Limitations of transoesophageal echocardiography in patients with focal cerebral ischaemic events

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Abstract

Objective—To investigate the detection rate of cardiac sources of embolism by transoesophageal echocardiography in patients with focal cerebral ischaemic events and to relate the echocardiographic findings to other clinical findings.

Design—Prospective study with blinded analysis of the echocardiographic data and subsequent comparison with the other clinical findings.

Setting—Regional cardiothoracic unit based in a teaching hospital.

Patients—131 consecutive patients with focal ischaemic cerebral events (49 with a transient ischaemic attack, 77 with a cerebrovascular accident, and five with a retinal arterial embolus) referred for echocardiography.

Interventions—Full M mode, cross sectional, Doppler, and contrast echocardiography by both the precordial and transoesophageal techniques.

Results—Precordial echocardiography detected a cardiac abnormality in 72 patients. Transoesophageal echocardiography confirmed all the precordial findings (except left ventricular hypertrophy, which at present cannot be defined with this technique) and detected other abnormalities in a further 20 patients (18 with potential right-to-left shunts and two with valve vegetations). It also showed spontaneous contrast echoes in 27 of 28 patients with a large left atrium and showed atrial thrombus in three. Cardiac abnormalities were clinically detected in 53 patients, all of which were confirmed or documented by echocardiography. In the 78 patients with no clinically detectable cardiac abnormality six had mitral valve prolapse and one had a regional wall motion defect (identified by precordial echocardiography) and 17 had potential right-to-left shunts (11 of which were identified only by transoesophageal echocardiography).

Conclusions—Transoesophageal echocardiography is more sensitive than precordial echocardiography in detecting potential sources of embolism in these patients. However, except for the detection of a potential right-to-left shunt, the yield in patients with no cardiac abnormality is low. Moreover, the abnormalities detected in those with previously detected cardiac disease merely confirm the clinical diagnosis. Patients with left atrial spontaneous contrast echoes may benefit from anticoagulation but this requires further study. Until more data are available on this feature and on the role of potential right-to-left shunts in this population, the contribution of echocardiography, precordial or transoesophageal, remains limited.

Clinical studies suggest that emboli arising from the heart cause 5–20% of focal cerebral ischaemic events.[1-3] Necropsy data suggest that emboli may be responsible for up to 50% of fatal events.[4 5] Though these data may suggest that cardioembolic events are more often fatal than other causes of focal cerebral ischaemia, another interpretation is that it is difficult to detect cardiac sources of emboli in patients who have focal cerebral events. The development of M mode and cross sectional echocardiography has enhanced our ability to detect cardiac abnormalities in these patients,[6-8] but the sensitivity and specificity of these methods for the detection of potential embolic sources (for example, thrombi) are not known. In this group of patients precordial echocardiography has other deficiencies. Firstly, in quite a few it is not possible to obtain adequate images. Secondly, abnormalities that are detected (for example, rheumatic mitral valve disease) have usually been detected clinically. Although the image quality in such cases is inadequate for the detection of the associated left atrial thrombus, the presence of the latter is assumed and the patient is treated with oral anticoagulants. Lastly, the detection of an abnormality does not prove a pathogenic mechanism for the neurological event.

The development of transoesophageal echocardiography[9] allows evaluation by high frequency ultrasound of the left atrium including its appendage, areas that are not imaged well from the precordium.[10 11] The left atrial appendage in particular is a common site for thrombus[12 13] and the transoesophageal technique has proved useful for its detection.[14]

Although some workers have reported on the enhanced detection of cardiac sources of emboli with transoesophageal echocardiography,[15-17] many of the limitations of precordial echocardiography in this group of patients also apply to the transoesophageal technique and it is not clear what impact this imaging method will have on decisions about treat-

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ment in individual patients. This study was therefore performed prospectively to evaluate the information provided by transoesophageal echocardiography in relation to earlier clinical information in a series of patients presenting acutely with a focal cerebral ischaemic events. Patients presenting with any of the following were considered for study: transient ischaemic attack, reversible ischaemic neurological deficit, cerebrovascular accident, or retinal arterial occlusion.

