acute endocarditis is excessively rare, and would have given rise to a more dramatic onset.

Purely right-sided causes of enlargement include chronic pulmonary disease (chronic bronchitis and emphysema, fibrosis of the lungs due to infection or pneumokoniosis) which were absent, and pulmonary infarction, which though compatible with the clinical type of failure present would have given rise to pleurisy and haemoptysis or at least some pulmonary signs.

The type of failure found in mitral stenosis was suggested very strongly by the relative degrees of dyspnoea and congestion, by the shape/

shape of the heart and evidence of pulmonary hypertension, and by the presence of a modest pleural effusion. The sudden onset, however, the age of the patient, and the curious absence of cyanosis cast doubt upon this, doubt which was confirmed by the absence of the faintest diastolic thrill or murmur. There was however a rough systolic mitral murmur, heard maximally between apex and sternum, and a moderately loud basal second sound not referred specifically to either pulmonary or aortic area. Sir Thomas Lewis, in discussing the criteria for the recognition of mitral stenosis, remarks that an accentuated apical first sound with an accentuated or reduplicated pulmonary second form an insufficient basis for diagnosis, and are due oftener to augmentation of the heart beat than to mitral stenosis. The sign only, he says, justifies the diagnosis, and that is the appropriate murmur. On the other hand, he concedes that a constant, long, harsh systolic murmur heard at or beyond the impulse usually signifies mitral disease. The diagnosis of mitral regurgitation, however, has a very limited importance; it may be useful as an early indication of muscular failure. Whether the mitral valve in this case has or has not been affected with rheumatism it is safe to say that mitral obstruction is not the cause of failure.

Since the case does not fit in to any of the commoner patterns of cardiac failure, the question of congenital morbus cordis may be considered. The absence of cyanosis disposes of those conditions which are by nature veno-arterial shunts such as pulmonary stenosis accompanied by septal defect (Fallot). The characteristic murmur of patent ductus arteriosus was absent, as were the radiological signs of the adult type of coarctation of the aorta. A defective interauricular septum, however, might well be compatible with the clinical picture.

Persistent/

Persistent foramen ovale occurs in 25 per cent. of all hearts and usually gives rise to no symptoms. A malformation of the upper septum produces a more considerable burden on the heart. The right heart and pulmonary artery may become greatly enlarged. The X-ray picture is of a large globular heart, a prominent conus and pulmonary arteries, and, as in this case, a small aortic knot. The E.C.G. shows right axis deviation, and often inversion of T in leads II and III. There is no characteristic murmur, though the pulmonary second sound may of course be loud. Crossed embolism may occur. Death occurs in the average at 35 years and may be from heart failure.

Patent interventricular septum is usually associated with pulmonary stenosis and the characteristic syndrome of dysonoea, cyanosis, clubbing and polycythaemia. Occasionally however the septum of the ventricles may be open in its upper membranous part without other defect in the heart ("Roger's disease"). Cyanosis will not be present unless there is extensive mixing of blood in right and left ventricles. The right ventricle is hypertrophied. There is a characteristic murmur - systolic in time - heard best in the third and fourth intercostal spaces just to the left of the sternum. It is not conducted to the vessels of the neck or to the axilla. This raises the question of whether the systolic murmur heard in this case was functional or organic. In deciding this point one takes account of the character, duration and propagation of the murmur, the presence or absence of a thrill, the permanancy or otherwise after treatment, and the response to effort. In this case though in the grossly overactive heart the murmur/

murmur seemed rough in quality, it was of short duration, was not propagated, was not accompanied by a thrill, and was absent on discharge. It is therefore likely to have been a so-called "functional" murmur dependent upon relative dilatation of the mitral ring. It was almost certainly not the murmur of patent interventricular septum which is prolonged, rough and permanent.

One diagnosis which ought to be considered in a case of this nature is chronic constrictive pericarditis, an explanation which has superficially several points in its favour. The failure was predominantly right sided with great venous, and especially jugular, engorgement; it improved rapidly with rest; the oedema was distributed especially to the face and legs, suggesting impediment to return from the superior as well as the inferior vena cava. On examination of the pericardium an observation was made that there was some systolic retraction of the ribs at the apex. On the other hand the degree of cardiac enlargement was too gross and the heart muscle too obviously active to suggest an adherent, constricting pericardium. There was a good pulse pressure. The liver was not enlarged as one would have expected. Finally the X-ray picture showed good pulsation of both ventricles and no calcification. The observation of systolic rib retraction demands some explanation; probably with a great degree of dilatation and an active apex the phenomenon is more truly described as a systolic release of pressure on the chest wall. There is the added factor of marked ventricular asynchronism.

Although acute pericarditis is clearly not a permissible suggestion, it is worth while noting that recently several cases have been reported of/

of tuberculous pericarditis producing gross failure of relatively short duration without other detectable cause. It is suggested that tuberculous pericardial effusion may be a commoner condition than has been previously recognised and that it may be insidious in its onset, transient in its course, and complete in resolution. It may be a late primary tuberculous manifestation and analogous to pleural effusion. In this case, however, the forceful apex and loud heart sounds eliminates the possibility of a gross effusion and the hypothesis must be regretfully withdrawn.

There is one further class of cases presenting cardiac enlargement of obscure origin. It comprises those due to the malabsorption syndrome, a deficiency of vitamin B being considered specifically responsible for the cardiac manifestations: and those associated with disorders of metabolism consequent upon thyroid dysfunction. With both of these groups curiously enough, this case has certain affinities. The patient has for long had a bowel habit of two or three stools per day, each of them bulky and semi-formed though not markedly pale or offensive. The type of failure, with great enlargement, especially right sided, venous engorgement and oedema of face and limbs is typical of the classical Beriberi heart. There was a decreased plasma protein level. The dry scaly skin might well represent an associated deficiency of nicotinic acid. The absence of peripheral neuritis does not exclude Beriberi heart. On the other hand the patient took an adequate diet and had no digestive upset. He had no weakness, wasting or malaria, and no haemorrhagic tendency was manifested. Also in Beriberi the failing heart tends towards tachycardia rather than bradycardia/

bradycardia.

Bradycardia is, however, a feature of the heart in myxoedema. The patient's general appearance suggested myscoedema in spite of its rarity in the male: the scanty eyebrows and hair, the thick dry scaly skin, and the face which remained rather puffy even after the subsidence of the manifest oedema. The blood cholesterol level was slightly elevated. The flattened T wave in the electrocardiograph is characteristic. Finally the first reading of the B.M.R. was -9, and although this estimation is a notoriously variable one it is more usual for error to occur in the opposite direction. The X-ray picture however was unlike that of myxoedemic heart. The heart though dilated was forcefully pulsatile. Also the circulation time was not markedly diminished. In interpreting B.M.R. readings, also, it must be remembered that the presence of oedema introduces a considerable error into the calculation.

It might perhaps be fanciful to suggest that the "cold" of those weeks before was was in reality a mild nasal diphtheria and that the present illness was in reality a post diphtheritic manifestation. Here however the failure is usually of a much more acute type, with rapid feeble pulse, vomiting and prostration.

It appears, then, that this case falls into none of the major categories of cardiac failure, and that it presents features which are not wholly compatible with any of the lesser causes. Perhaps the least objectionable diagnoses are patency of the interauricular septum and myxoedema. The patient was indeed treated with thyroid B.P. gr. II per day on assumption of the latter diagnosis, and his condition/

condition continued to improve. No deduction can however be drawn from this circumstance. In the absence of a diagnosis prognosis is a dangerous speculation, but it may be said that the features of the case, taken in association with the rapid improvement and good condition on discharge, are suggestive that the condition is not a peramament and progressive one. One of the most important parts of the subsequent case of this patient should be regular re-examination both clinically and with the X-ray screen and electrocardiograph in order to come to a diagnosis and if necessary institute rational therapy.

CASE II

Name: Allan Graham Anderson.

Age: 38

Address:

Date of Admission: 19.2.47.

Date of Examination: 20.2.47.

COMPLAINT: Weakness of limbs - 3 years.

Difficulty with vision, mastication, speech - 2 years.

HISTORY:

This patient, a shop assistant before the war, was called up in 1940 and was passed as grade I medically for service in the police. He was in perfect health until early in 1943 when one day, escorting a drunk man whom he had arrested in the course of his duties, he found himself unable to maintain his grip of the man's coat collar which he was grasping with his right hand. His arm became powerless and fell to his side. He was able however to substitute his left hand. Shortly after this incident he had a similar experience during the violent apprehension of another malefactor. Subsequently he became aware of occasional attacks of weakness in one or other of his arms. This weakness appeared to come on only while the limb was in action and sustained use. Over a period of months it became increasingly troublesome,/

troublesome, so that manual operations involving sustained muscular contraction, such as shaving and combing the hair, became impossible save by using both hands alternately. The patient found his writing becoming more clumsy and difficult. Medical and specialist advice having effected no improvement in his symptoms he was discharged from the police in 1943 and got a clerical job which did not involve writing continuously but adding columns of figures, etc., with which he found no difficulty.

Some two and a half years ago he noticed weakness of his legs on walking and especially when climbing stairs. Though this did not affect him badly at home he found great difficulty in, for example, boarding a tram car while a queue of passengers was waiting behind him. His difficulty in walking made his gait rather conspicious.

