exercise during a 12-minute walk. The ventilatory response to hypoxia is potentiated during exercise in both normal subjects¹⁰ and most patients with severe chronic obstructive lung disease.³ Correction of this hypoxaemia during exercise might therefore be expected to reduce their minute volume at any given work load and thus render them less breathless. We have shown that this did occur in 12 of the 15 patients we have studied. In addition to this reduction in ventilatory drive during exercise, correction of hypoxaemia will also improve oxygen delivery to working muscles, so that the more hypoxic a patient becomes on exercise, the more adequate correction of the arterial hypoxaemia should improve exercise tolerance.

In this single-blind study we have shown that oxygen at 2 or 4 l/min administered through nasal prongs (corresponding to an inspired oxygen concentration of about 30% or 45% when at rest) will improve exercise tolerance in a selected group of patients with chronic hypoxic cor pulmonale. Nevertheless, in these patients, whose exercise tolerance is severely limited, even the small extra load of carrying the 4.5-kg oxygen walker is sufficient to completely offset the advantage obtained by breathing 2 or 4 l of oxygen/min when walking. This disadvantage of the patient carrying his own oxygen supply can be overcome when he wheels the oxygen walker on a lightweight trolley. Although the increase in distance walked within the 12 minutes was small ($59 \pm (\text{SD}) 42$ metres), in the more severely disabled this may amount to 25% of the distance that they can walk in 12 minutes when breathing only air. When we considered only those patients whose 12-minute walking distance was less than 750 metres the mean increase in distance walked when breathing 4 l of oxygen/min, but wheeling the oxygen walker, amounted to 87 ± 25 metres. It seems probable that if the oxygen flow rate from the nasal prongs were increased above 4 l/min there might be further improvement in the arterial Po_2 , for even at this level there was still significant hypoxaemia (Po_2 less than 8.0 kPa (60 mm Hg)) during exercise in two of our five patients. Nasal prongs, delivering oxygen to the anterior nares both in inspiration and expiration, are obviously a relatively inefficient means of supplying oxygen, but they have the outstanding advantage that they are well tolerated by patients, in contrast to a tight-fitting face mask, which although mechanically more efficient, cannot be tolerated by these patients for any length of time.¹¹

Our studies suggest that oxygen during exercise can benefit

severely hypoxic patients by allowing a small but statistically significant increase in the distance walked within 12 minutes. But we also found that provision of such oxygen from a patient-carried system, the Union Carbide Oxygen Walker, although convenient and practicable, does carry the disadvantage that the extra weight of the equipment hinders the patient's performance. We suggest that wheeling the oxygen walker on a simple, cheap, lightweight trolley will allow these breathless patients to derive benefit from oxygen during exercise, in addition to the undoubted benefit that they already obtain from having a portable supply of oxygen, which allows them to obtain oxygen therapy for at least 15 hours of the day, a period known to be necessary if objective benefit is to be obtained from long-term domiciliary oxygen therapy.^{6, 12} Our results indicate that the challenge of providing this physiological benefit of oxygen on exercise from a patient-carried device remains a problem that the bioengineers have yet to solve.

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Visual complications of mitral leaflet prolapse

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Summary

Four young women and six older men with mitral leaflet prolapse presented with visual disturbances consistent with embolism in the ophthalmic or posterior cerebral circulation. Cardiac arrhythmias were common, but

these are rarely associated with focal ischaemia. The evidence that mitral leaflet prolapse caused the embolism in these patients is suggestive but not conclusive. Further studies are needed. All patients with acute cerebral or ocular ischaemia should undergo thorough cardiovascular assessment, which should include routine echocardiography.

Introduction

The symptoms and physical signs associated with mitral leaflet prolapse have recently aroused much interest.^{1, 2} Many patients with this condition have no symptoms, but some experience chest pain, palpitation, fatigue, or dyspnoea. Significant mitral regurgitation, infective endocarditis, serious arrhythmias, and, rarely, sudden death may occur. Malcolm *et al*³ reviewed 85 patients with mitral leaflet prolapse, 11 of whom presented with

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neurological symptoms. Barnett *et al*⁴ had earlier described six of these patients with cerebral ischaemic events attributed to emboli and subsequently added further cases.⁵ We also have found evidence that mitral leaflet prolapse may be associated with embolism. We report here on 10 such patients who were seen during the last two years with acute ischaemic events in the eye or brain that produced visual loss.

Case reports

The findings in the 10 patients are summarised in the table. The following case reports describe the features of two of the patients in more detail.