Patients and methods

PATIENTS

We studied 131 consecutive patients with a recent focal neurological ischaemic event who were referred for echocardiography. Echocardiography was performed within two weeks of the presenting neurological event in all cases. Before echocardiography, relevant details of the history and clinical examination were recorded. The underlying cardiac rhythm was recorded and the case notes examined for evidence of paroxysmal arrhythmias. A raised blood pressure on presentation was not taken as evidence of hypertension, which was defined as (a) blood pressure requiring specific antihypertensive medication before the presenting event or (b) raised pressures on admission that persisted for more than a week, either associated with electrocardiographic evidence of left ventricular hypertrophy or requiring antihypertensive treatment. Cerebrovascular disease was defined as the presence of a carotid bruit (10 patients), Doppler evidence for an internal carotid stenosis (four patients), or cerebral angiographic evidence of a vascular abnormality consistent with the presenting event (two patients). Duplex studies of the carotid arteries were not routinely performed in these patients. A past history of myocardial infarction and confirmatory electrocardiographic evidence were noted. To be a risk factor for the presenting event, the myocardial infarction had to have occurred in the three months before the neurological episode. Clinical diagnoses of infective endocarditis, hypertrophic cardiomyopathy, and dilated cardiomyopathy were recorded.

All patients with a cerebrovascular accident had been previously assessed by a consultant neurologist or senior physician in our centre. All such patients had undergone computed axial tomography of the head. Patients with cerebral haemorrhage or space-occupying lesions were excluded. The protocol had the full agreement of the local ethics research committee.

PRECORDIAL ECHOCARDIOGRAPHY

All patients had full M mode and cross sectional echocardiography in standard imaging planes, with relevant Doppler studies within two weeks of the presenting neurological event. A Hewlett-Packard 77020 or 77030A ultrasound system was used, series Sonos 500 or 1000. Images were obtained with a 3·5 MHz transducer, but if these were suboptimal a 2·5 MHz transducer was used. Standard measurements of the left atrium and ventricles

were made and ventricular function was assessed. Mitral valve prolapse was defined according to the clinical and echocardiographic criteria described by Perloff and colleagues.[18] At the end of the routine study a precordial contrast echocardiogram was obtained. An 18G Venflon (Viggo Products, Helsingborg, Sweden) was inserted into a left antecubital vein. With 10 or 20 ml syringes, 1-2 ml of blood was drawn into the syringe and 8–18 ml of physiological saline was added. Air was drawn into the syringe and the solution agitated either by vigorous shaking or by rapidly injecting it between two syringes via a three-way tap. The air and macrobubbles were then expelled and the resultant solution containing microbubbles was injected rapidly. Recordings of an M mode study across the mitral valve during the injection were made on video tape and a hard copy print out was obtained. Cross sectional images were recorded on video tape in the apical four-chamber view or if this was unsatisfactory in a subcostal four chamber view. Recordings of the contrast studies were made before and during a Valsalva manoeuvre. The release phase of the manoeuvre coincided with the arrival of contrast in the right atrium.

TRANSOESOPHAGEAL ECHOCARDIOGRAPHY

The transoesophageal study was performed immediately after or, if this was not possible, within three days of the precordial study. The patient was fasted for four hours before the study which was performed in the left lateral position with topical hypopharyngeal anaesthesia (10% lignocaine solution) and, in a few patients, mild sedation (2·5–10 mg midazolam).[19] After a complete anatomical examination the transducer was positioned with the scanning plane at the level of the atrial appendage. The gain and compression controls were modified to enhance weak echoes, thus optimising the ability to detect spontaneous contrast echoes (fig 1). The transducer was then rotated so that the scanning plane was at the level of the foramen ovale. Colour flow mapping was performed to evaluate the possibility of a shunt across the atrial septum. After this a contrast study was performed both before and during a Valsalva manoeuvre as described above. The following diagnostic definitions were used:

(a) Mitral valve prolapse—superior systolic displacement of the anterior or anterior and posterior leaflets above the level of the mitral annulus as seen in the four chamber view, irrespective of the position of the coaptation point or superior displacement of the posterior annulus together with superior displacement of the coaptation point.

(b) Atrial septal defect—evidence of left-to-right shunting on colour flow mapping or a "negative" right atrial contrast study irrespective of the detection of an anatomical defect in the atrial septum on two dimensional imaging alone.

