**Supplementary Table 6**

**Studies utilizing miscellaneous methods to alter cardiac output**

*AVM, arteriovenous malformation; CBF, cerebral blood flow; CCA, common carotid artery; CI, cardiac index; CO, cardiac output; ETCO2, end-tidal carbon dioxide; ICA, internal carotid artery; MAP, mean arterial pressure; MRI, magnetic resonance imaging; PAC, pulmonary artery catheter; PWA, pulse wave analysis; SV, stroke volume; TCD, transcranial Doppler; TPR, total peripheral resistance; Vmca, middle cerebral artery flow velocity; Xe, xenon*

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| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| **Author (Year) [reference #]** | **N** | **Subjects** | **Method of CBF measurement** | **Method of CO measurement** | **Intervention** | **Change in CO/CI** | **Change in MAP** | **Change in pCO2 (measure)** | **Change in CBF** |
| Smith (1998) [47] | 24 | 15 septic shock and 9 control (hypovolemic or cardiogenic shock) | TCD of CCA flow | PAC  | Nil | CI greater in septic shock (5.0±1.5L/min/m2) than control (3.1±0.7 L/min/m2) (p<0.01) | “No correlation” but data not shown | Data not shown, (no correlation) | Indexed carotid flow was greater, but not to a statistically significant degree: non-septic 204±72 vs septic 257±106 ml/min/m2 |
| Saha (1993) [29] | 45 | 45 adult perioperative cardiothoracic surgery patients  | TCD of ICA flow velocity & Vmca. Ocular PWA (ocular blood flow) | PAC  | Nil | CI varied between 2-4 L/min/m2  | No data | No data | Ocular blood flow was linearly correlated with CI, as was systolic Vmca, but not ICA flow velocity, |
| Diamant (2002) [32] | 7 | 7 healthy adults | TCD Vmca | PWA | Subjects were monitored for 24h  | No data | No data | No data | No dataVmca was associated with SV and CO (P < 0.001), to a lesser extent with mean arterial pressure (MAP; P < 0.005), not with heart rate or TPR.  |
| Erkelens (2017) [64] | 30 | 15 with idiopathic dilated cardiomyopathy and 15 controls  | Pseudo-continuous arterial spin labelled MRI | PWA | nil | CO was not different between cases (6.4 (5.7-7.3) L/min) and controls (6.0 (5.7-7.5) | MAP was different, but not statistically significantly different between cases 72.7 (IQR=19.9) and controls 80.8 (IQR=28.2) | ETCO2 was different, but not statistically significantly different between cases 5.1 kPa (0.9) and controls 5.5 (0.4) (difference of 3mmHg) | Global and regional CBF did not differ between cases (44.3 mL/100g/min) andcontrols (42.1 mL/100g/min) |
| Henriksen (2014) [65] | 31 | 31 healthy adults | Phase contrast MRI  | Cardiac MRI | nil | CI averaged 2.6 (1.8-4.4) L/min/m2 | MAP averaged 96.9mmHg (77-113) | No data | CO and CI do not corelate with CBF. The fraction of CO that goes to the brain is inversely related to CO. |
| Jefferson (2017) [66] | 314 | 314 adults  | Pseudo-continuous arterial spin labeling MRI | Echocardiography | MRI scan breathing room air and then a mix of 5%CO2 95% room air | CI = 2.6±0.6 | No data for MAPSBP = 143±18 | The change in CO2 with different FiCO2 has 7±3mmHg  | CI associatedwith CBF (p=0.001) in the temporal lobes only. |
| Hashimoto (2002) [68] | 20 | 20 patients undergoing removal of a cerebral AVM | 133Xe washout | PAC | Pre and post-operative | CI remained stable (3.8±1.1 to 3.9±0.9 L/min/m2) | MAP remained stable (83±8 to 81±15 mmHg) | paCO2 remained stable (27.0±3.1 to 27.9±3.2 mmHg) | Global CBF increased (25±8 to 31±13 ml/100g/min) as did CBF ipsilateral (25±8 to 31±13) and contralateral (24±7 to 30±13 to the AVM |