FOUR CASES
FROM A SINGLE GENERAL MEDICAL
OUTPATIENTS CLINIC

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Case presentations for the Wightman Prize in Clinical Medicine

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INTRODUCTION

The cases presented are those of four new patients seen at a single general medical outpatients clinic at Edenhall Hospital, Musselburgh. These cases are intended to demonstrate the extent to which patients can be investigated without hospital admission, and the selective use of hospital admission where it would be inappropriate to perform the investigations and initiate treatment as an outpatient.

All four cases loosely fall into the differential diagnosis of 'funny turns', two being of cardiac origin and two of neurological origin. Their management demonstrates the process by which serious illness is identified or excluded for each set of symptoms.

The three main questions which one is attempting to answer in an outpatient clinic are:

(1) Can this patient be dealt with on an outpatient basis, or is admission to hospital required?
(2) Are further investigations required to establish the diagnosis?
(3) Does this patient have a condition which requires treatment?
John Creelie is a fifty-four year old builder. He gave a history of being well until two weeks ago. Since then he had become aware of an occasional forceful beat in his heart. This was only present while he was relaxing and seemed to be more noticeable in the evenings. Then, nine days ago, he became aware of a more disturbing sensation while at work. He experienced bursts of up to ten beats at a rate of 160 beats per minute, (tapped out), intermittently for approximately half an hour. He stopped work and while walking home it settled. These episodes were not associated with any other symptoms such as chest pain, dyspnoea, or dizziness. Since then he has been fine, with no recurrence of his symptoms.

His only past medical history was the investigation of intermittent bouts of diarrhoea ten years ago. No cause was found and this subsequently settled with no treatment.

On systemic enquiry, he had none of the cardinal symptoms of any system. In particular he had no other symptoms of cardiac disease or of thyrotoxicosis. He smoked until recently and drinks alcohol in moderation. He denied undue anxiety but did consume at least six cups of tea per day, though no coffee.

On examination he appeared a genuine patient. His blood pressure was 150/80, pulse 84/minute and regular in time and force. His jugular venous pressure was not elevated and his apex beat was not displaced. There were no heaves or thrills to be felt on the precordium. The heart sounds were normal, with no added sounds. The peripheral pulses were all present, and there were no bruits apart from a venous hum audible over the right side of the neck. There was no evidence of cardiac failure. Nothing abnormal was found on abdominal or neurological examination. His fundi were normal and his thyroid
was not palpable.

The important factor in this case is to decide whether this tachycardia was of supraventricular or ventricular origin, as supraventricular tachycardias, usually, have no serious underlying cause, whereas ventricular tachycardias usually have underlying organic disease. In support of supraventricular tachycardia is the lack of associated symptoms during the palpitations, such as dizziness, fainting, dyspnoea and chest pain. The presence of these indicates that the heart is severely compromised by the increased rate to such an extent that it cannot maintain an adequate output.

A full blood count, erythrocyte sedimentation rate, serum urea and electrolytes, cardiac enzymes and random blood sugar were done as initial 'screening' tests. These were all normal. Although there was no clinical suggestion of thyrotoxicosis, the total serum thyroxine and the total serum tri-iodothyronine were measured as a check. As expected they were both normal. The chest X-ray showed hilar calcification but no evidence of cardiac enlargement or cardiac failure.

The primary investigations used in the diagnosis of arrhythmias are a resting electrocardiogram (ECG), an exercise ECG and ambulatory ECG monitoring.

In the case of tachycardia, the attack has often ceased before the patient has been seen by a doctor and an ECG recording taken. The ECG is, therefore, recording the subject's normal complexes, and so one is looking for signs which may give a clue to the type of tachycardia and the possible aetiology. These include:

1. **Extrasystoles** - the presence of multiple extrasystoles suggest a focus for initiation of tachycardia.
2. **Deformity of the P wave** may point to unidentified mitral valve disease or suggest intra-atrial conduction disturbances.
(3) Pre-excitation in the form of either Wolff-Parkinson-White syndrome, showing a short PR interval (less than 0.11 sec) and a widened QRS complex, due to the anomalous pathway which permits abnormal conduction between atria and ventricles, or the Lown-Ganong-Levine syndrome, in which the sole abnormality is shortening of the PR interval to 0.11 sec or less. Both of these syndromes are likely to facilitate supraventricular tachycardias.