(c) Patent foramen ovale—Anatomically intact atrial septum, no left-to-right shunting on colour flow mapping, right-to-left shunting detected on contrast study or by colour flow mapping (fig 2).
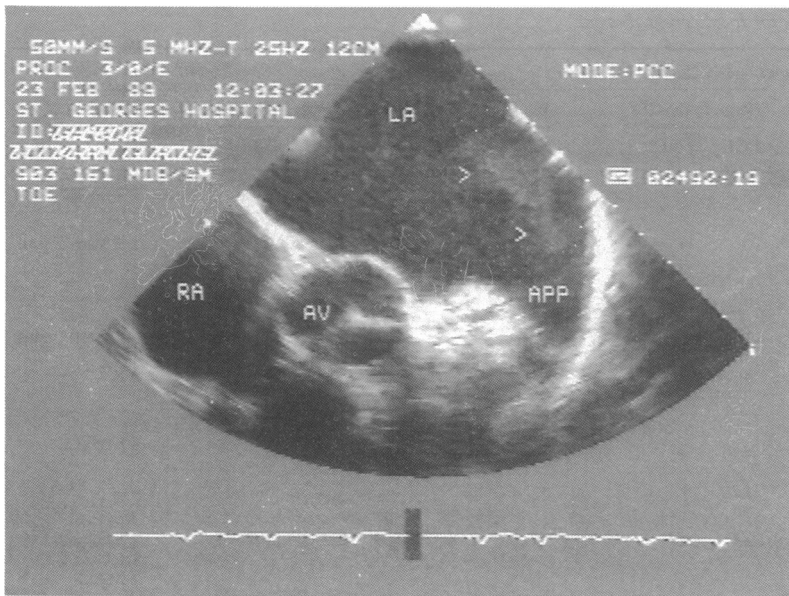
Figure 1 Cross sectional transoesophageal echocardiogram of the left atrium and its appendage. There was a wisp-like cloud of spontaneous contrast echoes (which were seen more clearly during real time echocardiography) in the appendage (arrow heads). LA, left atrium; APP, appendage; AV, aortic valve; RA, right atrium.
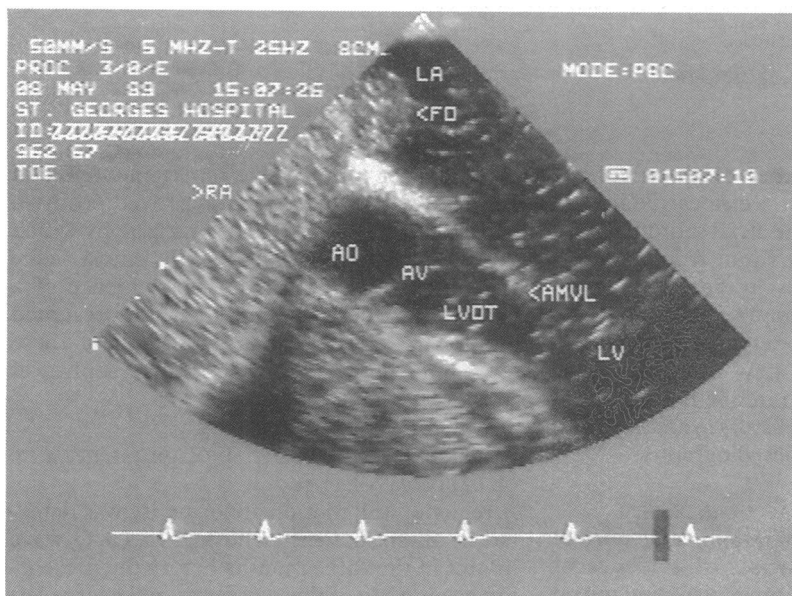

Figure 2 Cross sectional transoesophageal echocardiogram during a contrast study with the imaging plane at the level of the atrial septum. There was a cloud of echoes from microbubbles in the right atrium. Some microbubbles were detected passing across the atrial septum to the left atrium. Some of these had passed through the left ventricle and were seen passing from the left ventricular outflow tract to the aorta. LA, left atrium; RA, right atrium; FO, atrial septum at the level of the foramen ovale; LV, left ventricle; LVOT, left ventricular outflow tract; AMVL, anterior mitral valve leaflet; AV, aortic valve; AO, aorta.

