BRIEF COMMUNICATIONS

BRAIN FUNCTION MONITORING DURING BIDIRECTIONAL GLENN PROCEDURES

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Clamping of the superior vena cava (SVC) without decompressing the jugular venous system increases intracranial pressure and decreases cerebral blood flow.¹ Because of this, cardiopulmonary bypass (CPB) is frequently used to facilitate cerebral venous drainage during the bidirectional Glenn anastomosis. Jahangiri and associates² reported that a transcranial pressure of more than 30 mm Hg may minimize the undesirable cerebral effects of SVC clamping when these procedures are performed without CPB. We have used flow, oxygenation, and cerebral function indicators—transcranial Doppler ultrasonography (TCD), near-infrared spectrophotometry, and electroencephalography or middle latency evoked responses (MLRs), respectively—for monitoring these procedures. Continuous TCD provides information regarding the functional status of the brain vasculature. Near-infrared spectrophotometry measures the ratio of light absorption by oxygenated and total hemoglobin, thus providing an estimate of the mean cerebral mixed venous oxygen saturation.³ MLRs represent the electrical activity generated at the midbrain and primary auditory cortex.⁴ The electroencephalogram integrates the cortical activity paced by subcortical thalamic nuclei. Both modalities have been used for detecting brain dysfunction during cardiac procedures.^{4,5} These methods complement hemodynamic information regarding cerebral effects of SVC clamping during bidirectional cavopulmonary anastomosis. We report the use of these cerebral function indicators during these procedures under CPB. A clinical case of cavopulmonary anastomosis without support of CPB is also documented. In this case, brain function indicators revealed deleterious effects of SVC clamping and allowed assessment

of the effects of decompressing the jugular venous system through an intraoperative SVC shunt.

Clinical summary. A 4-year-old (13.6 kg) girl with hypoplastic right ventricle, tricuspid and pulmonary atresia, and previous bilateral (modified) Blalock-Taussig shunts underwent a bidirectional Glenn anastomosis. Anesthetic maintenance was primarily opioid (fentanyl) supplemented by isoflurane (end-tidal concentration: 0.3%). A 2-MHZ TCD system (Medasonics, Fremont, Calif) monitored cerebral blood flow velocities at the right middle cerebral artery via the temporal window. Monaurally evoked MLRs to clicks (duration: 100 μ s; rate: 11.1/s) of alternate polarity (intensity: 80 dB nHL—normal hearing level) were recorded from A₁ and A₂ referred to a mid-frontal electrode with the forehead as a ground. The electrical activity was filtered (10–3000 Hz) and digitized (8 bits; sampling rate 50 Hz) by a Quantum 84 averager (Cadwell, Kennewick, Wash). Input signals greater than 80 μ V were automatically rejected. The systolic and diastolic arterial blood pressure, central venous pressure, end-tidal carbon dioxide, and temperature were all continuously monitored. The estimated transcranial pressure was calculated as systolic blood pressure minus central venous pressure. Before SVC clamping, the peak, mean, and diastolic cerebral blood flow velocities were 81, 52, and 30 cm/s (systolic/diastolic blood pressure: 110/50 mm Hg; central venous pressure: 9 mm Hg, oxygen saturation by pulse oximetry: 81%; end-tidal carbon dioxide: 29 mm Hg; temperature: 37.3°C; estimated transcranial pressure: 101 mm Hg). Transient SVC clamping resulted in an acute elevation in central venous pressure (74 mm Hg), which led to a reduced estimated transcranial pressure (36 mm Hg; systolic/diastolic blood pressure: 110/50 mm Hg). As perfusion pressure decreased, TCD showed absence of diastolic Doppler flow (0 cm/s) and reduced peak and mean cerebral blood flow velocities (40 and 11 cm/s). Within the next minute, the latencies of the cortically generated MLR components Pa, Nb, and Pb, which had been 33, 50, and 68 msec before SVC clamping, increased to 41, 60, and 76 msec and their amplitude decreased by 30% (Fig 1). Because of concern for cerebral protection related to these events, it was decided to use an intraoperative shunt into the SVC to decompress the jugular venous system while the SVC was clamped. With the shunt in place, decompression was deemed inadequate as indicated by a continued high central venous pressure (40 mm Hg), abnormal TCD findings (no diastolic flow), and longer latencies of evoked potentials for

