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EDITORIAL

Preventing Strokes Associated With Carotid Endarterectomy: Detection of Embolisation by Transcranial Doppler Monitoring

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Intraoperative embolisation has been estimated as the cause of perioperative stroke in up to 80% of carotid endarterectomies (CEA), while reduced cerebral blood flow is responsible for less than 20%.[1] Monitoring or quality control methods which are unable to detect embolisation are unlikely to reduce perioperative mortality and morbidity associated with CEA. Because the majority of monitoring methods are used primarily to detect haemodynamic abnormalities, this may be one reason why no study has convincingly established the clinical advantage for perioperative monitoring and why a significant number of surgeons performing CEA do not employ this strategy.[2] For any monitoring method to have an impact on perioperative morbidity/mortality it must detect the majority of abnormalities while there is still time to correct the defect and prevent permanent damage. There is accumulating evidence that transcranial Doppler (TCD) monitoring can not only detect embolisation, but also that it can identify clinically significant patterns of embolisation early enough to permit therapeutic intervention to prevent stroke.[3] Even surgeons with low perioperative stroke rates have a duty to ensure that small numbers of preventable strokes are eliminated. The evidence emerging from studies of the perioperative use of TCD may be making the argument that there is no clinical advantage in monitoring more difficult to sustain.

The introduction of transcranial Doppler (TCD) monitoring of the middle cerebral artery (MCA) during CEA enabled emboli to be detected directly for the first time, whilst simultaneously providing haemodynamic data on the adequacy of cerebral blood supply.[4] Emboli were detected as short duration, high intensity signals (HITS) against the background blood velocity spectral display. However, initial studies failed to demonstrate a convincing link between TCD-detected emboli, which were frequent, and the development of strokes, which were uncommon, and this contradicted the current thinking, which could be summarised as "one embolus causes one stroke".[5] These findings raised doubts as to whether the HITS were indeed emboli or simply artefacts, and this stimulated a large amount of both experimental and clinical work to prove that the initial findings were correct.[5] Indeed, greater experience of the technique and advances in TCD technology have enabled us to detect emboli in over 95% of CEAs.[6]

The results of studies investigating the association of TCD-detected emboli and outcomes such as strokes, cognitive deficits and computed tomography (CT)/magnetic resonance imaging (MRI) infarcts have led to a better understanding of the clinical effect of TCD-detected emboli.[3,7] Whether intraoperative embolisation results in strokes is dependent on numerous factors such as the character of the emboli, the number, the size and the rate of embolisation. Patient factors are also important, with emboli more likely to cause deficits in poorly perfused brain with inadequate collateral circulation and areas of borderline ischaemia and/or previous infarction.[5] The most important factor is the character of the emboli, and the most clinically useful differentiation is into either air or particulate.

Air reflects 99% of incident ultrasound waves, and therefore produces a very loud TCD embolic signal out of all proportion to both the size of the air bubble and its clinical importance. TCD is very sensitive to

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air and can detect bubbles in solution less than 1 μl in volume. It is these small volumes of air, which persist despite adequate flushing and venting procedures, which give rise to the majority of embolic signals detected by TCD during CEA, and in the volumes normally associated with CEA these do not cause strokes.[3,5]

Particulate emboli, including plaque fragments and platelet aggregates, produce much weaker TCD embolic signals than air, and if the TCD is not set up specifically to detect them they are easily missed. However, these are clinically much more important, and it is these emboli which are responsible for the majority of intraoperative strokes. Although the number and rate of embolisation can be determined using standard, commercially available TCD machines, the absolute differentiation of air and particulate emboli using TCD signal criteria alone and the sizing of emboli is still the subject of much scientific study. Evidence has been produced that air and particulate emboli occurring during the same stage of the operation can be distinguished using a different form of spectral analysis known as Wigner, and this has been useful as a research tool in detecting particulate emboli during various stages of the operation.[8] At present, these results are only available after postoperative signal processing, and this limits its clinical application. However, future development aims to produce automated systems of emboli detection and differentiation available for routine intraoperative use.