(*d*) *Pulmonary arteriovenous fistula*—As for patent foramen ovale, except that no evidence of right-to-left shunting was detected across the atrial septum. Instead contrast was seen to pass into the left atrium from the direction of the right pulmonary veins and was detected only after a period of three or more cardiac cycles from the appearance of contrast in the right atrium—in all such cases the microbubbles detected were considerably smaller than those seen to pass immediately across the atrial septum in definitions (*b*) and (*c*) above.

The left ventricular wall thickness was not measured in the transoesophageal studies. The short axis view from the stomach transects the ventricle at papillary muscle level. We have noted that the ventricular wall appears thicker at this level by the transoesophageal technique than it does with standard measurements from the precordial studies (measured above the papillary muscles); no satisfactory normal data are yet available and it is thus difficult to confirm the presence of ventricular hypertrophy with the transoesophageal transducer.

INTERPRETATION OF ECHOCARDIOGRAPHY STUDIES

All hard copy print outs and video tapes were analysed by an experienced interpreter (GL) who was blinded to the interpretation of the operator performing the studies. Although this interpreter knew that all patients had had a neurological event, he was unaware of the other clinical details of the patient. Results were compared with the interpretation of the operator. Agreement was complete in all patients except two with a possible pulmonary arteriovenous fistula as defined above; the operator had detected a shunt during the study but this was not confirmed in the subsequent analysis. These patients were recorded as having no shunt.

Results

The mean age of the 131 patients was 57 years (range 20–88 years) and 77 (59%) were men. Forty one patients (31%) were smokers at the time of presentation, 17 (13%) were ex-smokers, and 73 (56%) were non-smokers. The presenting event was a transient ischaemic attack in 49 patients (eight with a history of previous events), a cerebral infarct in 77 (four with a previous history), and in five a retinal arterial occlusion had occurred (one patient with amaurosis fugax and four with persistent visual defects). The "risk factors" of the five patients with retinal artery occlusion were mitral valve prolapse (one patient), known cerebrovascular disease (one patient), hypertension (one patient) and no clinically detectable factor (two patients, one with mitral annular calcification on echocardiography).

Table 1 shows the echocardiographic findings with the precordial and transoesophageal techniques in relation to the clinically determined risk factor(s) for the neurological event.

PATIENTS WITH HYPERTENSION ALONE

Sixteen patients were being treated for hypertension on presentation: eight had left ventricular hypertrophy. No other abnormalities were detected in this group.

PATIENTS WITH ATRIAL FIBRILLATION (OTHER RISK FACTORS EXCLUDED)

Sixteen patients had atrial fibrillation (11 chronic, five paroxysmal). All five patients with paroxysmal atrial fibrillation were in sinus rhythm during the echocardiographic studies. Precordial echocardiography showed a large left atrium in 10, all with chronic fibrillation. Transoesophageal echocardiography showed spontaneous contrast echoes in and around the left atrial appendage in nine of these 10 patients: in five, these echoes were also seen in

Table 1   Echocardiographic findings in relation to risk factors for focal cerebral event

Risk factor(s) for event (n)	Echocardiographic findings									
	LVH		Large LA/SCEs		MVP		PFO/ASD (PAVF)		Other	
	PCE	TOE	PCE	TOE*	PCE	TOE	PCE	TOE	PCE	TOE
Hypertension (16)	8	?	—	—	—	—	—	.—	—	—
Chronic/paroxysmal atrial fibrillation† (16)	—	—	10	9	—	—	1	2 (1)	1 AS aneurysm	
Cerebrovascular disease (12)	—	—	—	—	1	1	—	2	—	—
Rheumatic mitral valve disease/ MVR (12)	—	—	11	11	—	—	—	2 (1)	—	1 vegns
MI/DCM/HCM (8)	1	?	3	3	—	—	—	1	6 RWMA	
Mitral valve prolapse (4)	—	—	—	—	4	4	1	1	—	—
Combinations‡ (10)	4	?	3	3	—	—	1	4	2 RWMA	
Other‡ (12)	2	?	1	1	—	—	2	4		1 AV vegns
None (41)	—	—	—	—	5	5	4	11 (1)	1 bicuspid AV 1 RWMA 1 MAC	
Total (131)	15	?	28	27	10	10	9	27 (3)	—	—