(4) Lengthening of the QT interval may point to possible ventricular arrhythmias.

(5) Features of a healed myocardial infarction with persistent ST elevation indicate a possible ventricular aneurysm which may be the basis for ventricular arrhythmias.

(6) Sinus rhythm which is unduly slow may suggest sino-atrial disease with the possibility of alternating tachycardia and bradycardia.

(7) Other signs which may draw attention to the possibility of cardiac ischaemia, cardiomyopathy, chronic cor pulmonale, and a wide range of other disorders, which are all capable of producing arrhythmias but are less specific than those listed above.

In Mr. Creelie's case, none of these abnormalities was present. This is often found in cases of recurrent tachycardia. This presents a diagnostic difficulty, but supports the clinical impression that there is no underlying cardiac disease.

Exercise ECG testing provides a definitive physiological test for ischaemic heart disease as a cause of arrhythmias. Under these circumstances a positive endpoint of the test would either be the occurrence of the arrhythmia or the development of ischaemic signs. Mr. Creelie's exercise ECG test was normal.

Ambulatory electrocardiograph monitoring has allowed the identification of many arrhythmias which previously could only be guessed at. Nowadays, any patient suspected of having paroxysmal arrhythmia should have the opportunity
to undergo this, since in this way identifiable arrhythmias can be correlated with symptoms. Also the occurrence of asymptomatic arrhythmias may point to the explanation for palpitations experienced at other times.

Ambulatory ECG monitoring involves continuous recording, onto magnetic tape, of the ECG of a fully active individual over prolonged periods - 6 to 48 hours. The data is recorded by surface electrodes attached to areas of alcohol cleaned skin. The placement of the electrodes varies, but it is usually either a modified V5 position or a V1 like system, which facilitates identification of ectopic and aberrant beat patterns. The electrodes are attached to a portable battery-operated tape recorder which weighs 0.5-1 kg, and is worn on the hip using a belt.

The data analysis is achieved by a high speed replay of the tape with an oscilloscopic display of the recorded data and the facility of real-time print out onto standard ECG paper of any desired event. This sampling usually involves an operator, but may be automated. The detection of abnormalities is achieved by comparison of incoming complexes with a memorised or displayed normal QRST complex on the basis of RR interval, QRS configuration or area, and ST segment shift from the baseline. Thus, it can detect any abnormality in rate and rhythm.

The interpretation of this data is far from straightforward. There are the limitations of the system used, the reliability of the quantitative analysis artefacts and physiological variations to take into account. The significance of the data obtained is also often very difficult to determine. It varies according to whether the population studied consists of normal individuals or patients with particular disorders, as well as whether it is symptomatic or not.

In normal individuals arrhythmias are commoner with increasing age and also in men. The relevance of recorded abnormalities to the patients symptoms can be...
be assessed by showing the temporal relationship between them. This can be done either by the patient keeping a diary of the daily activities, events and periods of symptoms, or by using an electronic event-recorder operated by the subject which marks the appropriate point on the tape. In the absence of such correlation great caution must be taken before inferring cause and effect, since many asymptomatic arrhythmias may be encountered.

Mr. Creelie had ambulatory ECG monitoring for a period of 24 hours. During this time he was asymptomatic. However, the tape showed fairly prolonged bouts of supraventricular tachycardia. Since he had had previously symptomatic attacks, which historically suggested supraventricular tachycardia, it was decided to treat him with an anti-arrhythmic drug. He was started on atenolol 100mg daily, and arrangement made to see him in one month to assess the effectiveness of treatment.

