

Transcranial Doppler: An Early Predictor of Ischemic Stroke After Cardiac Arrest?

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A 69-yr-old woman was admitted to the intensive care unit after cardiac surgery. Immediately after the discontinuation of cardiopulmonary bypass, she had a circulatory arrest. A 13-min open-chest cardiac massage was followed by 70 min of cardiopulmonary bypass. Right hemiplegia and right extensor plantar reflex were noted after the patient awakened. She had been included in a prospective study protocol measuring, before and after

surgery, cerebral blood flow with transcranial Doppler (TCD). The data were retrospectively analyzed, and it was established that the TCD had recorded cerebral perfusion defects. This is the first case of acute ischemic stroke after cardiac arrest with retrospective documentation of asymmetrical cerebral blood flow by a systematic postoperative TCD recording.

(Anesth Analg 2003;97:1262–5)

The diagnosis of ischemic stroke in the acute phase usually relies on a computed tomography (CT) scan, which is, however, often normal at the start of symptoms or which can show lacunae lesions during the first 24–48 h. Early diagnosis can be obtained with angio-magnetic resonance imaging (MRI) (1), diffusion-weighted imaging (2), or both. Nevertheless, the clinical presentation remains the determining element, because symptoms are the first to draw attention. However, in the intensive care unit (ICU), symptoms of stroke can be masked because of both sedation and muscle relaxation or because of other serious alterations of mental state or consciousness.

Here, we report the case of a patient who, after cardiac surgery, presented a right hemiplegia after awakening. Interestingly, because the patient had been included in a study protocol measuring pre- and postoperative cerebral blood flow with a transcranial Doppler (TCD) (3), we had the possibility of retrospectively analyzing recordings and noting that the transcranial examination showed cerebral perfusion defects.

Case Report

A 69-yr-old woman was admitted to the ICU after a quadruple coronary artery bypass graft surgery performed for an increasingly symptomatic angina pectoris at rest. The patient had a history of systemic hypertension, hypercholesterolemia, and a cholecystectomy. The preoperative cardiac function was moderately diminished (ejection fraction at 44%). Neck vessel echo-Doppler examination demonstrated discrete atheromatoses of bilateral internal carotid arteries.

Immediately after discontinuation of the cardiopulmonary bypass (CPB) and 10 min after the administration of protamine, the patient rapidly developed pulmonary artery hypertension (systolic arterial blood pressure >8 kPa measured with a pulmonary artery catheter), with subsequent circulatory arrest. A 13-min open-chest cardiac massage (mean arterial blood pressure (MAP) between 30 and 40 mm Hg) was followed by 70 min of CPB, which was finally weaned under epinephrine ($0.11 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-3}$). The total CPB time was 285 min, and aortic cross-clamp time was 210 min. The thoracic cavity was initially kept open because of hemodynamic instability and cardiac dilation and was closed after 48 h. Hemodynamic stability was progressively obtained, and epinephrine was weaned within 6 h after CPB. A Ramsay score of 3 was targeted until tracheal extubation (7 h after CPB) with intermittent bolus injections of midazolam (total of 5 mg). The patient's renal function was normal.

Awakening with marked agitation was noted after 24 h, with slight motor activity and reflex asymmetry. Right hemiplegia and an extensor plantar reflex were confirmed the next day. No intracranial hemorrhages or other pathologic lesions were noted on a noninjected cerebral CT scan (Fig. 1A). The patient was tracheally extubated on the third day, after which aphasia, swallowing difficulties, and confusion were noted. Clinical improvement enabled the patient to be discharged from the ICU on the sixth day. A right

Accepted for publication June 9, 2003.

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DOI: 10.1213/01.ANE.0000083373.19574.64