PCE, precordial echocardiography; TOE, transoesophageal echocardiography; LVH, left ventricular hypertrophy; LA, left atrium; SCEs, spontaneous contrast echoes; MVP, mitral valve prolapse; PFO, patent foramen ovale; ASD, atrial septal defect; PAVF, pulmonary arteriovenous fistula; AS, atrial septal; MVR, mitral valve replacement; vegns, vegetations; MI, recent myocardial infarction; DCM, dilated cardiomyopathy; HCM, hypertrophic cardiomyopathy; RWMA, left ventricular regional wall motion abnormality; AV, aortic valve; bicsp, bicuspid; MAC, mitral annular calcification.
*Numbers refer to TOE studies in which spontaneous contrast echoes were seen.
†Without evidence of mitral valve disease, hypertension, or recent infarction.
‡See text.
? Wall thickness measurements from transoesophageal studies not made (see text).

the main left atrial chamber. Both precordial and transoesophageal echocardiography showed an aneurysm of the atrial septum in one patient. The right-to-left shunt from an atrial septal defect was shown in one patient by precordial echocardiography but left-to-right shunting was only demonstrated on the transoesophageal colour flow map. A patent foramen ovale was shown in one patient and a pulmonary arteriovenous fistula in another only by transoesophageal echocardiography.

PATIENTS WITH CEREBROVASCULAR DISEASE
The heart was echocardiographically normal in nine of 12 patients who had evidence of cerebrovascular disease. One patient had mitral valve prolapse and two had evidence of right-to-left shunting across a patent foramen ovale, which was detected only with a transoesophageal contrast study.

PATIENTS WITH RHEUMATIC MITRAL VALVE DISEASE OR PREVIOUS MITRAL VALVE REPLACEMENT
Eleven of the 12 patients in this group were in atrial fibrillation. All eleven had a large left atrium as determined by precordial echocardiography and spontaneous contrast echoes shown by transoesophageal echocardiography. Three patients had echogenic masses in the left atrium consistent with thrombus. In one the thrombus was seen in the atrial appendage only, in another it extended from the appendage across the roof of the atrium, and in the third it was visualised on the posterior wall of the atrium adjacent to the right upper pulmonary vein. A clinical diagnosis of mitral prosthetic endocarditis was made in one patient who had two transient ischaemic episodes three

months after mitral valve replacement. Blood cultures (which subsequently proved positive) had been taken before echocardiography which showed echoes consistent with mitral valve vegetations. Two patients had evidence of a patent foramen ovale and one had a presumed pulmonary arteriovenous fistula; all of these were only apparent on the transoesophageal studies.

PATIENTS WITH A RECENT MYOCARDIAL INFARCT OR CARDIOMYOPATHY
A regional wall motion abnormality was detected in all six patients who had had a Q wave infarct in the three months before the presenting event. One of these patients had a large left atrium, as did one patient with dilated cardiomyopathy and the patient with hypertrophic cardiomyopathy. In these three patients spontaneous contrast echoes were shown in the left atrium on the transoesophageal study. In addition, spontaneous contrast echoes were visualised in the left ventricle of the patient with dilated cardiomyopathy and the patient with the previous infarct. A patent foramen ovale was shown by transoesophageal echocardiography in one patient with a previous infarct.

PATIENTS WITH MITRAL VALVE PROLAPSE
Four patients had clinical diagnoses of mitral valve prolapse; all were confirmed on echocardiography. One of these patients also had a patent foramen ovale.