On his return, he reported that he had been perfectly healthy, with no further symptomatic episodes of tachycardia and no side effects from the drug therapy. It is now left to the physician the task of deciding how long Mr. Creelie should remain on this treatment. The patient will be reviewed again in three months.
Jane Chapman is a fifty nine year old housewife. She gave a history of having been well until two weeks previously. While out walking with her sister at this time, she took a dizzy turn. She felt it coming on and linked arms with her sister to stop herself from falling. It passed off in a few minutes and was not associated with breathlessness, palpitations or chest pain.

In June 1980 she had experienced a similar 'funny turn', but at this time she was referred to the ophthalmic surgeons who informed her that there was a haemorrhage behind her right eye. The vision in this eye had been poor since it was hit by a tennis ball about fifteen years previously.

Her past medical history included attending the Royal Infirmary of Edinburgh at the age of 17 years, when she was told she had a heart murmur. The murmur was thought to be the result of rheumatic fever. For the last six years she has been treated by her own general practioner for hypertension. At the time of presentation this was being controlled with moduretic one tablet mane and methyldopa 250mg twice daily.

On systemic enquiry there was nil of note. In particular there were no symptoms suggestive of cardiac failure, thyrotoxicosis or systemic emboli.

Her family history included a brother with known hypertension who had had two non-fatal myocardial infarctions, and a brother who had died at the age of 31 years from a myocardial infarct. Mrs. Chapman was a nonsmoker and only drank alcohol occasionally.

On examination she was a pleasant, overweight, lady. Her radial pulse was 108/min, irregular in time and force, and there was a pulse deficit of 20 beats per minute.
Her blood pressure was 140/90. The apex beat was no palpable. On auscultation heart sounds I and II were judged to be normal. In addition there was a 2/6 pansystolic murmur maximal at the apex and a 1/6 mid-diastolic murmur at the apex. There was no accompanying opening snap. There was no evidence of cardiac failure. All her pedal pulses were intact. Abdominal and neurological examination were unremarkable. Both fundi showed silver wiring and slight arterio-venous nipping.

It seemed most likely, from the history and examination, that this patient had underlying rheumatic valvular disease affecting her mitral valve, and had now developed atrial fibrillation. It was decided to admit her to the Eastern General Hospital directly from the clinic, because of the life threatening potential of her condition. During this period of admission for anticoagulation and digitalisation, it was planned to carry out a few simple investigations.

On admission to hospital she was found to be hypokalaemic, with a serum potassium level of 2.8mmol/l. (It should be noted that she was being treated with a combined diuretic preparation, moduretic, which is theoretically potassium sparing.) It was, therefore, necessary to delay the commencement of digoxin until this had been corrected. The other serum electrolytes, including calcium and phosphate, were normal, as was the serum urea. Her liver function tests, serum cardiac enzymes and random blood glucose were, also, all normal. Her haemoglobin concentration was 15.1g/dl and her total white count and platelets were also within the normal range.

In patients with atrial fibrillation, thyrotoxicosis should be excluded even when alternative aetiological factors are present. Her total serum thyroxine, total serum tri-iodothyronine and serum thyroid stimulating hormone were all within the normal range. The tri-iodothyronine resin uptake test and thyrotrophin releasing hormone stimulation test were also normal.
The electrocardiogram showed atrial fibrillation, with left axis deviation and occasional ventricular ectopic beats.

The chest X-ray, postero-anterior projection and right anterior oblique position, showed the heart shadow to be a little enlarged. All chambers appeared to be involved but, in particular the right ventricle. There was some aortic unfolding but no evidence of valve calcification was detected.

An echocardiogram was performed by the cardiologists at the Western General Hospital. This showed evidence of rheumatic mitral stenosis, but good anterior excursion of the anterior mitral valve leaf. The posterior mitral valve leaf did not move anteriorly. The cusps did not appear symmetrically thickened or calcified. There was a rather enlarged left atrium. This, in the presence of the above signs, suggested dominant mitral regurgitation. The aortic valve was normal and there was reasonably good left ventricular function. The left ventricle was not enlarged.

