**Supplementary Table 1**

**Studies utilizing vasoactive medications to alter cardiac output**

*CBF, cerebral blood flow; CCA, common carotid artery; CI, cardiac index; CO, cardiac output; CPP, cerebral perfusion pressure; CVP, central venous pressure; CVR, cerebrovascular resistance; ECA, external carotid artery; ETCO2, end-tidal carbon dioxide; ICA, internal carotid artery; MAP, mean arterial pressure; PAC, pulmonary artery catheter; PWA, pulse wave analysis; SAH, subarachnoid hemorrhage; TBI, traumatic brain injury; TCD, transcranial Doppler; 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** |
| Sato (2000) [49] | 12 | 12 patients with SAH post clipping | thermal diffusion flowmetry | PAC | Olprinone bolus of 10mcg/kg over 5 min then infusion at 0.2 mcg/kg/min for 25 min | Mean CI rose from 3.7±0.8 to 4.3±1.1 L/min/m2 (p<0.01)  | MAP decreased from 81±9.6 to 77±11mmHg (p<0.05) | No data | Cortical CBF increased from 43±18 to 49±21 ml/100 g/min (p<0.01) |
| Bouma (1990) [53] | 35 | 35 patients with severe TBI | Inhaled or intravenous 133Xe | PAC  | Infusions of phenylephrine and trimethaphan camsylate. Patients were grouped by intact or defective autoregulation, with intact defined as dCPP/CVR being >0 and ≤2 | CO increased with phenylephrine, but only significantly in the defective autoregulation group (+15±21%) and not in the intact group (+7±31%).CO did not change significantly with trimethaphan camsylate in either the defective autoregulation group (+22±6%) nor the intact group (-10±13%). | Although not statistically significant, substantial changes in MAP occurred with phenylephrine infusion (intact 96±12 to 127±14; defective 92±10 to 123±8 mmHg) and trimethaphan camsylate (intact 111±10 to 86±7; defective 108±13 to 77±9) | No data | CBF did not change significantly from baseline in those with intact autoregulation with either intervention: CBF change (%) -1±12, and -2±8 with phenylephrine, and trimethaphan camsylate respectively.With defective autoregulation, CBF had substantial (but not statistically significant changes) with both interventions. CBF change (%) +53±20, and -31±1 with phenylephrine, and trimethaphan camsylate respectively. |
| Davis (1980) [54] | 70 | 40 cats in series 1, 30 cats in series 2 | Intraarterial 133Xe  | PAC  | In series 1, controlled haemorrhage was performed, then administered propranolol, isoproterenol, and isoproterenolcombined with alpha blockade and colloid infusion. In series 2, MAP was elevated ina stepwise fashion with angiotensin, with some given either phenoxybenzamine or propranolol.  | Propranolol, 1 mg/kg, was associated with a 23% fall in CO. After propranolol, the cardiac autoregulatory response was abolished and no change in CO occurred with changes in pCO2.Isoprenaline infusion was associated with an increased in CO of 38%Phenoxybenzamine was associated with a 7% decrease in CO. Subsequent colloid infusion increased CO by at least 7% (no quantitivae data). Subsequent isoprenaline increased CO by 72%, | No comment of the effect of propranolol, 1 mg/kg, on MAP (no quantitative data). CVP did not alter with propranolol (no quantitative data). Isoprenaline infusion caused the MAP to decrease slightly (no quantitiative data).Phenoxybenzamine was associated with a 24% decrease in MAP. Subsequent colloid infusion increased MAP and CVP (no quantitivae data). Subsequent isoprenaline increased MAP “slightly” (no quantitiave data).  | CBF, MAP and CO remained responsive to CO2 changes (no quantitative data).Propranolol, 1 mg/kg, was associated with a decrease in pCO2 of 2mmHg. No comment of the effect of isoprenaline on pCO2. CVP did not alter with isoprenaline (no quantitative data).No comment on the effect of Phenoxybenzamine on pCO2 | Propranolol, 1 mg/kg, was associated with a 30% fall in CBF. When controlled for the change in PCO2 there was still a decrease of 16%. Isoprenaline infusion was not associated with a change in CBFPhenoxybenzamine was associated with a 22% decrease in CBF. Subsequent colloid infusion increased CBF, but less than 22% (no quantitivae data). Subsequent isoprenaline did not change CBF.  |
| Ha (2016) [33] | 8 | 8 patients undergoing FESS for chronic rhinosinusitis | TCD Vmca | PWA | Unilateral surgery with noreadrenaline to maintain MAP 80-100mmHg, contralateral side was control | No date | No data | In all patients, ETCO2 was 32.7±3.3mmHg | MAP correlates with Vmca (r = 0.70, p < 0.0001);CO correlates with Vmca, but with less strength thanMAP (r = 0.57, p < 0.0001); |