PATIENTS WITH COMBINATIONS OF THE ABOVE RISK FACTORS
Twelve patients had combinations of risk factors—for example, hypertension and atrial

Table 2   Summary of echocardiographic findings in patients with and without clinically detected cardiac abnormalities

| | | Echocardiographic findings* | | | |
	No	MVP	Large LA or SCEs	R-to-L shunt	Other
Patients with no clinical cardiac abnormality	78	6	0	17	1 RWMA 1 MAC
Patients with clinically detected cardiac abnormality	53	4	25	10	1 AS aneurysm 8 RWMA 2 vegetations 14 confirmed valve lesions

*Excluding left ventricular hypertrophy.
R-to-L, right-to-left. See footnote to table 1 for other abbreviations.

fibrillation. Left ventricular hypertrophy was shown in two patients with hypertension. A large left atrium was detected in three patients in chronic fibrillation, all of whom had spontaneous contrast echoes. A regional wall motion abnormality was detected in two patients with recent Q wave infarction; both were in atrial fibrillation. The foramen ovale was patent in three patients and in one an atrial septal defect was detected.

PATIENTS WITH OTHER RISK FACTORS

Other risk factors included migraine, polycythaemia, sarcoid, connective tissue disorders, atrioventricular conduction disease, aortic valve endocarditis, and in three patients there was evidence of vascular disease from a history of angina or intermittent claudication (none of them had carotid bruits). Two patients had mild concentric left ventricular hypertrophy, although there was no history of hypertension. A large left atrium with spontaneous contrast echoes was found in the patient with sinoatrial/ atrioventricular conduction disease (who had a right ventricular pacemaker). Transoesophageal echocardiography showed echoes consistent with vegetations in the patient with aortic valve endocarditis. Four of these patients had a patent foramen ovale.

PATIENTS WITHOUT RISK FACTORS

There were 41 patients with none of the risk factors described. Five were shown to have mitral valve prolapse and a bicuspid aortic valve was confirmed in another. A regional wall motion abnormality was detected in an elderly patient with no evidence of previous infarction. Eleven of these patients had a patent foramen ovale, the right-to-left shunts being detected only by the precordial studies in four. One patient had a pulmonary arteriovenous fistula and one had mitral annular calcification.

PATIENTS WITH AND WITHOUT CLINICALLY DETECTED CARDIAC ABNORMALITIES

The patients were separated into those with clinically detected cardiac abnormalities and those with no evidence of cardiac abnormality. For this purpose patients with hypertension alone as a risk factor were classified as having no cardiac abnormality irrespective of the electrocardiographic criteria of left ventricular hypertrophy (table 2).

Discussion

The discrepancy in the number of patients who have a focal ischaemic neurological event as a consequence of a cerebral embolus between clinical and necropsy studies suggests, in part, that cardiac sources of emboli often go undetected.[1-5] In a study of patients with a fatal event, it was estimated that only about 60% of patients who had a cardioembolic event would have been identified by clinical criteria alone (history of cardiac disease, atrial fibrillation, or auscultatory abnormalities), and that there would have been a 20% false positive rate and a 19% false negative rate.[20] M mode and cross sectional echocardiography have clearly improved the detection of cardiac abnormalities that are associated with embolic events[6-8] but several groups have pointed out that the yield is low in those without abnormal clinical signs.[21-23]

There is no doubt that the transoesophageal echocardiographic technique enhances our ability to detect cardiac abnormalities in patients with focal neurological events. Another advantage is that good quality images can be obtained in virtually all patients. However, many of the criticisms of precordial echocardiographic studies in these patients also apply to the transoesophageal technique. In particular, many of the abnormalities that are detected have been identified or suspected from previous clinical evaluation. In addition, the finding of a cardiac abnormality does not prove a pathogenic mechanism for the neurological event.

Cerebrovascular events are most common in the elderly, and in this group both cerebral and cardiac pathologies coexist. Clinical decisions, especially those about treatment, must take into account the probability of events based on a knowledge of the pathologies most commonly associated with them. Age is the most important risk factor for stroke, with hypertension being the second most important.[24] These statements take account of the relative risk attached to a risk factor and the attributable risk based on the frequency of the risk factor in the general population.[25] The highest relative risk for stroke is associated with rheumatic mitral valve disease and atrial fibrillation, but this combination is uncommon in the general population; overall less than 2% of all strokes are related to this pathology.[26] Moreover, atrial

fibrillation itself carries only a small relative risk.[27] It is interesting that about 10% of patients with primary intracerebral haemorrhage or lacunar infarction (neither of which are due to thromboembolism) are in atrial fibrillation on presentation.[28] Moreover, there seems to be relatively little if any excess risk of recurrent stroke in patients in atrial fibrillation at the time of the presenting event compared with those in sinus rhythm.[28]