Mrs. Chapman was commenced on warfarin using the dosage regime 15mg, 10mg, 5mg orally on three consecutive days and then adjusting the dosage according to the prothrombin time result on the third day. Within eight days all the investigations had been completed and she had been stabilised on 3mg warfarin per day. She was discharged on the following drug treatment:

- Warfarin 3mg daily
- Digoxin 0.25mg mane
- Amiloride 5mg mane

Three weeks later she attended Edenhall as an outpatient. She had been keeping well and her prothrombin time ratio was satisfactory.

Since then she has spent a trouble free two months in New Zealand. She took with her a letter from the consultant physician explaining her condition and listing her treatment. She continues to be seen, at intervals, at Edenhall hospital, but her own general practitioner monitors her prothrombin time ratio.
Starting a patient on long term anticoagulation therapy is not a step to be taken lightly. There are several reasons why commencing this treatment requires more consideration that most other drug treatments. There is a continual need for laboratory tests to guide the dosage of warfarin. In the initial days, after the loading doses, daily prothrombin time ratios are required to ensure the patient is being adequately anticoagulated without running the risk of unreasonably high ratios. There is a marked difference in individual response to warfarin, so that one regime of dosages will not suit all patients, and the final maintenance dose varies. There is the inconvenience to the patient of having to take regular tablets and having to have their blood checked regularly. Patient compliance is important, and there is a need for vigilance on the part of doctors in the matter of interaction with other drugs. All these are necessary because of the major adverse effect of bleeding. Even with these precautions it still remains a great risk.

In patients with lone atrial fibrillation embolic complications are uncommon, and anticoagulation can be omitted unless emboli occur. However, atrial fibrillation complicating mitral valve disease or thyrotoxicosis is an indication for immediate anticoagulant therapy.

Goulshed et al (1970) studied 693 patients with mitral valve disease. Of those with dominant mitral stenosis, 392 patients were in sinus rhythm and 31 of these had emboli (8%), whereas 109 of the 342 patients with atrial fibrillation had emboli (32%). In those with dominant mitral regurgitation 3 of the 39 in sinus rhythm had emboli (8%) and 14 of the 63 in atrial fibrillation (22%).

Therefore the single most important factor resulting in emboli from mitral valve disease is atrial fibrillation. In Mrs. Chapman's case the long term risk and inconvenience of anticoagulation therapy are far outweighed by the risk of embolisation.
John Aitken is a sixty one year old shop manager. He said he had been in perfect health until two weeks previously. At this time he woke with 'pins and needles' in his left arm and thought he must have slept on it. He sat on the edge of the bed and rubbed the arm, then got up and fell. He managed to struggle to his feet, only to fall again. He then realised that his left leg was also numb and weak. He spent the next few days in bed having to use a stick to walk to the toilet as the weakness persisted for over two days and the numbness for ten days. By the time he attended the outpatient clinic he had completely recovered.

He had no past medical history of note. Prior to this episode, he insisted that he had enjoyed good health all his life and was in the habit of playing two rounds of golf a week. He used to smoke forty cigarettes a day, until this episode, but has now stopped. He denied undue breathlessness, palpitations, chest pain or discomfort. There was no significant family history other than the fact that his mother is still alive and his father only died last year at the age of 93 years.

On examination this patient was found to be overweight. His pulse was 84/min, regular in time and force, and his blood pressure 140/75. The jugular venous pressure was not elevated and his apex beat was not palpable. Heart sounds I and II were normal and a 3/6 ejection systolic murmur was heard all over the precordium. There was no early diastolic component. He had a soft bruit over his aorta but this may well have been transmitted. There were no other major vessel bruits. Dorsalis pedis was not felt in either foot but he had strong posterior tibial pulsation. All other pulses were intact. His chest was clear and there was nothing to find on abdominal examination.