Case 2—While walking this 62-year-old man experienced two episodes of vertigo, each lasting about five minutes and followed by frontal headache. The next morning vision with both eyes was obscured as though by fog. This continued for 24 hours and was associated with headache. Thereafter vision improved, although he continued to have difficulty in seeing to the left. Examination showed a left homonymous inferior quadrantanopia. An apical holosystolic murmur was heard. Computerised axial tomography scanning showed a small low-density lesion in the right occipital cortex. An electrocardiogram confirmed atrial fibrillation and an echocardiogram showed pansystolic mitral leaflet prolapse with a pronounced “U-shaped” pattern.⁶

Case 9—A 45-year-old man said that he had had recurring episodes of transient loss of vision in the left eye for 12 years: “Like a blind being drawn over the eye from the top.” Each episode lasted one to five minutes. There were no associated symptoms. A midsystolic murmur was audible at the apex only in the left lateral position on expiration. Examination otherwise showed nothing abnormal, and a left carotid angiogram was normal. Twenty-four hour electrocardiographic monitoring showed episodes of supraventricular tachycardia (160/min) and sinus bradycardia (53/min). An echocardiogram showed late systolic mitral leaflet prolapse. Subsequently he was anticoagulated with warfarin and the attacks stopped.

Discussion

These four young women and six older men presented with visual disturbance consistent with acute ischaemia due to embolism in the ophthalmic or posterior cerebral circulation. Four of the 10 presented with recurring episodes of uniocular amaurosis fugax, two with retinal artery occlusions and persisting visual deficit, and four with homonymous field defects. No patient had a history of rheumatic fever, none had evidence of polycythaemia or arteritis, and none had a carotid artery bruit.

There is clinical^{7, 8} and pathological^{9, 10} evidence that amaurosis fugax is due to the passage of emboli through the ocular circulation. An abnormality of the appropriate carotid artery is found at angiography in only about a third of the unselected patients so presenting.¹¹ Of our four patients with repeated episodes of amaurosis fugax three underwent angiography and in all three the ipsilateral carotid arteriogram was normal. A cardiac source for emboli is therefore often suspected. Similarly an embolic basis was probable in the two patients with retinal artery occlusions. One patient had a branch occlusion, and pathological studies¹² in such cases have suggested that these are more often due to embolism than to local occlusive

disease. The other patient, with a central retinal artery occlusion, was a young woman with no other evidence of occlusive disease. The four patients with homonymous field defects were all normotensive and without histories of migraine; one had also had repeated episodes of transient cerebral ischaemia with limb symptoms. We have encountered two other patients, not reported here, with retinal vein thrombosis and mitral leaflet prolapse. Although Woldoff *et al*¹³ reported a similar association and suggested an underlying retinal artery embolus, the relation between venous and arterial occlusion remains uncertain.¹⁴

Cardiac arrhythmias are common in patients with mitral leaflet prolapse,^{2, 3, 15} and this was so on 24-hour ECG monitoring in the patients reported here. But it is rare for focal cerebral ischaemic events to be caused by such disturbances, with their associated reduction in cardiac output.¹⁶ Moreover, cerebral ischaemia associated with cardiac arrhythmias is global rather than focal in its effect,¹⁷ and in patients with mitral leaflet prolapse there is a poor temporal correlation between symptoms and recorded arrhythmias.¹⁸

Investigation in each of these 10 patients with probable embolic events found no abnormality other than the mitral leaflet prolapse, though one woman was taking oral contraceptives. None of our patients reported specific cardiac symptoms. In one there was no auscultatory abnormality; in several the abnormality was not heard at the initial examination, and elicited only with proper positioning. Careful clinical examination and echocardiography are required when this condition is suspected. Left ventriculography is the definitive investigation and may show the abnormality even when the mitral echogram is normal.³

Several cardiac disorders cause embolism.¹⁹ Mitral leaflet prolapse is common,² and if it causes emboli, as Barnett^{4, 5} has suggested, it may be the mechanism producing ischaemia in some of the many patients in whom routine investigations have revealed no cause. The site of origin in the heart for such emboli in this condition remains speculative. Occlusion of any of the main cerebral arteries may result from emboli from a left atrial thrombus from rheumatic mitral valvular disease and from mural thrombus in the left ventricle overlying infarcted myocardium. The repeated transient ischaemic episodes of some of the patients described here are, however, more consistent with transient occlusion of smaller arteries. Such episodes have been reported in patients with rheumatic valvular disease²⁰ and are attributed to microemboli from the fibrin and platelet thrombi commonly seen on the affected valves. Small areas of ulceration and thrombus deposition have been seen on mitral leaflets from patients with leaflet prolapse,²¹ and such thrombi might have been the source of emboli in our cases.