Two years ago the patient began to experience diplopia particularly when looking fixedly at an object above the level of his eyes such as a cinema screen. This was worse at night. He found that relaxation of his gaze effected a temporary amelioration. At the same time his eyelids began to droop under similar circumstances necessitating frequent blinking and even tilting back his head. The jaw tended to droop during the course of meals, and he suffered difficulty in mastication. Occasionally he choked while drinking, and sometimes food regurgitated into his nose. His speech became thick and tired easily. The influence of any emotional stimulus in precipitating an attack became marked. His gait became worse, and walking so difficult sometimes that he actually took a taxi to his work.

During three years the patient had been under almost continuous medical/

medical observation. When he first consulted his doctor he was treated with a 'bottle.' The patient was unfavourably impressed by the fact that he was not examined on this occasion, and is inclined to ascribe his subsequent tribulations to his fact. He later insisted on seeing a specialist and was examined in M.O.P.D. Owing to the fact that no objective signs could be elicited, a diagnosis of functional disorder was made and physiotherapy advised. The patient was however quite incapable of carrying out at home the exercises prescribed for him at the Physiotherapy Department.

He then consulted a chiropractor who manipulated his spine twice weekly with extreme profit to himself but none whatever to the patient.

The patient's doctor then sent him to the Davidson Clinic where from May to November last year he was treated by dream analysis with no conspicious results.

To add to the patient's worries his job became redundent in January, 1947. His anxiety was becoming extreme; he had a wife to support; and he became fearful for his sanity. Periods of panic he found accentuated all his symptoms. Finally he was examined by Dr Steele who admitted him to Jordanburn Nerve Hospital for investigation, where two weaks ago he was successfully treated by injections.

EXAMINATION:

The patient was a tall well-built, intelligent man of average muscularity and showing no signs of malnutrition. He was of normal colour, with no cyanosis or pallor or jaundice, not oedematous, and with a healthy skin. His facial expression was rather striking: it appeared/

appeared heavy and rather flabby and the corners of his mouth drooped. He spoke in a low voice and rather indistinctly, a feature which increased in the course of examination. The patient was in bed, but his gait later observed and was found to be a slow rather waddling one with a tendency to slide the feet along the floor and to grasp hold of objects for support. The temperature was normal.

Nervous System:

The mental functions - intelligence, emotional state and memory - appeared normal. The patient had no difficulty in sleeping normally, and was inclined to drowsiness. The speech had the features described above, being thick and indistinct, and tending to deteriorate after 5-10 minutes.

Cranial Nerves -

1. No anosmia or parosmia.
2. Glasses worn. Visual activity normal: fields normal. Eye grounds - no abnormality detected.

3,4,6. Pupils equal in size, regular, react to light and accommodation. No obvious squint or ocular paresis; no nystagmus. A strong tendency to ptosis was present especially if the patient fixed on one object for a few minutes, when he stated that diplopia also occurred.

5. No weakness of muscles of mastication found at first, but on examination after 15 minutes very marked.
7. No weakness of facial muscles or loss of taste.
8. Hearing normal. No tinnitus or vertigo.
9. Taste normal. No dysphagia.
10. Movement/

10. Movement of palate equal on both sides.

11. Shrugging of shoulder normal.

12. Tongue not wasted, protruded normally but not long sustained.

Motor - The motor power of the limbs was unimpaired and equal on both sides, with no sign of paralysis or wasting. The muscle tone appeared normal. The hand grasp, however, was poorly sustained.

No abnormal movements or tremor were present and no error in position sense could be detected although the grip appeared ataxic.

Sensory - No abnormal subjective sensations. No headache. Touch, pain, temperature and position sense normal. No abnormal vasomotor phenomena or trophic disturbances.

Reflexes - Biceps, triceps, supinator reflexes present and equal.

Knee, ankle present and equal. No clonus.

Babinski negative.

Abdominal reflexes present but easily fatiguable.

Micturition and defaecation normal.

Cardiovascular System:

Pulse normal in rate, rhythm, pressure. Vessel wall impalpable. Blood pressure 135/85.

No dyspnoea, cyanosis, oedema, or venous congestion.

Heart - apex 5th space in midclavicular line. No thrills. Heart sounds normal and closed in all areas. No murmurs.

Respiratory System:

No cough, sputum, breathlessness or pain.

Respiration normal in rate and chest moved well and equally on both sides.

Percussion/

Percussion note resonant and breath sounds vesicular all over chest. No accompaniments.

Alimentary System:

No symptoms referable. Abdomen soft on palpation with no enlargement of liver or spleen.

Urinary System:

Kidneys not palpable. No frequency or dysuria. Urine examination normal.

Haemopoietic System:

No enlarged lymph glands or spleen. Not anaemic.

Endocrine System:

No thirst or glycosuria, no goitre. No spasms or pigmentation or skeletal changes.

DISCUSSION:

Although the text book picture of myasthenia gravis would appear to be highly characteristic and easy to recognise, yet as this case shows the diagnosis may present considerable difficulty in practice and involve much ineffectual therapy for the patient. The reason for this, apart from the rarity of the condition, is probably to be found in an insufficient attention to the detailed history of the attacks of weakness, and secondarily in a too hurried examination of the nervous system. It serves to emphasise the prime importance in neurological investigation of the time factor; the clinical features of an affection of the nervous system depend not alone upon the localisation of the lesion but also upon its behaviour in time, and this applied not only to the obvious instances of sudden and gradual spinal compression, or slow-growing and sudden cerebral space-occupying lesions, but also in conditions like disseminated sclerosis, postero-lateral sclerosis and tabes dorsalis, conditions in which the tendency to regard the neurological picture as a static one, delineated by the eliciting of certain constant signs, is well known. Myasthenia is a case in point; it is the function and not the local topography of the nervous system which requires investigation. The result in this case was that repeated examination of the usual type, namely running over the body with a tendon-hammer, revealed no objective signs whatever: a vicious circle was established in which the nature of the complaint, the lack of localising signs, the patient's increasing anxiety and despair and the long list of medical failures pointed more and more insistently to a functional - neurasthenic or hysterical - origin for the symptoms.

It/

It is a remarkable fact that this patient's malady was only diagnosed when he was unable to walk the length of a room, unable to eat a meal without great difficulty or to write for more than a minute or two, and that the diagnosis was established in a hospital for mental disorders.

The salient features of the case were the relation of the symptoms to fatigue both locally in particular muscle-groups, and generally in the deterioration during the course of the day, the heavy rather expressionless facies with a strong tendency to ptosis, the fluctuating difficulty in articulation and the heavy, sliding, unsteady gait. The conditions from which such a picture must be differentiated include disseminated sclerosis, in which unequal tiring of one or more limbs, with growing disability, and fatiguability of reflexes are also features, but features which are more localised, and which tend to fluctuate and shift much more gradually. In addition there are often localised signs of spasticity in the reflexes and muscle tone, and paraesthesiae may be complained of. In particular transient retrobulbar neuritis is a common early lesion, or slight precipitancy of micturition, while in a late case the characteristic triad of nystagmus, intention tremor and scanning speech are present with perhaps some pallor of the temporal halves of the optic disc. By that stage, however, the patient is usually reduced to bedridden helplessness. When, as sometimes happens, myasthenia begins with a unilateral ophthalmoplegia or laryngoplegia the diagnosis may be difficult, but other forms of nuclear ophthalmoplegia do not show a long history of variability and fatigue phenomena. The myopathies may give some difficulty in differentiation, in particular dystrophia myotonica where there is weakness and wasting of the facial muscles, a mournful, set expression, wasting of the sternomastoids, forearm/

forearm and leg muscles, and delayed relaxation of muscles. The wasting, weakness and fibrillation of motor neurone disease, affecting particularly the hands and arms and in some cases leading to spasticity in the lower limbs, is easily differentiated, but chronic bulbar palsy with its increasing difficulty in articulation and deglutition may give difficulty. The wasted and fibrillating lower facial musculature and wrinkled, constantly-moving tongue with limited protrusion are characteristic. The marked subjective symptoms of spontaneous hypoglycaemia and the transient nature of the condition are not likely to lead to confusion.

Although still a rare condition myasthenia is said to be more prevalent in Britain now than twenty years ago. It rarely occurs before puberty, begins in the third decade usually, and affects the sexes equally. Myasthenia is said sometimes to be associated clinically with Graves' disease, which it may follow, and in which the ophthalmoplegias which may occur bear no small resemblance to those of myasthenia. When myasthenia develops in middle-aged or elderly persons it is apt to run a more chronic and benign course than in young persons. It shows no familial tendency.