On neurological examination no abnormality in power, light touch, pin prick
or vibration could be found. There was no sensory inattention. Both plantar responses were flexor. All the reflexes were present and there was no impressive difference between the right and left side.

Serum urea and electrolytes and liver function tests were all normal. The full blood count was normal with a haemoglobin concentration of 14.5g/dl, thus excluding polycythaemia. The erythrocyte sedimentation rate was 14mm/hr. in the first hour. His random blood glucose lay within the normal range and his glucose tolerance test was non-diabetic.

The electrocardiogram showed no evidence of cardiac dysrhythmia or myocardial ischaemia, although there was peaking of the T wave in V2-4 but within normal limits. The chest X-ray excluded cardiac enlargement and there was no evidence of bronchial carcinoma, metastases, or calcification or unfolding of the aorta. The cervical spine X-ray showed some narrowing of the sixth disc space with no loss of alignment and no features which would suggest cord compression. The skull X-ray was normal.

The Venereal Disease Research Laboratory and Treponema pallidum haemagglutination tests were both negative. It is always important to exclude syphilis, particularly in the elderly, as it is a great mimicker of all types of neurological disorder.

A computed tomogram showed minor cortical shrinkage, particularly in the upper frontal regions but no focal features were present, other than the fact that, if anything, the left ventricle was minimally larger than the right.

Stroke is a major cause of devastating disability and of death in this country. For every 1000 of the population there are 3 strokes per year, one of which is fatal. Of those surviving 70% have obvious neurological deficit after maximal recovery. The prevention of strokes is therefore of great
importance to society.

At present there are only three types of treatment aimed at the prevention of strokes. These are anticoagulation, antiplatelet drugs and vascular surgery. In investigating a patient who is at high risk of a stroke the major decision is whether or not to proceed to the invasive investigation of angiography. It is impracticable and unrewarding to carry out arteriography in all patients who develop focal neurological signs. It is particularly indicated in the younger patient who develops evidence of cerebral infarction, especially if they make a full recovery from the first attack. It is mandatory when there is evidence of extracranial vascular disease, aneurysm, or other vascular malformations.

Mr. Aitken had previously been a fit and active man. Therefore, especially since the computed tomogram had shown no clear evidence of haemorrhage or infarction, it was decided to proceed to cerebral angiography. This required admission to the neurological department of the Western General Hospital overnight. Cerebral angiography is not without significant risk as it can lead to morbidity including hemiparesis and mortality. This is thought to be due to cerebral arterial spasm. This risk should be less than 1% and can be minimised by the practise of neuroradiodiagnosis by experts in this field. Cerebral angiography is done to establish the presence of proximal carotid stenosis which might be operable, or to show vascular anomalies which, again, may be remedied surgically. The operation most often performed for proximal carotid stenosis is endarterectomy.

In Mr. Aitken's case there was no evidence of arterial stenosis though possibly there was a reduction in the vascularity of the right parietal area. The other treatment options were, therefore, anticoagulant therapy or antiplatelet drugs. The reasoning behind the use of these is that the majority of strokes are caused by thromboembolism. The aim is to decrease thrombus formation. This has
limitations in that the mechanism of thrombus formation in arteries differs from that in veins. Dicoumarol anticoagulants have proved more effective against venous thrombosis. Also cerebral artery emboli are of varied composition. There are the microemboli, composed mainly of platelets, variably stabilised by fibrin formation, which may detach from thrombi on ulcerated atheromatous lesions in the neck. There is also embolic material from the cardiac chambers in rheumatic heart disease and ischaemic heart disease, which is composed of more stable fibrinous masses. Occasionally, fragments of scarred heart valves are detached as emboli. Finally atheromatous debris, including cholesterol crystals, may form emboli. Therefore the pharmacological inhibition of platelet function or fibrin formation cannot be equally effective in all these situations. Also, it is often impossible to identify the kind of emboli in a particular patient.