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Fig 1. Middle latency auditory evoked responses (**A**) and transcranial Doppler ultrasonography (**B**) during a case of bidirectional Glenn anastomosis without cardiopulmonary bypass. **Panel A:** (1) The components *Pa*, *Nb*, and *Pb* of the middle latency response were identified before clamping of the SVC; (2) the middle latency responses increased as cerebral perfusion decreased during transient SVC clamping, but the wave *V* of the brain stem response remained unaltered; (3) latencies returned to preclamping levels when the intraoperative shunt was functioning; (4) latencies remained unaltered at the time of the release of the SVC clamp. **Panel B:** (1) transcranial Doppler waveforms before clamping of the SVC; (2) absence of diastolic Doppler flow and low flow velocities in the right middle cerebral artery as the internal jugular pressure increased with SVC clamping; (3) diastolic Doppler flow returned near preclamping levels during functioning of the intraoperative shunt.

approximately 1 minute (systolic/diastolic blood pressure: 112/52 mm Hg; estimated transcranial pressure: 72 mm Hg). After the shunt had been repositioned, central venous pressure decreased to 19 mm Hg (estimated transcranial pressure: 100 mm Hg) and the diastolic cerebral blood flow velocity was re-established to 60% (18 cm/s) of preclamping values with peak and mean cerebral blood flow velocities of 66 and 36 cm/s, respectively (systolic/diastolic blood pres-

sure: 120/50 mm Hg). The return of the latencies and amplitudes of the MLRs to preclamping values (Fig 1) confirmed this hemodynamic improvement (clamping time: 20 minutes). No neurologic deficits were identified. Recently, we have used hypothermic CPB and brain function monitoring for these procedures (Table I). In these cases, the average central venous pressure during SVC clamping was -7 mm Hg (range: -22 to 9 mm Hg). The TCD and electroen-

Table I. Results of patients undergoing bidirectional Glenn anastomosis with CPB

Case	Age (mo)	CBFV (%)	S/D BP (mm Hg)	CVP (mm Hg)	rCVOS(%)		EEG	Temp (C°)	Duration (min)	
					Before clamp	Clamp on			Clamping	CPB
1	9	126	50/37	-5	68	66	NC	29.2	60	140
2	7	113	42/26	-13	70	68	NC	31.7	34	70
3	9	92	69/52	-15	Nr	Nr	NC	29.5	35	165
4	7	106	64/45	-22	70	71	NC	25.3	10	140
5	8	100	59/49	-5	76	74	NC	31.7	22	140
6	7	117	57/42	9	73	66	NC	31.1	15	118
7	30	90	88/70	5	64	62	NC	33.8	48	87

CPB, Cardiopulmonary bypass; CBFV, cerebral blood flow velocity expressed in percentage relative to the preclamping measurement; CVP, central venous pressure; EEG, electroencephalogram recorded during clamping of the superior vena cava (SVC); NC, no electrocortical changes; Nr, not recorded; rCVOS, noninvasive regional cerebral mixed venous oxygen saturation by near-infrared spectrophotometry (INVOS 3100; Somanetics, Troy, Mich); S/D BP, systolic and diastolic blood pressure during clamping of SVC; temp, temperature (rectal).

cephalogram did not show alterations during clamping, and cerebral mixed venous oxygen saturation decreased by only 2% from preclamping values, indicating adequate cerebral perfusion.

Discussion. The exposure of infants or children to the effects of hemodilution, microemboli, or complement activation⁶ may be minimized when bidirectional Glenn procedures are performed without CPB.² However, intracranial blood volume increases as a consequence of SVC clamping, thereby increasing intracranial pressure and decreasing cerebral perfusion.¹ Thus the strategy of cerebral monitoring can be critical when CPB is not used. Although an estimated transcranial pressure of more than 30 mm Hg has been suggested as sufficient to ensure cerebral perfusion,² this may not always be true. In our reported case, an estimated transcranial pressure of 36 mm Hg resulted in absence of diastolic Doppler flow and longer MLR latencies. This indicates reduced flow leading to electrocortical dysfunction. The use of cerebral indicators clearly complements hemodynamic monitoring. Brain function monitoring may be critical to ensure adequate function of the intraoperative shunt or to confirm that adequate cerebral venous drainage is achieved during venous cannulation in cases of bilateral SVC. The stability of cerebral function indicators in the cases performed

with CPB suggests that this method tempers the undesirable cerebral effects of SVC clamping.

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