The pathology of the condition is somewhat obscure: the individual muscle fibres appear normal but in about 50 per cent. of cases there are small collections of lymphoid cells in the affected muscles - so-called lymphorrhages. Similar collections are found in the thyroid, adrenals and pancreas. The thymus is enlarged in half the cases, and definite thymomas have been described. It may be noted that the thymus undergoes hypertrophy in hyperthyroidism and also in hypofunction of the adrenal cortex and the sex glands. The most important clue to the

immediate/

immediate cause of myasthenia is the dramatic improvement (reported by Walker in 1934) which follows the administration of physostigmine (or the synthetic product prostigmin) and which suggests that the fundamental defect is in the humoral transmission of impulses from nerve endings to muscle fibres. Indeed the disease affects a very pretty illustration of the now widely accepted hypothesis of the action of acetyl choline as chemical mediator in the neuromuscular junction. Experiments involving a comparison of the effects of autonomic and motor nerve stimulation with the pharmacological actions of acetyl choline, or the stimulation of the autonomic nerves to an organ and testing the venous blood issuing from it, or the perfusion fluid for acetyl choline-like action have revealed that in addition to the parasympathetic effects of acetyl choline ("muscarine" action) it has a stimulant action upon ganglion cells, upon voluntary frog muscle and upon denervated mammalian muscle ("nicotine" action). The nicotin effects are not annulled by atropine as are the muscarine effects. Further, the ester is rapidly hydrolysed in alkaline media into acetic acid and choline, and blood and other body fluids contain an enzyme "cholinesterase" which rapidly inactivates it. The action of acetyl choline is greatly intensified by physostigmine or eserine, which acts by inhibiting this cholinesterase. The exact manner in which physostigmine (or "prostigmin" presumably) exerts its beneficial effect in myasthenia is unknown; in the disease the cholinesterase level is not higher than normal. On the other hand a fall in the cholinesterase activity of the blood after prostigmine administration has been demonstrated. The creatinine excretion is considered to be an index of the magnitude of tissue and especially muscle metabolism, and as might/

might be expected myasthenia is accompanied by a high degree of creatinuria and a reduction in creatinine excretion. Besides protigmin, promising results have also been obtained following oral administration of glycine, especially when combined with ephedrine, and also with potassium, the action of which in synaptic transmission appears to be the enhancement of nicotine action by potentiating the effort of acetyl choline upon ganglion cells (Brown and Feldberg).

The treatment of myasthenia gravis at the present time consists in the first instance of the administration of prostigmin. The oval route is the most convenient, some four to eight 15 mgm. tablets being taken during the day. In this case treatment was started with a tablet two-hourly, and a dramatic improvement ensued, the patient becoming almost normal while under the influence of the drug and relapsing immediately if a dose was omitted. This dosage must be adjusted to suit the activities of the patient, who soon learns to regulate the dosage accurately according to his needs. In some cases restricted activity may still be necessary. The prognosis is very variable. An early fatal issue (due to respiratory paralysis) is more often seen in adolescents or young adults than in older patients, while on the other hand young subjects may make a rapid and complete recovery from the greatest manifestations of the malady.

It will be seen that this form of therapy, life-saving as it may be, leaves much to be desired, and in most cases tends to be palliative rather than curative. The observation of a frequently enlarged thymus gland has recently led to the trial of thymectomy as a curative measure, and there seems to be no doubt now that remarkably good/

good results have been obtained in a high proportion of cases. At present, however, the operation is technically a very difficult one, and carries a mortality of the order of 20 per cent. Its recommendation is therefore still a matter for very serious deliberation. Thymectomy is entirely an empirical form of treatment, the great body of experimental work carried out to elucidate its functions having achieved very little success, except to suggest that status thymolymphaticus, a postmortem diagnosis favoured by anaesthetists, does not exist as a pathological entity. The results of thymectomy, however, seem to suggest that the thymus will eventually be classified among the endocrine organs.

CASE III

Name: Thomas Whitson.

Age: 24.

Date of Admission: 9.2.47.

Date of Examination: 16.2.47.

COMPLAINT: Febrile illness with slight muscular pain following a head cold four weeks ago.

HISTORY:

At the age of 13 the patient had an acute febrile illness said to have been rheumatic fever, but was otherwise healthy until 1942, when he was conscripted and served in the infantry. Nine months later on routine medical examination he was down-graded from category A1 to B for no reason that he could suggest. At no time did he suffer from effort dyspnoea or any other manifestation of cardiac disease. He was demobilised in June, 1946, and remained in good health until one month ago when he developed an unusually severe head cold. A week later he began to suffer slight intermittent pain in the calf and thigh muscles of both legs and later in the left thenar eminence and left forearm. He had no pain of an arthritic type. He was markedly febrile, and began to sweat during sleep, sometimes profusely. His doctor treated him with salicylate therapy, without any apparent response. He had not any recent sore throats, nor was he subject to them. He smoked six cigarettes/

cigarettes per day and consumed a moderate amount of alcohol.

The patient's parents and two sisters were healthy, and there was no history of familial predisposition to any disease. The patient lived at home, however, and there was a suggestion of considerable frustration and unhappiness in his domestic relations.

EXAMINATION:

On admission the patient had no symptoms. He was a pale, slightly-built youth with somewhat scanty dark hair, but not markedly anaemic. He was responsive to suggestion but inclined to depression and anxiety over his circumstances. His skin was of a fine texture and moist to the touch.

Temperature 99.8°F. Pulse 85. Respirations 22.

Cardiovascular System:

No dyspnoea, cyanosis or oedema. No distended veins or ascites. Pulse equal on both sides, normal in rhythm and force. Vessel wall impalpable. Blood pressure 124/80.

Precordium - normal on inspection. No thrills felt. Apex beat palpable in 5th space just inside mid-clavicular line. Right border not enlarged.

Auscultation - a soft blowing systolic murmur was heard in the mitral area propagated for a short distance outwards towards the axilla. The murmur became less easily detectable on exercising the patient mildly. The other heart sounds were closed. There was no accentuation of the second pulmonary sound. No diastolic murmur was detected.

Respiratory System:

No cough or spit. No nasal abnormality detected though patient has/

has some tendency to mouth breathing. Pharynx healthy.

Chest - inpection - normal in shape and movement.

palpation - movement equal on both sides. Vocal fremitus normal.

percussion - normal resonant note throughout.

auscultation - faint vesicular breath sounds save in apical region where bronchovesicular sounds were heard.

Alimentary System:

No symptoms; good appetite, bowels move normally. Teeth - one or two carious.

Abdomen - inspection - normal contour. No peristalsis seen.

palpation - slight voluntary guarding. No masses palpable. Liver not enlarged. Spleen not enlarged. No tenderness on deep palpation.

Urinary System:

Kidneys not palpable. No renal pain or tenderness. Bladder not enlarged. No dysuria. Urine normal in amount, frequency and colour. Urine examination - Acid 1020. No albumen, sugar or blood. Microscopically: no pus, casts, or R.B.Cs.

Locomotor System:

No noticeable muscle wasting or weakness in any part of body. Joints normal save perhaps slight muscular tenderness on extreme movements of left wrist.

Nervous System:

Cranial Nerves - No lesion found. Pupils equal and reactive.

Motor system - No weakness of muscles, wasting or tremor.

Sensory/

Sensory - No anaesthesia or paraesthesia. Position and vibration sense unimpaired.

Reflexes - biceps, triceps and supinator jerks normal.

knee, ankle jerks normal.

plantar responses flexor.

brisk abdominal reflexes.

Haemopoietic System:

Liver, spleen not enlarged. No pain in bones. Not anaemic. No jaundice.

Haemoglobin 92%. W.B.C. 14,500. No enlarged glands.

FURTHER INVESTIGATION

Chest X-ray - Heart normal in size and shape. Lung fields normal.

B.S.R. 88 mm/hr.

Throat Swab - Haemolytic and non-haemolytic streptococci.

Blood cultures - 3 negative results.

Widal reaction - negative.

Paul-Bunnell - negative.

Agglutination tests - negative.

G.C.F.T. - negative.

Wassermann reaction - negative.

Blood film - normal.

TREATMENT AND PROGRESS:

For two weeks after admission the patient remained unchanged, running a mild pyrexia with marked tachycardia. He continued to lose weight. He was kept in bed while the relevant investigations were/

were carried out, and treated with 20 gr. sodium salicylate with alkali four-hourly and phenobartitone to allay his restlessness and anxiety. Then he suddenly began to complain of pain in the left wrist and thenar eminence, followed a day or too later by the small joints and wrist of the right hand. The salicylate, which was then increased to 30 gr., had no effect on the pain, which was at times very severe and distressing to the patient. The affected joints were immobilised in heavily padded cock-up splints. In a day or two slight wasting of the inter-metacarpal tissues on the dorsum of the hands became evident, the adductors of the thumbs and forearm muscles being also affected to some extent. Some swelling of the joints was present, and they were intensely tender to touch. At this stage there was no doubt of the diagnosis of acute rheumatoid arthritis.

DISCUSSION:

With the widespread and increasing use in general practice of powerful antibiotic agents such as the sulphonamides and penicillin it is inevitable that their indiscriminate employment should lead to to many cases of missed or vague diagnosis, and there is considerable danger that their profound value as accurately indicated and scientifically controlled therapeutic agents may be offset by the understandable temptation to use them as empirical 'cures'. A great many cases, then, of pyrexia of vague or unknown origin will be successfully treated, and others will remit spontaneously, but there remain some cases which present considerable difficulty in diagnosis, and in whom prolonged observation or clinical laboratory tests provide the only solution.

In this case considerable confusion was added to the diagnosis by the misleading history of an illness twelve years ago labelled as rheumatic fever but not characterised by joint pains, and of the patient's subsequent down-grading in the Army, presumably for cardiac disability though the effort tolerance was not impaired and at present no sign definitely indicative of mitral stenosis is to be found. His present illness started insidiously with marked pyrexia and slight muscular pains, and continued thus for some four weeks before symptoms definitely indicative of the nature of the pathological process were forthcoming.