Anticoagulants have been used in patients with transient ischaemic attacks, but although a decrease in the frequency of these attacks was shown, their effectiveness in stroke prevention remains unproven. It should be remembered that the major cause of death in patients who have transient ischaemic attacks is myocardial infarction.

In the emboli from the thrombi in diseased neck vessels there is an essential role of platelet reaction with the vessel wall. The same is true of thrombi formation within the cerebral vessels. Therefore inhibition of platelet function, for example with aspirin, has been tried. This is simpler than anticoagulant therapy and there are fewer side effects, especially as only a small dose is required. Trials show a trend to decreasing numbers of strokes and deaths, but this has not been statistically significant.

Mr. Aitken was commenced on 300mg of soluble aspirin daily. He has been well since this with no recurrence of symptoms.
Mary Storrie is a forty year old housewife. She was seen with her husband in the clinic. Her husband described episodes of her falling to the ground. She would go rigid for ten minutes and then start kicking and throwing her arms all over the place. This was accompanied by frothing at the mouth and pallor. There had been about six of these episodes in the last year. He thought they were becoming more frequent.

There was no history of urinary incontinence and biting her tongue was impossible as she has no teeth. On two occasions she has hurt herself, once lacerating the right side of her forehead. She has no memory of these episodes herself, but she complained of severe and persistent headache following these episodes which might last all day.

These 'turns' were unrelated to menses and did not appear to be related to alcohol consumption. Both Mrs Storrie and her husband agreed that they may come on during periods of increased tension or anxiety.

She admitted to a productive cough and to smoking more than twenty cigarettes a day. She had nocturia (four times every night) but without other urinary tract symptoms. She also said that for a long time she has had trouble with intermittent discharge from her right ear. She complained of heartburn.

On examination she was judged to be of rather low intelligence. Her pulse was 80/minute, regular in time and force and blood pressure 130/80mm Hg. Heart sounds I and II were present and normal, and there were no added sounds. Her chest was clear and there was nothing of note on abdominal examination.
Her reflexes were all present and equal. There was no sensory or motor deficit in her limbs or in her cranial nerves apart from the fact that air conduction was impaired in the right ear and on examination she had a perforated right tympanic membrane.

This patient had been referred to the Consultant Physician in 1971 with a history of similar 'turns'. At this time she said the attacks were related to her menstrual periods, but she was apparently having three periods per month. No underlying pathology was found to account for these episodes. She was started on diazepam.

On all of these occasions the only witness of these attacks has been her husband. The General Practitioner commented in his letter that there was a history of many dramatic domestic crises, fights, overdose of pills, and psychiatric admissions. However, there have been fewer of these crises brought to her notice in recent years.

In the initial investigations, blood urea and electrolytes, liver function tests, calcium, phosphate, alkaline phosphatase and random blood glucose all proved to be normal. Her haemoglobin was 14.9g/dl and the total white cell count and erythrocyte sedimentation rate were normal. This does not exclude a cerebral abscess as, although an acute abscess may be accompanied by fever, malaise and fever (seldom more pronounced than could be accounted for by the original source of infection), in chronic abscess there may be no clinical evidence of infection at all.

Urinalysis was negative and a midstream specimen of urine sterile. Serological tests for syphilis were not performed, which was possibly an omission. The electrocardiogram was normal and the chest x-ray unremarkable, showing no evidence of a primary lung tumour, metastases or abnormality of the cardiac contour. A plain skull x-ray showed the mastoid to be sclerotic but there was no intracranial calcification, or features of raised intracranial pressure.
such as erosion of the dorsum sella.

The electroencephalogram (EEG) was within normal limits. This does not exclude epilepsy as many patients with epilepsy have so-called normal interseizure recordings. Conversely, 10-15% of the population with no history of seizures may have abnormal recordings. However, a spike focus or paroxysmal discharge strongly supports the diagnosis of epilepsy. A slow-wave focus may indicate a structural lesion, such as a tumour or abscess, and suggests the need for further investigation.