Fortunately, even in the absence of these sophisticated systems, a clinically applicable differentiation of air and particulate emboli is still possible using the standard TCD machine. One important lesson derived from experience with TCD monitoring is that strokes are seldom caused by one big embolus. The majority of strokes result from a period of multiple embolisation and, most importantly, the development of clinically detectable strokes are preceded by a period of asymptomatic embolisation which can be detected by TCD monitoring. Intervention during this period to prevent further embolisation can prevent the development of serious neurological deficits.[9] However, there are occasions when a single large embolus can result in an intraoperative stroke, and one of these is immediately after final clamp release and restoration of blood flow. An embolus occurring at this stage happens too quickly for corrective action to be taken and, therefore, prevention is the better strategy. One preventative measure is to perform completion angioscopy, which can detect residual thrombus in the lumen of the endarterectomised artery and permit its removal before final clamp release. Quality control methods applied after clamp release, such as angiography or duplex, do not have the potential to detect or prevent this complication.[9]

Clinically, we have found it useful to divide the operation of CEA into seven distinct stages (based on our practice of routine shunt insertion): dissection, shunt opening, during shunting, restoration of ECA flow, restoration of ICA flow, manipulation, and recovery. Pathological embolisation and haemodynamic complications can occur at any stage during the operation; therefore it is important to have continuous TCD monitoring. Wigner analysis has identified that air and particulate emboli occur during each stage, but with the exception of dissection and recovery the emboli are predominantly air and not commonly associated with neurological deficits.[8] However, emboli detected in the dissection and recovery phases are predominantly particulate, and it is embolisation during these stages which is associated with the majority of strokes.[3]

Dissection includes time from skin preparation to clamping of the internal carotid artery. During this stage the arterial system has not been entered, and therefore any emboli detected can be considered to be particulate. With friable, ulcerated plaques or those with associated thrombus, even gentle dissection around the carotid bifurcation can precipitate showers of emboli. When this occurs, further embolisation can be prevented by early clamping of the ICA and insertion of a shunt before completing the dissection. Strokes occurring during the dissection phase were well known before the introduction of TCD, but recent studies have described an association between particulate embolisation during dissection and postoperative cognitive deficits and MRI lacunar infarcts.[3,7]

The recovery phase is the time after final restoration of flow and after all manipulations of the artery, such as the insertion of extra stitches to bleeding points, have finished. In 1990 Spencer described two strokes associated with persistent embolisation detected in this phase in 91 patients monitored with TCD during CEA. One of these patients underwent emergency reoperation and a thrombosed CCA was found.[5] In our study of 100 consecutive patients, three patients were identified with persistent embolisation in the first hours following final clamp release, which were associated with both early carotid artery thrombosis and the development of neurological deficits. In two of these cases persistent embolisation was associated with a gradual fall in MCA velocity as the ICA occluded with thrombus, and in the other case MCA velocity remained fairly constant as the ICA only partially occluded. In each case re-exploration and removal of

the intraluminal thrombus, combined with systemic heparinisation, was successful in preventing further emboli, and in the final case, based on our experience of the previous two, re-operation was performed soon enough to reverse a developing stroke. Interestingly, no technical defect was found or corrected in any of these cases, the thrombus having formed on the endarterectomy surface itself.[3]

Subsequent prospective studies of postoperative embolisation have confirmed these initial findings. Roberts *et al.* monitored 80 patients and identified postoperative emboli in 58. However, only eight of these patients experienced greater than 50 emboli in the first hour, and five of these then suffered strokes.[11,12] Evidence that reoperation may represent an overtreatment in some patients, and occasional practical problems in returning a patient to theatre soon enough to be effective, prompted the investigation of an alternative approach to treat postoperative embolisation and prevent stroke. In a recent study, the institution of an incremental infusion of Dextran-40 in patients demonstrating persistent postoperative embolisation resulted in cessation of embolisation in the eight patients in whom it was used, and no patient in this prospective study of 100 patients suffered a postoperative thromboembolic stroke.[13] However, further experience suggests that in the presence of an uncorrected technical error, Dextran may be unable to arrest thrombus formation, and reoperation is then indicated based on the TCD evidence of continued embolisation, possibly combined with a falling middle cerebral artery velocity.[3] This only serves to emphasise the importance of TCD monitoring in the postoperative phase and the potential inadequacy of blind Dextran administration.

In conclusion, a low perioperative stroke rate is important for all surgeons performing carotid endarterectomy, especially if the operation is to be performed for asymptomatic disease. Equally important is the need to minimise the number of preventable strokes, and these can only be prevented if the underlying mechanisms are understood. Experience with TCD monitoring has modified our understanding of how many perioperative strokes occur and enabled the development of intervention strategies. The key to preventing the majority of perioperative strokes is the detection of embolisation, and TCD is the only monitoring method which supplies this information.

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