In dealing with such a case it is first necessary to exclude obvious sites of sepsis causing toxaemia by examination of the mouth, nose, sinuses, throat, chest, urine, prostate and skin, and by excluding any abdominal surgical condition. If no further evidence is forthcoming, the/

the various causes of unexplained pyrexia should be considered. A recurrence of rheumatic fever is one possibility that had to be considered in this case: the features that one would expect in addition to fever are a transient acute arthritis of the large joints and moving rapidly from one to another leaving no residual disability, a low grade leucocytosis, and, almost invariably, a favourable response of the pain and pyrexia to large doses of salicylates, which in this case were entirely ineffective. Other cardiovascular diseases which might be considered are subacute bacterial endocarditis, and ulcerative endocarditis, which however is generally overshadowed by the primary causative disease, but which should be suspected if multiple abscesses occur. Subacute bacterial endocarditis is associated with a history of previous rheumatism, pallor, clubbing, a dilated heart with often changing organic murmurs, embolic signs, and (on repeated examination) a positive blood culture.

After a respiratory infection a persistent pyrexia is usually indicative of a complication such as empyema, lung abscess or pericarditis, but in a spontaneously arising case like the present the question of tuberculous infection should be very seriously considered. Any condition characterised by a general toxaemia with some loss of weight, asthenia, dyspepsia of a flatulent type, shortness of breath, pyrexia, tachycardia and hypochromic anaemia should raise a suspicion of tuberculosis especially if the temperature should show a persistent evening rise with sleep sweats. This is the stage at which pulmonary tuberculosis should be diagnosed, not when gross pulmonary signs and symptoms are evident, and the diagnosis should be confirmed or excluded by (1) bacteriological examination of the sputum,/

sputum, gastric contents, faeces or urine; (2) X-ray examination of the lungs and bones; (3) Mantoux reaction; (4) B.S.R. though this is less helpful as a diagnostic measure than as a guide to activity.

Typhoid and paratyphoid fevers are the most important medical diseases which cause pyrexia with abdominal symptoms, but again fever may be the only well-defined symptom. The insidious mode of onset with epistaxis or bronchitis is suggestive. The intestinal symptoms include vague abdominal pain, tenderness, tympanites, and either diarrhoea or constipation. The slow rise of temperature in the first week, sustained fever of the second, and fall by lysis in the third are characteristic, as are a relative tachycardia, leucopenia, and positive blood culture or Widal. Though acute hepatitis is usually evident in three or four days by the development of jaundice and signs of biliary obstruction, liver abscess, particularly amoebic abscesses nowadays, may give considerable trouble. History of residence in the tropics, diarrhoea with blood and mucus or pain and tenderness in the right hypochondrium are suggestive, but frequently the condition is diagnosed by the coughing up of the contents of the abscess which has tracked through the diaphragm. Low grade cholecystitis, pelvic or subphrenic abscesses or a perinephric abscess are all occasionally responsible for persistent pyrexia.

In the haemopoietic system Hodgkin's disease is perhaps the commonest cause of fever which may occur in the acute form of the disease, in the terminal stages of the chronic form, or in the abdominal form. The pyrexia is usually of the Pel-Elstein type.

Hodgkin's/

Hodgkin's disease cannot be diagnosed clinically only; it demands also blood examination (eosinophilia in pyrexial stages), and above all biopsy of an affected gland. Acute leukaemia may give pyrexia, but some pallor, weakness, haemorrhages, sloughing necrosis of membranes, pleurisy and the blood picture make the diagnosis clear. In children and young adults glandular fever may be misleading as the gland involved may be mediastinal or mesenteric instead of, as is common, behind the sternomastoid. An excess of atypical mononuclear cells is present on blood examination, and the Paul-Brunell test for heterophile antibodies is positive in 92 per cent. of cases. Agranulocytosis is suggested by a history of drug taking and inflammed pharynx.

Frequency of micturition or dysuria should call attention to the possibility of urinary infection and a full examination of the urine is necessary. Bacilluria may cause no fever, but if pyuria is also present fever generally occurs. Pyelitis, cystitis, urethritis (gonococcal or non-specific) may be found, and the accessory organs, prostate and seminal vesicles, should be examined for tenderness.

Several infections of the nervous system may present difficulties in the early stages. Anterior poliomyelitis may be ushered in with fever and generalised pains, and is often thought to be an 'influenzal attack' until evidence of paralysis appears. Acute myelitis may also present difficulties, but as a rule sensory and motor changes in the limbs and disturbance of micturition soon make a diagnosis possible. Meningitis, especially the insidious tuberculous variety, should be considered/

considered as a possible cause of unexplained pyrexia. Photophobia, neck stiffness and Kernig's sign should be looked for, but lumbar puncture is generally necessary to establish the diagnosis. Encephalitis is also a protean disease. Mental symptoms, changes in sleep rhythm, and ocular paresis of transient nature should suggest the possibility of this disease. Cerebral abscess may be suspected when signs of a cerebral tumour are associated with fever.

A pyrexia waxing and waning at weekly intervals, and showing a daily swing of a few degrees should suggest relapsing fever, which starts indefinately with tiredness, pains in the limbs and anorexia. The spleen is usually palpable, and the agglutination test for Br. abortus is positive from the fifth day onwards. The periodicity of malarial pyrexia, the enlarged spleen, the Leishmann-stained blood picture and also the therapeutic test with quinine are diagnostic.

In children especially osteomyelitis should be suspected, and the bones examined for points of tenderness.

In this case the evolution of the disease process enabled a diagnosis of rheumatoid arthritis to be made. The aetiology of this condition remains obscure. It has been widely held that chronic focal infection is the most important single factor. The fact that many persons without arthritis have septic foci and that many arthritics show no manifest septic foci makes the evaluation of the theory difficult. The infective basis for the disease is suggested by the acute features of fever, sweating, leucocytosis, raised B.S.R. and generalised wasting. The organism most frequently blamed is the streptococcus, various workers having obtained serological indications of streptococcal infection/

fection in 40 to 75 per cent. of cases. Bacteriological examination of joint fluid and blood has yielded essentially negative results. In some cases the muscular and arthritic response appears to be in the nature of an allergic reaction, the arthritis for example appearing some days after a streptococcal tonsillitis or in the course of bacillary dysentery. The history of upper respiratory infection in this case may be significant. The relation of rheumatoid arthritis to acute rheumatic fever is obscure; it was long held that the two conditions are entirely separate from the point of view of pathology and prognosis and that rheumatoid arthritis is a benign condition as far as life is concerned. This may in the main be so, but recent long-term follow-up in America of persons who have suffered from rheumatoid arthritis has shown that a significant proportion of them eventually develop mitral valvular disease. A family history of rheumatoid arthritis is quite often obtained.

The course is a long one, punctuated by frequent remissions and relapses. Phases of activity may last from a few weeks to many months. Spontaneous arrest occurs in about 50 to 60 per cent. of cases, but in others the disease progresses causing gradually increasing deformity and disability through fibrous adhesions and ankyloses. Relapses are precipitated by cold, damp, overwork or excessive use of affected joints. One of the most interesting features of the disease is that it seems sometimes to be precipitated, and often aggravated by psychological disturbance - worry or overwork.

The treatment consists of confinement to bed until all signs of activity are gone, with, if possible, the advantages of a sanatorium regime,/

regime, a high calorific diet with vitamin supplement, and eradication of any forms of sepsis. The joints affected must be splinted in optimum positions, for example, slight dorsiflexion of the wrist, extension of the knee. The limb should be taken out twice a day and moved passively through the pain-free range. By these means pain, swelling and muscle spasm are reduced, and, most important of all, the formation of adhesions and flexion deformities is prevented. When the acute phase is over heat may be applied in the form of hot paraffin wax or by the use of radiant heat or infra red lamps. In the later stages active physiotherapy may be employed.

The use of drugs, apart from analgesics, sedatives, and ferrous sulphate to combat anaemia, is a controversial subject. Gold salts are among the most dangerous drugs to administer, with the possibility of exfoliative dermatitis, leucopenia and agranulocytosis, purpura, toxic nephritis and liver damage unless the strictest examination of urine, blood and skin is carried out repeatedly. Severe toxaemia, renal, hepatic, cardiac, or skin disease, leucopenia or purpura naturally contra-indicate the use of gold. It is perhaps advisable that its administration should only be carried out under hospital conditions. The results of gold therapy are inconstant, but in the absence of material improvement in severe cases by simpler methods of treatment it might well be given a trial.

CASE IV

Name: Thomas Neilands.

Age: 17½ Single.

Address: Newcraighall, Musselburgh.

Occupation: Electrician.

Date of Admission: 30.10.44.

Date of Examination: 14.11.44.

COMPLAINT: Cough, developed one week before admission.
Spit, developed one week before admission.
Pain in lower right axilla, occasionally for same period.

HISTORY:

1. Present illness.

The patient stated that he was in good health until three weeks before admission, when he began to experience a sharp epigastric pain coming on about two hours after meals and lasting about five minutes. He was sick once or twice after the onset of the pain. At this time he noticed no other symptoms. A week after the commencement of the pain, he saw his doctor, who prescribed a diet and a medicine which completely relieved the pain. A few days later, however, (25th October), the patient felt unwell, and developed a dry cough which continued throughout the day, was not accompanied by pain, and was productive of a little greenish-yellow viscid sputum. The patient/

patient went to bed, and the following day he experienced occasional sharp pain in the lower right axilla. The cough became more frequent and troublesome, but the sputum was never abundant and was not blood-stained or rusty. The patient sweated at night, and complained of heat. The chest pain was not disturbing.

The doctor examined the patient again, and prescribed sulphonamide tablets of which 36 were administered in the five days prior to admission. As these tablets seemed to be having no effect, the patient was sent to hospital.

Apart from the coughing, the patient never felt really unwell. There was no history of shivering attacks, dyspnoea or haemoptysis. The appetite was good, the bowels fairly regular, and the urine apparently normal. He thought it probable that he had been losing a little weight.