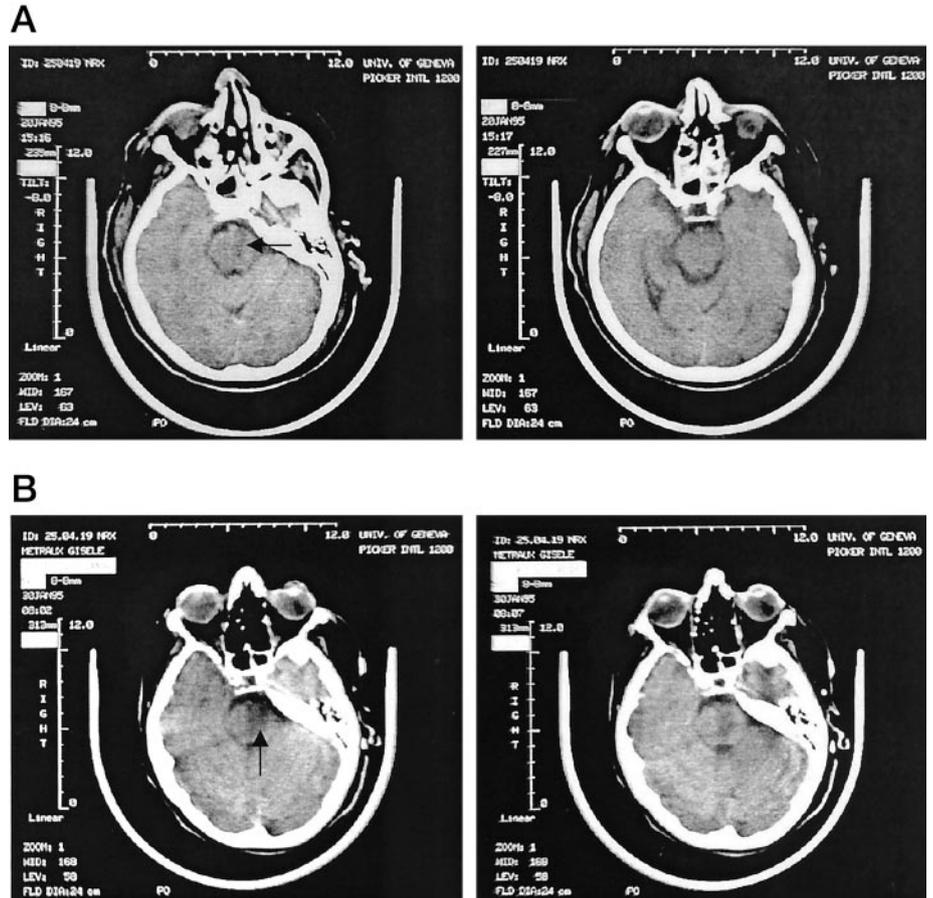


Figure 1. A, No intracranial hemorrhage or other pathologic lesion was noted on a first (Day 2) noninjected cerebral computed tomography scan. Retrospectively, it can show a mild left hypodensity of the midbrain (protuberance; arrow). B, A cerebral computed tomography scan done 12 days after surgery revealed a voluminous left hypodensity of the midbrain (protuberance; arrow) associated with signs of infarction.

amotor and sensory hemisindrome and swallowing difficulties were still present, but with recovery of verbal communication. A cerebral CT scan done 12 days after surgery revealed a voluminous hypodensity of the left midbrain (protuberance) associated with signs of infarction (Fig. 1B).

This case report is of interest because the patient had agreed to take part in a prospective study measuring cerebral blood flow during the pre- and postoperative period after CPB (3). Indeed, she had four TCD examinations: preoperatively, immediately on arrival from the operating room, after the warming period, and again 18 h after operation. The results clearly showed a significant reduction in the flow speed measured in the left hemisphere (middle cerebral artery blood flow mean velocity), the same side on which the lesion developed. This was observed on the first measurement on arrival in the ICU. It then indicated diminished left cerebral perfusion during the early stage after circulatory arrest (Fig. 2). Thus, the diagnosis of ischemic stroke could have been suspected before the clinical picture became apparent on the patient's awakening.

Discussion

TCD is currently increasingly recognized as a useful technique for early detection of cerebral blood flow abnormalities during the acute phase of stroke. The

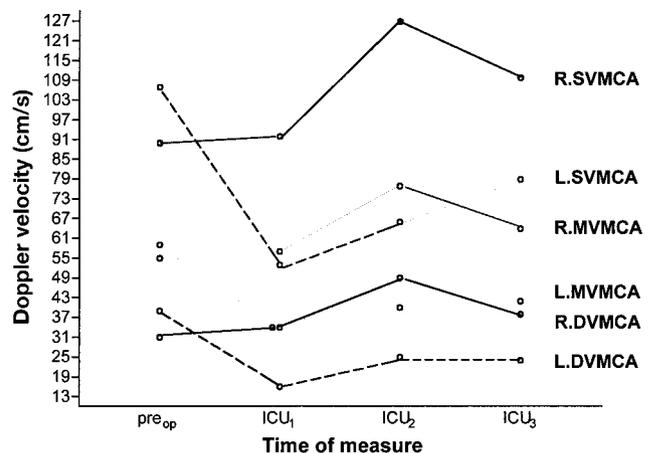


Figure 2. Changes in values of blood flow velocities in the right and left cerebral arteries over time. Solid line—R.SVMCA = systolic velocity of the right middle cerebral artery; R.MVMCA = mean velocity of the right middle cerebral artery; R.DVMCA = diastolic velocity of the right middle cerebral artery. Dashed line—L.SVMCA = systolic velocity of the left middle cerebral artery; L.MVMCA = mean velocity of the left middle cerebral artery; L.DVMCA = diastolic velocity of the left middle cerebral artery. The figure clearly shows a significant decrease in left velocities after surgery with a compensatory increase in right velocities after the warming period in the intensive care unit (ICU₂). pre_{op} = before surgery; ICU₁ = immediately on arrival from the operating room; ICU₃ = 18 h after operation.