| Van der Giessen (1990) [56] | 14 | 14 pigs | Post-mortem measurement of radioactive tracer  | Electromagnetic flow probe in the aorta | Four consecutive10 min i.v. infusions of nimodipine (0.5, 1, 2 and4 Mg kg 1 min 1; n = 7) or equivalent volumes of crystalloid(n = 7). | CO increased with administration of Nimodipine 4mg/kg by 54%(2.4±0.2 to 3.7±0.4 L/min) (mostly due to an increase in HR) | MAP decreased by 9% with Nimodipine 4mg/kg (108±8 to 97±7) (diastolic (-14mmHg) dropping much more that systolic(5mmHg)) | No data | CBF did not change with administration of nimodipine  |
| Kim (2003) [58] | 16 | 16 patients with vasospasm after subarachnoid hemorrhage | Inhaled 131Xe CT | PAC (only in the dobutamine group). | 5 patients had vasopressor therapy with phenylephrine. 5 patients had inotropic therapy with dobutamine | CI increased with dobutamine from 4.1 to 6.0 L/min/m2. Not measured in phenylephrine group | MAP with phenylephrine increased from 102.4 to 132.1 mmHgMAP with dobutamine decreased from 112.5 to 108.0 mm Hg | No data | With phenylephrine (19.2 to 33.7ml/100 g/min) and dobutmine (24.8 to35.4 ml/100 g/min) mean CBF increased |
| Levy (1993) [34] | 23 | 23 patients with vasospasm after SAH  | TCD Vmca | PAC | Dobutamine infusion | CI increased with the addition of dobutamine by 52% (3.30±0.22 to 5.0±0.26 L/min/m2) | MAP increased by 11% with dobutamine | No data | All patients had increase in Vmca on dobutamine |
| Ogoh (2017) [48] | 12 | 12 healthy volunteers | TCD ICA, CCA and ECA velocity and flow | Echocardiography | Dobutamine infusion at low (5μg/kg/min and high (15 μg/kg/min) doses | CO increased from baseline (6.8±1.9) with low dose (8.5±1.8) and high dose (10.0±1.4) dobutamine (p<0.001) | MAP increased from baseline (82±6) with low dose (91±6) and high dose (101±8) dobutamine (p<0.001) | ETCO2 decreased with increasing dobutamine dosing; from baseline (48.3±2.7mmHg) to low dose (44.4±3.8) to high dose (43.6 ± 2.4) (p<0.001) | ICA flow decreased from baseline (317±61 ml/min) with low dose (290±50) and high dose (300±51) dobutamine (p=0.002) |
| Treib (1996) [43] | 10 | 10 patients with acute ischemic stroke of the MCA | TCD Vmca  | Impedance cardiograph | 500ml each of colloid and crystalloid were infused over 1h with a mix of dobutamine and dopamine simultaneously.  | CO increased from 5.8(±1.3) to 8.9(±2.2)L/min (+53%) | MAP increased from 102.3 to 114mmHg | Not measures | Vmca was ~25% lower on the affected side (44.8±15.7 cm/s vs 59.6±12.1) prior to therapy.With therapy, Vmca increased from 58.8 to 64.2 cm/s in the unaffected hemisphere and decreased from 44.1 to 42.6 in the affected hemisphere.  |
| Berre (1994) [45] | 10 | 10 patients with sepsis | TCD Vmca | PAC | 20-minute infusion of 5 kg/kg/min of dobutamine and a 20-minute infusion of 5 ng/kg/min of PGI2 (prostacyclin) | CI increased from 3.4±0.3 to 4.2±0.4 L/min/m2 (p<0.001) with dobutamine; and from 3.5±0.3 to 4.1±0.4 (p<0.001) with PGI2 | MAP increased with dobutamine from 85±8 to 91±5mmHg (p<0.05); and decreased from 87±6 to 77±5mmHg with PGI2 | PaCO2 remained similar with dobutamine (38±2 to 38±2 mmHg) and PGI2 (39±2 to 38±2) | Mean Vmca increased with dobutamine (52±4 to 62±6 cm/s; p<0.005) but not with PGI2 (55±5 to 57±5) |
| Berre (1997) [44] | 14 | 14 mechanically ventilated septic patients | TCD Vmca | PAC | Dobutamine was infused at increasing rates of 2, 4, 6, 8, and 10 micro g/kg/min | CI increased from 3.8±0.3 to 6.3±0.5 L/min/m2 (p<0.001) with 10mcg/kg/min dobutamine in a dose dependent manner | MAP increased with 10mcg/kg/min dobutamine from 77±3 to 82±5mmHg (not significant), however more moderate doses (4mcg/kg/min) did produce small significant increases in MAP | PaCO2 remained similar with 10mcg/kg/min dobutamine (35±1 to 35±1 mmHg) | Mean Vmca increased with 10mcg/kg/min dobutamine (68±6 to 80±7 cm/s; p<0.001). Dobutamine was associated with a reduction in cerebral arterial-venous oxygen content difference and oxygen extraction ratio.  |
| Larsen (2000) [46] | 9 | 9 patients with fulminant acute liver failure | TCD Vmca | PAC | Noradrenaline infusion | CO increased from 5.7 (3.2-17.3) to 7.1 L/min (3.0-18.1) with noradrenaline (not significant) | MAP increased from 75 (54-105) to 97mmHg (90-128) (p<0.005) | Not measured | Mean Vmca increased from 49 (34-69) to 63 (58-90) cm/s (p<0.005) |