2. Previous Health.

Previous to the onset of the present symptoms the patient stated his health was good. His only illnesses were scarlet fever at the age of eleven and measles in childhood. Newcraighall is a mining district. The patient's occupation - electrician - required him to spend a good deal of his time in the open air. He did not smoke or drink.

3. Family

The patient's father and mother were alive and well, and not predisposed to any particular type of disease, in the patient's knowledge, though the father suffered from a cough. He had two elder sisters and two younger brothers - all well.

GENERAL/

GENERAL EXAMINATION:

The patient was of average height (5 ft. 8½ in.) but poor muscularity for his development. Weight 8 st. 6 lbs. His face was thin and flushed, especially in the malar region. He had large bright deep-set eyes and long black eyelashes. The hair was black. The skin was moist and warm, but the hands had no sign of clubbing. The mucous membranes of the lips and conjunctiva showed a degree of anaemia. Sitting up in bed, the patient seemed quite comfortable and interested in his surroundings, though his general appearance suggested a degree of fever. The respiration rate was slightly increased, and the breathing mainly abdomino-thoracic in character. The spit-cup was empty. Temperature 101.8°F (axillary).

SYSTEMATIC EXAMINATION:

Respiratory System: Relevant symptoms - cough, spit, pain.

Inspection - The shape of the chest was normal and symmetrical, though there was some wasting of muscle, and myotactic irritability could be demonstrated. The respiratory-excursion was not great, and was slightly diminished on the right side in comparison with the left when viewed posteriorly. The rate of respiration was 23 per minute. The breathing was regular.

Palpation - On palpation it was confirmed that movement was diminished on the right side.

The vocal fremitus was found to be normal or slightly increased over the lower part of the right lung, but absent over the right base at the back.

No lymph glands were palpable.

Percussion - The anterior aspect gave a uniform normal note. On

percussing/

percussing the posterior aspect, however, it was found that the note was definitely dull over the right lower lobe. At the base of the right lung the note became stony-dull and increased resistance to the finger was felt.

Auscultation - In comparison with the left lung, the right showed four regions as regards breath sounds. Down to the level of the scapular spine normal sounds were heard. Below this, however, they changed to bronchovesicular, which merged into an area of medium pitched bronchial breathing. Here the breath sounds were typically harsh and blowing in character, and had a pause between inspiration and expiration. Co-extensive with inspiration and expiration were crepitations of medium quality. Over the base no breath sounds could be heard.

Vocal resonance was strikingly louder and harsher over most of the right lower lobe, with the exception of a small area at the base where it was faint and muffled. Whispering pectoriloquy was elicited.

Cardiovascular System: Relevant symptoms - cough, spit.

Pulse - 100 per minute, regular, not very forcible or well-sustained; artery wall not palpable.

Blood pressure - 110/60.

Inspection - No pulsations seen. Shape of precordium normal.

Palpation - Apex beat in 5th space, 3 in. from mid-line. Diffuse. No pulsations or thrills.

Percussion - Right border of heart just protruding beyond right border of sternum.

Auscultation - Heart sounds normal, regular, slight tachycardia (100). No murmurs; no pulse deficit.

Alimentary/

Alimentary System: Symptoms - dyspepsia, vomiting, pain. Tongue - clean and moist. Teeth - some carious. Throat - healthy. Inspection - Abdomen thin, symmetrical. No swelling, scars, veins. Moved regularly with respiration.

Palpation - Fair degree of rigidity. No tenderness. Lower border of liver palpable but not enlarged. No sign of fluid.

Percussion - Liver normal size. Spleen not enlarged.

Auscultation - Usual gurgling noises audible.

Haemopoietic System: Symptom - anaemia.

Spleen was not enlarged, and no palpable lymph glands were found.

Endocrine System: Symptom - pyrexia.

No indications of endocrine upset were discovered; the thyroid was not enlarged.

Urinary System: Kidney was not palpable. The urine output averaged 1 litre per day, being lessened due to sweating. There was no frequency. S.G. 1030. Reaction: acid. No albumen, but some urates.

Nervous System: The patient was of average intelligence, and slept well. No lesion of the cranial nerves could be detected. No paralysis, tremor, or abnormal movements. Reflexes present and correct (plantar reflex was flexor).

Locomotory System: Muscles somewhat wasted. Bones not deformed; no joint pains or swellings.

SPECIAL EXAMINATIONS

Blood.

	W.B.C.	Haemoglobin	B.S.R.
30.10.46	8,600	95%	55 mm/hr
8.11.46			

SPECIAL EXAMINATIONS (contd.)

Blood.

	W.B.C.	Haemoglobin	B.S.R.
8.11.46	7,800	80%	
14.11.46	7,200	68%	50mm/hr

Weight

4.11.46	8 st. 11 lbs.
11.11.46	8 st. 6 lbs.
18.11.46	8 st. 8 lbs.

X-Ray: Chest - The antero-posterior plate showed marked cloudiness of the right lung field extending from the diaphragm upwards to about the hilum. The right costophrenic angle and the outline of the diaphragm were obscured. The lateral plate showed a shadow above the diaphragm and rising in the axilla which seemed to indicate fluid. The left lung field seemed normal.

Bacteriological Examination of Sputum: The first such examination produced evidence of mixed diphtheroids and non-haemolytic streptococci. The second and third examinations revealed in addition the presence of tubercle bacilli.

DIFFERENTIAL DIAGNOSIS:

Conditions explaining some of the symptoms but not the lung lesion (e.g. nutritional iron-deficiency anaemia, early thyrotoxicosis, psychoneurosis, etc.) may be dismissed at once. Possibilites are:-

1. Bronchiectasis.
2. Neoplasm.
3. Collapse.

4./

4. Actinomycosis.

5. Chronic bronchitis.

6. Unresolved lobar pneumonia.

7. Fibrosis of lung.

8. Confluent bronchopneumonia.

9. Pneumokoniosis.

10. Pleurisy with effusion.

11. Pulmonary tuberculosis.

1. Bronchiectasis has usually a history of chest troubles. The sputum is foul and is coughed up in paroxysms on change of position. The condition tends to remain stationary or progress gradually. One would not expect a rapid onset and high fever. The X-ray does not suggest bronchiectasis.

2. Neoplasm would not cause toxaemic symptoms like tachycardia. There would probably be evidence of displacement of the mediastinum, or of cancerous lesions elsewhere in the body. Supraclavicular or axillary lymph glands might be involved.

3. No history of trauma which might produce collapse is given. Dyspnoea and cyanosis would be expected, and a diminished or absent vocal fremitus. The apex beat is not markedly displaced.

4. Actinomycosis of the lung is rare, and could only be demonstrated by the presence of streptothrix in the sputum. This was not found.

5. The obvious loss of weight does not suggest chronic bronchitis nor does the sudden onset of symptoms without previous history. Dyspnoea and paroxysmal coughing would be expected.

6. There/

6. There is no history of rusty sputum, severe pain, and rigor. Sulphonamide therapy had no effect. The patient never felt acutely ill.

7. There is no evidence of contraction or mediastinal displacement. Fibrosis is a secondary change rather than a separate disease; there is no history of acute or chronic inflammation affecting bronchi, lungs or pleura. A diminution in vocal fremitus and resonance would be expected.

8. Bronchopneumonia commences less abruptly, and is usually preceded by an infection of the upper respiratory tract leading to bronchitis. Physical signs usually bilateral.

9. Pneumokoniosis is an occupational disease; the history of this case does not suggest liability to it. The onset is insidious, not sudden.

10. The evidence of pain in the right lower axilla initially in this case (although not stressed by the patient) suggests that a degree of pleurisy had been present. The physical signs on examination also show a small effusion at the base of the right lung. Probably this effusion had been sufficient to relieve the pain by separating the inflamed surfaces. The pleurisy is however secondary only; it is not sufficient to explain the sputum and loss of weight.

DIAGNOSIS: 11. Pulmonary tuberculosis.

This diagnosis was arrived at by a consideration of the following symptoms and signs:-

1. The onset of the disease with stomach trouble and pleuritic pain.

2. Cough.

3./

3. Sputum.

4. Fever (showing nocturnal rise) and flushed appearance.

5. Loss of weight.

6. Anaemia.

7. Physical signs of unilateral consolidation and cavitation in the chest (with some pleural effusion).

8. Radiograph of the chest.

The diagnosis of tuberculosis was made certain by the discovery of the tubercle bacillus in the sputum.

The symptoms may be divided into three classes as regards causation: (1) pulmonary, e.g. expectoration and pleurisy; (2) reflex, e.g. cough, and (3) toxaemic, including tachycardia, pyrexia, loss of weight, weakness, and the disturbance of digestion which was the earliest symptom noticed.

The pain was due to pleurisy, the cough and spit to the softening of caseous material and its discharge into the bronchi. This is shown by the fact that the case is one of "open" tuberculosis, i.e. the tubercle bacillus is found in the sputum. There will thus be some degree of cavitation present.

As regards aetiology, the disease is caused by reinfection with the tubercle bacillus of an individual whose immunity was not sufficient to withstand it. The bacillus can survive for long periods outside the body. In adults it is usually acquired by the inhalation of dust or droplets, and spread by the lymphatics. The outcome of the infection depends on the size of the dose and the degree of resistance. There is much evidence that in the case of

secondary/

secondary infection the disease is really an allergic reaction, with gross loss of tissue, to bacterial products liberated by the death and destruction of the bacilli.