The patients investigated in this study may not reflect the general population of patients presenting with focal ischaemic neurological events, because the referral for echocardiography may have been biased by the detection of cardiac abnormalities. However, most of the patients were investigated as part of a continuing study to which all patients with a stroke or a transient ischaemic attack are referred: thus the patients cover a wide range of those presenting with such events. Although many patients with retinal arterial events have these as a consequence of cerebrovascular disease we included such patients in this study because the sources of embolism for these events have not been fully categorised. Patients in this study did not routinely undergo carotid duplex studies, mainly because of lack of the appropriate facilities and expertise at our centre during the period of this study. Thus the four patients who were studied were referred to another centre because they had carotid bruits. The role of such studies in patients with neurological events who either have no obvious risk factor for the event or who have a clinically obvious factor (such as mitral stenosis) remains to be determined.

Although the patient selection must be considered our data accord with the fact that patients with hypertension or overt cerebrovascular disease as the sole identifiable risk factor and patients with no clinically apparent cardiac disease have a relatively low yield of cardiac abnormalities on full echocardiographic evaluation. However, two causes in particular that are not detected clinically are mitral valve prolapse and a patent foramen ovale, particularly the latter. The potential for right-to-left shunting in these patients, especially if young and without other obvious risk factors, is becoming increasingly recognised.[29–31] In Lechat et al's study there was an association between mitral valve prolapse and the presence of a patent foramen ovale, and after the presence of a patent foramen ovale had been controlled for, mitral valve prolapse no longer correlated with stroke.[31] Although mitral valve prolapse has been associated with embolic events, clinically silent mitral valve prolapse seems to be benign, and it is therefore inappropriate to attribute a cerebral event to this finding.[32] In addition, the difficulties in diagnosing mitral valve prolapse from single plane transoesophageal studies have not yet been fully addressed. Moreover, for both mitral prolapse and a patent foramen ovale, there is no convincing evidence that any specific treatment can reduce the likelihood of recurrent events. Thus until more data are available the detection of these abnormalities may not be clinically

useful. This also applies to mitral annular calcification, detected in one patient in this study. Although believed by some to be associated with thromboembolic events,[33] there is no convincing evidence of this.

For the group of patients with a clinically detected cardiac lesion it could be argued that echocardiography, whether precordial or transoesophageal, simply confirms the diagnosis. For patients in atrial fibrillation, we expect to find a large left atrium and evidence of flow abnormalities or thrombus. Thus we should not be surprised that the precordial echocardiogram shows an enlarged left atrium. The fact that no flow abnormalities are shown in the left atrium by the precordial technique relates to problems with resolution. Transoesophageal echocardiography is useful in these patients because (a) the transducer can be placed directly behind the left atrium and (b) a 5 MHz transducer is used. Evidence of flow abnormalities can be detected either as echoes consistent with thrombus[14–17] or as spontaneous contrast echoes.[34–36] Whether this information is useful or not remains debatable. Given, however, the epidemiological data that demonstrate an overall small relative risk of atrial fibrillation in stroke, it may be that the detection of these spontaneous contrast echoes provides strong evidence relating the arrhythmia to the cerebral event. Patients with these echoes, showed convincingly with transoesophageal echocardiography may benefit from anticoagulation.[37 38] This needs to be studied further.

In conclusion, transoesophageal echocardiography detects more cardiac abnormalities in patients with focal ischaemic cerebral events than precordial echocardiography. However, many of the abnormalities detected will have been predicted from previous clinical assessment of the patient. The yield in patients with no clinically detectable cardiac lesion remains relatively low. Abnormalities that are not suspected include mitral valve prolapse, which is probably not related to the cerebral event in such cases, and the possibility of right-to-left shunting. Although there is growing evidence that such shunting may have a pathogenic role in such patients, at present there are no data demonstrating that recurrences can be reduced or avoided with specific treatments. Until such data are available, echocardiography, whether precordial or transoesophageal, has a limited role in patients with focal ischaemic cerebral events.

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