Although we have noted the association of visual loss and mitral leaflet prolapse in these 10 patients, we have not performed echocardiography in every patient with such ischaemic episodes, and we do not know the prevalence of mitral leaflet prolapse in our community. Our data, though suggestive, do not therefore firmly establish a causal relation. Prospective echocardiographic and laboratory indices of platelet consumption should help clarify the significance of this association and determine whether treatment aimed at reducing the risk of embolism is warranted. Meanwhile, pending the results of current co-operative studies of the effect of such drugs on recurrence of cerebral ischaemic

Details of 10 patients with visual loss and mitral leaflet prolapse

Patient Case No	Age and sex	Visual problem	Cardiac findings	Mitral echogram	Left ventriculogram
1	52 M	Amaurosis fugax	Normal	Mitral leaflet prolapse	
2	62 M	Homonymous field loss	Systolic murmur	" " "	
3	62 M	Homonymous field loss	Systolic click and murmur	" " "	Mitral leaflet prolapse
4	34 F	Retinal artery occlusion	" " "	" " "	
5	21 F	Homonymous field loss	" " "	" " "	
6	49 M	Retinal artery occlusion	" " "	" " "	
7	24 F	Amaurosis fugax	" " "	Normal	Mitral leaflet prolapse
8	64 M	Homonymous field loss	" " "	Normal	
9	45 M	Amaurosis fugax	Systolic murmur	Mitral leaflet prolapse	
10	16 F	Amaurosis fugax	Systolic click and murmur	" " "	Mitral leaflet prolapse

events, we think that the evidence is sufficiently suggestive to prescribe drugs affecting platelet function such as aspirin and sulphinpyrazone. These observations emphasise the importance of thorough cardiovascular assessment in all patients with acute cerebral or ocular ischaemia, and we suggest that echocardiography should be part of the routine investigation of such patients.

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Television epilepsy and pattern sensitivity

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Summary

Properly functioning domestic television sets may induce seizures in epileptic patients (TV epilepsy). We investigated the effects of different types of visual stimuli on paroxysmal electroencephalographic (EEG) activity in 32 epileptic patients known to be sensitive to intermittent photic stimulation (stroboscopic light). We monitored sensitivity to patterns of horizontal and vertical lines, both stationary and vibrated (pattern sensitivity), and to normal broadcasts on a domestic, black and white (405- or 625-line) TV receiver (TV sensitivity). Twenty-three of the 32 patients were sensitive to pattern. Twenty-two were sensitive to vibrated patterns, and 11 to static patterns ($P < 0.01$). All patients sensitive to pattern were also sensitive to TV. The association between sensitivity to pattern and to TV was significant. Clinical history of TV epilepsy (16 out of 32 patients) and laboratory evidence of pattern or TV sensitivity were not significantly associated. The high incidence of pattern sensitivity

among flicker-sensitive patients and its association with TV sensitivity suggests that linear patterns produced by the raster of a black and white set as it scans, or "line-jitter" produced by the raster in areas of low TV-signal strength may contribute to the epileptogenic effect of TV.

Introduction

In patients with a history of epileptic seizures apparently induced by watching television (TV epilepsy) stimulation with stroboscopic light (or "flicker") induces paroxysmal electroencephalographic (EEG) activity.¹ Conventionally TV epilepsy is attributed to flicker, either the slow flicker that occurs when the TV picture slips²⁻³ or the 50-Hz component present when the set works normally.¹

Not all patients with TV epilepsy show EEG sensitivity to 50-Hz flicker, and we have rarely found any evidence that the set was malfunctioning at the time of the fits. We therefore decided to investigate other mechanisms by which TV might induce seizures, particularly the linear patterns which form the TV picture.

Patients and methods

We studied 32 patients found to be flicker-sensitive on routine clinical EEG investigation during a period of 16 months in 1975 and 1976. These patients formed a consecutive series, excluding 12 who attended when the necessary research facilities were not available. The mean age of the subjects was 13 years (range 6-31), with a 19:13 preponderance of women. Thirteen of the patients had had previous EEG and were known to be sensitive to light. All were referred because of known or suspected epilepsy, and 19 were receiving anticonvulsant treatment. Sixteen patients gave a history of major or minor seizures associated with TV viewing and in none of these were the attacks associated with known malfunction of the TV set. Only six of the 16 patients were close to the screen whenever seizures occurred. As most patients with known or suspected epilepsy who attend hospitals in the Southend and Basildon areas are referred to our EEG department for

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