Predisposing factors are: age (commonest 15-45), heredity, climate and environment (overcrowding, defective sanitation, dampness, dirt, insufficient sunlight and ventilation) and lowered resistance through deficient or improper diet.

TREATMENT:

During his stay in hospital the patient was treated with rest in bed and an ordinary light nutritional diet supplemented by cod liver oil and ferrous sulphate gr. III three times a day. Three weeks after admission he was moved to a sanatorium.

The most beneficial treatment in this case would probably be (a) sanatorium treatment and (b) collapse therapy, e.g. artificial pneumothorax.

The advantages of sanatorium treatment are the strict regime under optimum conditions of fresh air and good feeding, constant medical supervision, freedom from ordinary environment and cares, and the appreciation by the patient of the most suitable mode of life for him. He must be kept at rest in bed until the pyrexia has disappeared, and until the B.S.R. has fallen to a reasonable level indicating cessation of activity of the disease. Thereafter gradually increased exercise should be taken, but subject always to the control of the temperature. After three months' stay it is usually possible

to/

to say whether the patient is responding to treatment, and whether it should be prolonged.

As soon as possible the right lung should be immobilised to promote healing. The best method of achieving this is the operation of artificial pneumothorax. This is contra-indicated in cases of dense pleural adhesions, emphysema, or cardiac embarrassment, none of which conditions appear to be present. Cases in which the disease is unilateral, as the present one is, and which remain febrile despite treatment in bed are specially suitable. The collapse is maintained for as long as three years, re-expansion then being cautiously permitted.

If pneumothorax should be unsuccessful through adhesions or other cause, crushing of the phrenic nerve might be considered in order to produce basal collapse. The effect lasts about six months, and does not prejudice further operative treatment.

PROGRESS:

On admission the patient was thought to be somewhat cyanosed, and oxygen was administered for the first night. This caused bleeding from the nasal mucous membrane, and gave rise to temporary haemoptysis. During the patient's three weeks' stay in hospital he lost weight in the first two weeks in spite of a good diet supplemented by cod liver oil, but he gained slightly in the third week. He was slightly constipated. In spite of ferrous sulphate the haemoglobin fell steadily. The temperature curve was always febrile. It showed a gradual rise during the first two weeks, and a corresponding fall in the third. The daily rise, which was present at first at 4 p.m. became/

became less pronounced. The pulse rate followed the temperature curve. The patient's cough became much less troublesome, and less productive.

PROGNOSIS:

The chances of complete recovery are difficult to estimate in the early stages of a disease like tuberculosis, where the course is a long one and setbacks frequent. It would seem, however, that the patient's prospects are reasonably good, as the disease was caught fairly early, had an abrupt febrile onset, and later seemed to show some improvement. The patient's age is also a factor, and the fact that the case is apparently suitable for collapse therapy. The treatment is prolonged, and afterwards the patient will require a long period of medical supervision. Return to work, which in this case seems of a suitable nature, should not be unduly delayed after recovery, but the patient should understand the desirability of taking a large amount of rest. The home surroundings are probably not ideal, but are virtually impossible to change, even if it were desirable to do so. Fresh air, good food, and as far as possible protection against colds and other infections are important during the long convalescence.

CASE V

Name: Peter J. Burgess.

Age: 20.

Address: 3, East Cromwell Street,

Leith.

Occupation: Merchant Navy fireman.

Date of Admission: 2.1.47.

COMPLAINT: Nil.

HISTORY:

This young man has served as a fireman in the Merchant Navy for the past 18 months, and two months ago, on being returned to the Merchant Navy Pool, he underwent a routine medical examination by the Merchant Navy medical officer, who detected a cardiac murmur and sent him to the Infirmary for investigation as a possible case of early rheumatic heart disease.

The history is that the patient left school at the age of 14 with no record of any significant illnesses in childhood save measles and whooping cough. He became a labourer, doing hard manual work in the open air, until in May, 1945, he joined the Merchant Navy, since when he has been doing exceptionally heavy work without any symptoms of distress in the nature of dyspnoea or palpitations. His hobby is boxing./

boxing. For the past three years he has had frequent sore throats, each lasting about a week and occurring at intervals of some 3-6 months, but on none of these occasions have there been sequelae of joint pains, and the patient has never to his knowledge suffered from acute rheumatism or chorea.

As regards habits the patient smokes 40 cigarettes per day and "occasionally" takes alcohol.

His parents are both aged 42 and are in good health. He has two sisters and four brothers alive and well.

EXAMINATION:

The patient was a healthy-looking, dark complexioned young man of slight build but good muscular development. He was not pale or cyanosed or jaundiced; his skin was healthy and of fine texture. Temperature normal.

Cardiovascular System:

No dyspnoea, palpitation, pain or faintness.

Pulse - normal in rate, rhythm, force and tension. Vessel wall impalpable.

Blood pressure 124/84.

Precordium - apex in 5th space in mid-clavicular line. No visible pulsations or dilated veins.

Palpation - position of apex confirmed. No thrills felt.

No extension of cardiac dullness detected.

Auscultation - the first sound in the mitral area was loud and rough and there was a loud blowing systolic murmur which was not propagated/

propagated towards the axilla. No diastolic murmur could be detected though the patient was examined lying on his left side after moderate exercise. A systolic murmur was also present in the pulmonary area and the pulmonary second sound was markedly accentuated.

Respiratory System:

No symptoms referable. Nose healthy but pharynx showed enlarged, slightly injected tonsils especially on the right side.

Chest moved well on respiration and equally on both sides. Percussion note resonant; vesicular breath sounds in all areas with no accompaniments.

Alimentary System:

Appetite and bowels healthy. Mouth and tongue clean; teeth good. Examination of the abdomen revealed no abnormality. Liver and spleen not enlarged.

Urinary System:

No urinary symptoms. Kidneys not palpable. Urine examination - no albumen, sugar or blood; no cells or casts.

Nervous System:

Cranial nerves - no lesion detected. Pupils equal and reactive.

Motor power - no paresis or weakness, wasting or tremor.

Reflexes - all present and normal.

No sensory loss found.

SPECIAL INVESTIGATIONS

Haemoglobin 112%

W.B.C. 9,000.

X-Ray - Chest: Heart probably within normal limits. Lung fields clear.

Electrocardiogram/

Electrocardiogram - Normal. Rate 68/min. P.R. 0.16. No axis deviation or T wave abnormality.

TREATMENT:

Tonsillectomy was carried out four days after admission without any unfavourable reaction. The patient was discharged after the relevant investigations had been carried out.

DISCUSSION:

The discovery during routine medical examination of an apparently fit man of a cardiac murmur not infrequently presents the conscientious medical practitioner with one of the most difficult problems of diagnosis he is called upon to make, and one of the most important. Cardiac crippledom as a result of mistaken diagnosis of heart disease is still met with, and to condemn a patient to the known prognosis and management of mitral stenosis is a serious responsibility; on the other hand it would be indefensible to neglect a case of suspected organic disease without the most careful examination.

This patient presented no symptoms of heart disease; he leads a life of great physical stress: he had no history of rheumatic fever though he suffered from frequent tonsillitis; and the X-ray and E.C.G. findings were those of a heart within normal limits. The diagnosis therefore turned on the clinical examination of the heart, and in particular upon the auscultatory phenomena. The positive findings were:- a loud mitral first sound merging into a loud blowing systolic murmur not propagated to the axilla, a systolic pulmonary murmur and a loud pulmonary second sound.

The time-honoured classification of murmurs as "functional" and "organic" is often unsatisfactory, for the former term has been applied alike to physiological and pathological signs and has no relevance whatever to the actual state of the heart. A simpler classification would appear to be into physiological and pathological groups, the latter comprising extracardial causation, as from anaemia, and intracardial, as from myocardial disease or valvular deformity.

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The three main systolic murmurs heard at the cardiac apex are:-

1. A blowing, moderately high-pitched murmur, beginning with or immediately following the first sound, varying in duration and intensity, and propagated, according to loudness, towards the left axilla. This type of murmur is due to systolic regurgitation through the mitral valve, and results from dilatation of the heart from organic disease or temporarily from extracardiac causes, or from deformity, usually rheumatic, of the mitral valve itself.

2. Another cause is transmission of a systolic murmur from the base, for example an aortic systolic murmur.

3. Finally it is sometimes due to movement of air into and out of the overlying or adjacent lung tissue - the cardiorespiratory murmur.

In this case clinical examination left no doubt that it was the first group that was represented, and the problem then became that of deciding whether dilatation or valvular deformity was at fault. Mitral regurgitation and mitral stenosis are very frequently combined pathologically, and from this point of view it would be preferable to make a clinical diagnosis of "mitral disease with preponderant stenosis" or "with preponderant regurgitation." If the lesion results primarily in stenosis, the left auricle and right ventricle bear the brunt of the burden and become hypertrophied and dilated. The left ventricle may be unaffected. If, on the other hand, regurgitation is the chief defect, the left ventricle becomes involved as well, and with marked and chronic regurgitation all four chambers may participate in enormous enlargement. Here no gross enlargement was present/

present, the heart being radiologically just within the upper limit of normal. The development of mitral stenosis is a gradual one, the earliest defect being more regurgitant than stenotic. It requires at least two years for the establishment of true stenosis. At times it is the valve lesion itself, and not myocardial disease, which is the cause of death, although, at least according to the view emphasised by Sir Thomas Lewis, the converse is generally the case.