An appointment was made for this patient to attend the Radiology Department at the Western General Hospital for computed tomography.

In the case of Mrs. Storrie, although the history was a little atypical in that the tonic phase of the seizure apparently lasted for ten minutes, with no cyanosis, it was felt that she must be treated as a case of adult onset epilepsy.

The onset of primary generalised epilepsy, otherwise known as idiopathic epilepsy, is rare under the age of 4 years. The peak age of onset is 8 to 12 years with a decline in the number of new cases up to the age of 20 years, after which onset is rare or problematic.

The onset of epilepsy in adult life must be presumed to be of focal origin even if there is no focal element in the clinical features of the individual attacks. Focal epilepsy can result from virtually any form of damage to the cerebral cortex. For example cerebral tumour, angiomatous malformation, cerebral abscess and cortical scarring as a result of meningitis may all be causes. Also head injuries and cortical atrophy due to Alzheimer’s disease can be the cause of epilepsy.
Middle ear infection is a relatively common cause of brain abscess, the great majority being in the temporal lobe. Symptoms may include those of raised intracranial pressure and the focal features of the lesion. The EEG will show a gross focal discharge of slow delta waves from the region of the abscess in the cerebral cortex.

Other causes of epilepsy include alcohol either during a bout of excessive drinking or on withdrawal. The frequency of fits may increase with the menstrual period or be precipitated by late nights, but overwork and stress are not regarded as precipitating factors.

Primary generalised epilepsy is thought not to present in adult life, but there remain a substantial number of adults who develop infrequent major fits, in whom thorough investigation and prolonged observation fail to reveal any cause for epilepsy. A small proportion of those thoroughly investigated will eventually be found to have a cerebral tumour. It should be remembered, however, that while meningiomas are best removed while small, little is to be gained by operating on astrocytomas causing epilepsy alone. Symptoms and signs of raised intracranial pressure, or of increasing disability from focal brain damage will naturally necessitate thorough investigation.

Mrs. Storrie returned to the follow-up clinic at Edenhall on 1/2/82. She had failed to attend the appointment for computed tomography at the Western General Hospital, apparently because her house had been flooded. She said she had had a further 'turn' 10 days previously, when she had fallen without warning hurting the left side of her face and her right arm. She thought she had been unconscious for thirty minutes.

It was decided, because of the apparent damaging effect of these 'turns', to start Mrs. Storrie on an antiepileptic drug. She was therefore
commenced on phenytoin 100mg twice daily. An appointment was made for her to see an ENT surgeon and another appointment for her to attend the Radiology Department at the Western General Hospital for the computed tomography scan.

Since then Mrs. Storrie has failed to attend three ENT appointments, two appointments for computed tomography, and two follow-up clinics at Edenhall, the last of these expected attendences being on 24/5/82. Despite her failure to attend, it has been established that she has been keeping reasonably well. Her non-attendance may be considered to be due to her rather low intelligence, or may support the view of the physician that these attacks were of a non-organic nature.
CONCLUSION

Outpatient clinics have always been a major part of the service offered by hospital physicians. During the last few years, the development of non-invasive techniques of investigation, such as computed tomography and ambulatory electrocardiogram monitoring, has enabled extensive investigation as an outpatient as well.

This has reduced the expense of investigation by eliminating the need for hospital admission, except for invasive techniques. It causes less inconvenience to the patient who is fit to carry on with normal life, and avoids the trauma of unnecessary institutionalisation.

This is illustrated by these four cases from a single outpatient clinic. Two of these were investigated entirely as outpatients, one was investigated extensively as an outpatient, only being admitted to hospital overnight for an invasive investigation and one patient was admitted directly from the clinic to hospital because of the life threatening potential of her condition.

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