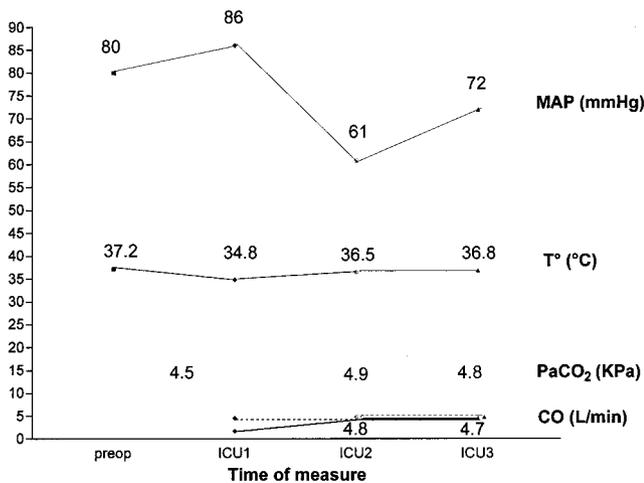


Figure 3. Changes of cardiac output (CO), PaCO₂, temperature (T°), and mean arterial blood pressure (MAP) during the rewarming period. preop = before surgery; ICU₁ = immediately on arrival from the operating room; ICU₂ = after the warming period; ICU₃ = 18 h after operation.

procedure is simple to do at the bedside and is non-invasive, quick, reproducible, and sensitive (3–6). Indeed, asymmetrical cerebral blood flow observed by TCD is a sign that can evoke a thromboembolic lesion in the acute phase of ischemic stroke (7), and, therefore, the examination could provide a basis for early reperfusion/thrombolytic therapy (8), which has been shown to significantly improve outcome after stroke (9).

This case is of particular interest because the patient, whose surgery was complicated by a cardiocirculatory arrest, had been included in a study protocol that systematically measured cerebral blood flow by using TCD. The patient was intubated and sedated, and, therefore, the detection of clinical stroke symptoms was delayed. However, TCD had detected a clear flow asymmetry two hours after the arrest. It seems clear to us that the approach to and management of stroke in this case could have been optimized with the introduction of a prophylactic therapy applied within the first 48 hours. Indeed, even if the clinical examination showed the neurological defect at an early postoperative stage, the TCD abnormalities were observed before the patient awakened from the anesthesia, which could have generated further examinations (MRI, angiography, and so on) to confirm the diagnosis and tailor an anticipated specific reanimation. Although TCD can record cerebral perfusion defects, it cannot confirm the etiology of an ischemic stroke. Therefore, in this setting it is difficult to define an appropriate therapeutic goal. Nevertheless, the primary aim of managing patients with acute brain injury in the ICU is to minimize secondary injury by maintaining optimal cerebral perfusion, oxygenation, and normal glycemia and natremia (10).

The postoperative neurologic description of our patient disputes the hypothesis of a single lesion observed on the CT scan. Nevertheless, a severe perioperative hemodynamic alteration can be the starting point of multiple emboli, even if just a single midbrain lesion is confirmed by the CT scan. In contrast, because the duration of CPB was long, we also cannot exclude that this situation could have been the starting point of multiple gaseous embolisms (a rare phenomenon in non-open-heart surgery). The inconsistent relation between TCD recording data and the left midbrain hypodensity on CT scan can also be attributed to a reverse flow perfusion through the Willis polygon. Finally, a cerebral diaschisis phenomenon cannot be excluded, because local cerebral dysfunction in regions remote from the principal cerebral ischemic lesions in the acute stage of stroke has been demonstrated (11,12).

Evidently, the cerebral CT scan performed 12 days after surgery was not typical of persistent middle cerebral artery flow abnormality, because this perfusion defect is usually responsible for severe hypodensities. However, in the absence of cerebral metabolic assessment or MRI during this period, we cannot infer the effective cerebral blood flow of the middle cerebral artery. Thus, the reduction of the flow recorded on the left side was possibly not severe enough to cause hypodensity in the concerned cerebral region. Moreover, the immediate hemodynamic improvement (cardiac output; see Fig. 3) could have participated in this paradoxical status.

Therefore, in anesthesia and ICUs, given the fact that clinical neurological examinations are often of limited value, TCD could be used systematically for monitoring deeply sedated patients who have undergone major hemodynamic instabilities. This new tool for neurological surveillance of postoperative and ICU patients could be further explored in careful clinical investigations.

The authors are grateful for the translation provided by Samia Brunner.

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