The only two pathognomonic signs of mitral valve disease are auscultatory and radiological, the presence of a rumbling apical mid-diastolic murmur with or without presystolic accentuation, in the absence of any cause of left ventricular dilatation: and the presence of a considerable increase in the size of the shadows of the right ventricle and of the pulmonary artery combined with well marked enlargement of the left auricular shadow. The murmur must be sought under the most favourable conditions: it is heard most frequently with the patient lying upon the left side, and sufficient exercise to raise the pulse rate slightly often serves to bring it out.

Neither of these two signs is present in this case and therefore mitral disease cannot be diagnosed confidently: on the other hand the presence of a loud apical systolic murmur without any acute or subacute illness or evidence of left ventricular enlargement must be regarded as highly suggestive of early mitral disease, especially as two other corroborative signs - accentuation of the first sound at the apex, and of the second sound at the pulmonary area - are in evidence. The absence of right axis deviation is by no means incompatible with early disease.

The conclusion from examination would therefore be that while a diagnosis of valvular disease cannot be made positively there exists/

exists a strong suspicion of its future development, suspicion which can only be confirmed or disproved by periodic examination. The insecure nature of mitral regurgitation as a diagnosis makes its value in prognosis very dubious. Prognosis in any case should be based not upon a count of murmurs but upon experience, and it appears to be proved that many patients carry the murmur of mitral regurgitation through life without any deterioration in the condition of the lesion.

However strong therefore the suspicion of mitral valve disease may be the wise course in the treatment of the patient as a whole would appear to be to leave physical activity unrestricted, strenuous as it is, since the heart is of normal size and the effort tolerance unimpaired and there being no grounds to suspect the presence of active infection. The provisos to this course of action would be that the first sign of limitation of effort tolerance should indicate a review of the patient's health and conditions of work, and that a periodic but not too frequent examination should be made to elucidate the progress or otherwise of the disease process.

CASE VI

Name: James Millar.

Age: 43. Married.

Address: 18, Upper View, Craig Row, Edinburgh

Occupation: Newspaper packer.

Date of Admission: 19.12.46.

Date of Examination: 10.1.47.

COMPLAINT: Attacks of unconsciousness on two occasions.

HISTORY:

This patient was called up in 1942 and served as a medically fit man in the Pioneer Corps. One day in 1943 having woken up in the morning feeling well he became progressively weaker during the course of the forenoon while engaged in his duties, and by mid-day found himself unable to lift a limb and compelled to sit still. He lost consciousness for a period which appeared to him to have been a few minutes but which might have been longer. He recovered consciousness to find that he had been incontinent of urine. He had not bitten his tongue and he gathered from subsequent accounts that he had not had a convulsion. He had a persistent cold sensation in the back on recovery. He was taken to a military hospital and there kept under hypnotics for three days before being returned to normal duties apparently completely cured.

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The patient saw active service in Normandy, France and Germany; his rank was a sergeant's; and he is not disatisfied with his record of service. During this period he had no symptoms. He was demobilised one year ago and returned to his job as a newspaper packer.

Three days before admission to hospital he felt out of sorts on returning from work and slightly shivery as though a cold were imminent. He also complained of a cold sensation in the back. His symptoms remitted however and next day he went to work. He felt weak in the afternoon and went to bed at five o'clock. His weakness and tiredness progressed; he did not sleep, and at 4 a.m. he found he could scarcely move. With difficulty he emptied his bladder, mindful of the outcome of his previous experience, and thereafter he lost consciousness. His wife stated that for about two hours he was restless and talked confusedly, but had no convulsions. He came to panting and sweating and his strength gradually returned though on admission he still felt rather weak. He had no headache at any time.

The patient had never suffered from effort dyspnoea or oedema, from headaches or dizziness, visual disturbances or localised weakness of limbs. On further questioning, however, he recollected that for some two weeks before the latter episode, and also he thinks before the former, he had slight paraesthesia which he referred to his ankles; a feeling of pins and needles, as though his feet were crossed and the circulation impeded.

He says that he is satisfied with his work and has no particular worries. He has a happy home life. He has had no previous illnesses. He is one of a family of ten, four of whom only are alive and well. Father/

Father died aet 76 with gangrene of both legs. Mother died aet 56 of septicaemia. His wife, two daughters and one son are alive and well. No member of his family has been subject to attacks similar to the ones described.

EXAMINATION:

The patient was a well-built, broad-chested man of good colour and intelligent disposition. There was no evidence of previous disease or injury. He was apyrexial at the time of examination, but had been febrile the day before.

Cardiovascular System:

No dyspnoea, oedema, cyanosis or dilated veins.

Pulse - normal in rate, rhythm and force with occasional extra-systoles. Blood pressure 160/90.

Precordium - apex beat palpable in 5th space inside mid-clavicular line. No thrills.

Heart sounds faint in all areas. No murmurs detected.

Nervous System:

Cranial nerves - no lesion detected. Pupils equal and reactive to light and accommodation. No visual disturbance.

Motor system - no weakness, paresis, wasting or spasticity.

Reflexes - biceps, triceps, supinator jerks present and normal. Knee and ankle jerks brisk. No clonus. Abdominal reflexes not obtainable. Plantar reflexes flexor on both sides. No sensory loss or paraesthesia detected.

Respiratory System:/

Respiratory System:

No breathlessness. Slight unproductive cough. Slight degree of clubbing.

Respiration rate normal and abdominal in type.

Nose and pharynx healthy.

Chest rather barrel-shaped but good movement. Symmetrical. Percussion note unimpaired in all areas. On auscultation the breath sounds were harsh vesicular in type and a few fine crepitations and rhonchi were to be heard diffusely over the chest.

Alimentary System:

No relevant symptoms. Appetite and bowels normal. Mouth and tongue healthy. Abdomen normal in contour; no abnormal findings on palpation. Liver and spleen not enlarged.

Renal System:

Kidneys not palpable. Urine normal in amount and constituents.

Haemopoietic System:

No glandular or splenic enlargement. No evidence of anaemia.

SPECIAL INVESTIGATIONS

Haemoglobin 110%

W.B.C. 5,800.

X-Ray - 26.12.46 - Chest: Linear increase in density in left cardio-phrenic angle suggesting unresolved inflammatory process.

Biochemical

20.12.46: CO_2 71

Urea 13

23.12.46: Agglutination tests - No agg. of B typh., Para A & B. abortus Paul/

23.12.46: Paul-Bunnell - negative.

Blood culture - no growth.

24.12.46: Sputum culture - pure growth pneumococci.

27.12.46: Sputum culture - haemolytic streptococci.

28.12.46: Sputum - no tubercle bacilli. non-haemolytic streptococci, micrococcus catarrhalis.

30.12.46: C.S.F. - Cells 1.

Glob. normal.

Gold sol. - 0000000000

W.R. -

Cl 696 mg.

Sugar 77 mg.

Protein 70 mg.

Electrocardiogram - Rate 65/min. Normal sinus rhythm. No axis deviation. Broadening of P waves in II and III with slurring of Q.R.S. in II and III. May be abnormal.

TREATMENT AND PROGRESS:

On admission the patient had a pyrexia of 101°F without leucocytosis which persisted for a few days. With the possible exception of a unilateral positive Babinski sign on admission which could not be confirmed later there was no sign of neurological or of cardiovascular disease. A few days later the patient began to have a cough with some purulent sputum. He had mild physical signs of acute bronchitis. The temperature rose to 102°F and he felt weak and faint as before. No change in the physical signs occurred in the cardiovascular system or central nervous system. The patient was treated with sulphathiazole.

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The sputum disappeared, the temperature subsided and he had no further faintness or weakness. He was discharged at the end of three weeks after complete investigation.

DISCUSSION:

Fainting or loss of consciousness is one of the most alarming symptoms a patient can experience, especially if it comes on without warning and is repeated. Under such circumstances the direct interpretations are apt to be put upon it and the association with cardiac disease or the mysterious and dreaded epilepsy invoked to the full. It is also true medically that attacks of unconsciousness demand the most careful investigation and diagnosis, for though they may be indicative of nothing more serious than a tendency to vasovagal instability they may also be a manifestation of such conditions as cerebral tumour, hypertensive encephalopathy, heart block and cardiac arrhythmias, diabetes or Addison's disease. Hence the necessity of careful examinatio as a basis for reasoned reassurance of the patient.

Much help in the diagnosis can be obtained from a full and accurate history, for in many conditions loss of consciousness is attended by certain particular circumstances, the aura, cry, tonic and clonic phases, tongue biting and incontinence of the classical attack of grand mal for example. In this case the features which may be noted particularly are the progressive weakness preceding the attacks, the shivering or cold feeling which the patient experienced in the back, the fact that actual loss of consciousness occurred (a point which may sometimes be difficult to establish) and that it was followed by a period of confusion. No convulsions occurred, but incontenence of urine was present. The other important point is that apparently complete recovery took place in a matter of a day or two, and that the patient was in normal and vigorous health between the attacks. One might also perhaps comment

on/

on the treatment of the patient after his first attack when he was taken to hospital, rested in bed and given sedatives for three days and then returned to duties. This strongly suggests that at that time the view was taken that the origin of the condition was functional.

On seeking for the cause one might consider first the central nervous system of which consciousness is a function. The most likely nervous causes are idiopathic epilepses, symptomatic epilepsy associated with cerebral tumours or possibly general paralysis, vascular lesions, subarachnoid haemorrhage, acute poliomyelitis, encephalitis, and perhaps narcotic poisining.

A vascular accident at the age of 43 is unlikely save in the case of embolism associated with endocarditis, or possibly meningovascular syphilis, neither of which is present. No paralysis was left after the attack and there were no localising signs as in meduallary thrombosis for example. Angiospasm occurs only in gross arterial disease and causes pareses rather than unconsciousness alone. Subarachnoid haemorrhage is ushered in by sudden intense headache without prodromal symptoms. Pyrexia and signs of meningeal irritation follow the attack which is likely to give rise to prolonged disability. An unruptured aneurysm might give rise to localising signs, especially visual, but not to unconsciousness. Another variety is cortical venous angioma. Here fits are more typical than fainting attacks. Usually they occur first in the third or sixth decades and for several years physical examination may be wholly negative. Finally after an attack some residual symptoms are left, for example a hemiparesis which is usually transient but may in time become permanent.

Poliomyelitis/

Poliomyelitis may in childhood start with an attack of unconsciousness in the acute stage but this is rare in adults, and the subsequent neurological phenomena are unmistakable. Likewise acute encephalitis is associated with delirium at night and drowsiness or coma during the day, but the duration of some ten days and the presence of other, especially eye, signs, make this an improbable diagnosis. Virus diseases in general are very unlikely to be recurrent.

General paralysis may be associated with epileptiform seizures which may give rise to some difficulty in diagnosis, but this patient's general health, his mental condition and the absence of tremor or dysarthria, as well as his negative serology make this diagnosis impossible.

Poisining with narcotics, especially nowadays with the barbiturates, may give rise to a clinical picture of unconsciousness with a cold, clammy skin, pallor, slow and irregular respiration, and a diminution in reflexes. There may be present an extensor plantar response, and the eye signs include miosis and strabismus.

The characteristic and indeed the only essential feature of epilepsy is the fit, of which there is no clear history in this case. Incontinence on the other hand did take place, and on admission there was a doubtful finding of a positive Babinski sign. Idiopathic epilepsy almost always makes its appearance before adult life is reached, and the occurrence for the first time in middle life of epileptic fits commonly betokens the presence of some structural cerebral lesion. There is no family history of epileptic attacks. The long interval between attacks (two and a half years) does not suggest epilepsy. There is a degree of constancy about the major fit that rarely leaves considerable doubt when a reasonably good account is available. No true aura was experienced, but instead a feeling of weakness lasting/

lasting several hours. Finally the electroencephalogram, for what it is worth, is not typical of epilepsy.

Symptomatic epilepsy may however be the first sign of a cerebral tumour, and in such cases it is generalised epilepsy which more frequently occurs than the Jacksonian type. A slowly infiltrating astrocytoma may occur with little or no increase in intracranial tension with no localising signs and with minimum cerebral disturbance so that the patient is in good physical condition. Nevertheless the absence of papilloedema or headache, the normal cerebrospinal fluid pressure and constituents, the absence of any signs of intellectual or emotional upset and the completely negative neurological examination make the presence of a tumour very unlikely.

Cardiac syncope may often be differentiated generically from that due to cerebral causes. The cardiac case tends to have loss of pulse beats and pallor, clonic movements are rare, and there is no sense of rotation accompanying; the cerebral tends to have a normal or full pulse, not to be pale, and to suffer from clonic movements culminating in the severest cases in the characteristic picture of grand mal. While it is not rare for a minor or mild major epileptic attack to be dismissed as a "faint" it is also not unknown for a converse error to be made, with serious consequences for the patient. Vasovagal syncope is a familiar example of sudden and transient cerebral anaemia which though most frequently seen in children or adolescents may occur in the adult. The factors which give rise to it are the fasting state, fatigue, unpleasant emotions, debility following illness, and confinement in a hot, ill ventilated room. The sudden assumption of the erect position may/

may sometimes be sufficient stimulus ; a faint rarely occurs when the subject is lying down. The onset is sudden or preceded by unpleasant subjective sensations of nausea, dry mouth, weakness, and a heavy feeling in the abdomen. Pallor, feeble pulse and low blood pressure, flaccidity, dilated pupils and sighing respiration characterise the usually brief period of unconsciousness and there may be slight convulsive movements of face and limbs. Recovery is rapid. Vagal inhibition and splanchnic dilatation are the underlying pathology. In this case the gradual onset in recumbency, unusual length of unconsciousness, confusion, and incontinence are quite atypical of vasovagal syncope.

Mention may be made of aortic valvular disease of rheumatic or syphilitic origin which although not present in this case is a possible cause of fainting attacks. Other features - palpitations, angina pectoris pallor and capillary pulsation-are commonly present at such a stage, and the patient is all but bedridden.

Hypertension is another possible cause of minor cerebral attacks; these may consist of dizziness without rotation, faintness or actual brief loss of consciousness, transient confusion and loss of memory, paresis or paraesthesia. Visual disturbances from retinitis or intraocular haemorrhage occur. High blood pressure is not present in this case, though very occasionally transient hypertension occurs in rare suprarenal tumours (phaeochromocytoma).

The history of the attacks is not typical of paroxysmal tachycardia which begins and ends abruptly. It may occur at any age, but mainly in the twenties and thirties. Those afflicted by it often present no signs of disease or history of past illness. Rheumatic heart disease

is/

is the commonest association. Paroxysms are often provoked in those predisposed by exercise, flatulence or emotion. The symptoms proper to paroxysmal tachycardia are those resulting from the rapid and abnormal action of the heart. They vary chiefly with the rate of the heart beat (160-200) and the reaction of the nervous system. Attacks may pass unnoticed. Precordial discomfort, palpitation, flutter and throbbing in the neck are common. Pallor, sweating and tremulousness may occur. The attack ends often with a sharp stab of pain in the chest or a few forceful beats followed by speedy relief. If the attack is prolonged cardiac embarrassment may cause acute congestion and failure with dyspnoea, oedema and grossly enlarged and tender liver. Rigidity, abdominal pain and vomiting may be deceptive. In very few cases however does one attack end fatally. This patient's attacks were not abrupt in their onset and remission; he had no symptoms or signs of cardiac failure or indeed of palpitation.

Instances of syncope in which the heart fails to transfer incoming blood from the venous to the arterial side, are rare. This is cardiac syncope properly so-called and results when the ventricles cease beating and beat very slowly, or fibrillate. Such a condition is heart block, which can occur at any age. Commonly it is rheumatic in origin, sometimes syphilitic or degenerative in later life. Coronary arterial disease is the commonest single cause of high grade block. Minor grades of heart block, however, such as are responsible for intermittence of the ventricle, are commonly associated with infections, especially subacute rheumatism, diphtheria, influenza and pneumonia, when its occurrence is significant of a severe infection and it may be the only

outstanding /

outstanding sign of cardiac damage. In most of such infections the block is transient. Attacks of ventricular arrest - Stokes-Adams attacks - are associated with loss of consciousness, and almost always occur in cases of chronic heart block. In this case there was no pulse abnormality present and no clinical, radiological or E.C.G. evidence of cardiac disease.

Metabolic causes of attacks of unconsciousness include some which are not uncommon; diabetic coma, for example, may occur in a person as the first indication of disease. The main features of such coma are the smell of acetone in the breath and presence of acetone with or without glucose in the urine, flushing, a full bounding pulse and hyperpnoea. It is only relieved by administration of glucose, fluid and insulin. In this case the urine is free of both sugar and acetone, and none of the common symptoms - thirst, emaciation, sepsis, eye trouble - of diabetes are present.

Spontaneous hypoglycaemia is a not infrequent cause of subjective sensations similar but subtly different from those of the vasovagal attack. It may proceed to unconsciousness unless the necessity of taking castohydrate or at least fluid is recognised. The features are quite unlike those of this case.

Addison's disease is another cause which may be mentioned only to be dismissed. This patient does not suffer from low blood pressure or dehydration.

In such cases it is always as well, after excluding the more obvious causes, to entertain the idea of a functional basis for the symptoms, probably in the nature of a quasi-psychotic hysterical manifestation. In stupor seen typically in weary soldiers under bombardment the/

the patient lies motionless, non-resistant, sometimes incontinent of urine and faeces and without any predominant emotional tone. Confusional or delirious states may occur which are closely akin to the dissociation phenomena of fugue. The first episode recounted in the patient's history might possibly represent some such functional disturbance, especially in view of the treatment though the particular situation from which escape might be postulated as a motive is not clear. But the patient's personality, even allowing for the notorious fallibility of such impressions, and his previous history lend no colour to such speculations.

There remains the possibility, suggested by the patient's pyrexial attack and mild bronchitis on admission to hospital, that these fainting attacks represented in fact the response of a somewhat unstable vasomotor system to infection, influenzal infection in particular being a well-known cause of somewhat protean symptomatology such as is present here. An influenzal attack is prone to occur with comparative suddenness, and with little or no catarrhal symptoms. The victim may be prostrated in a few hours with rigor, aching pains, often headache, conjunctivitis and high pyrexia. Furthermore the defervescence is quite frequently by crisis. It is known also that the nervous systems of certain people are more prone to suffer upset from severe pyrexia and toxaemia than are others; particularly is this so with children, in whom convulsions or coma not infrequently accompany acute infections of any kind. It is probable that some adults are also hyperreactors.

At all events the main purpose of investigation was to exclude certain serious nervous and cardiovascular conditions which might manifest/

manifest themselves first in this way. Reassurance can be given the patient with a considerable degree of certainty that his symptoms do not betoken any serious malady. He should be warned to remain in bed should they again supervene until all symptoms have disappeared.