

Intravenous saline administration in patients with severe acquired brain injury and orthostatic intolerance for tilt-table mobilization

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Introduction

Severe brain injury leads to immobilisation, which affects several physiological control systems, even in healthy young subjects. These changes are likely due to changes in endocrine systems such as the renin-angiotensin-aldosterone system (RAAS) and osmotic regulatory systems.

Patients with severe acquired brain injury and low levels of consciousness need early and intensive rehabilitation but at the same time they develop orthostatic hypotension, which can affect their cerebral autoregulation of blood flow.

Administration of intravenous isotonic saline have shown to increase both intra and extracellular volumes and stabilize mean arterial pressure.

We investigated the effect of intravenous fluid administration with isotonic saline in a group of patients with severe acquired brain injury and orthostatic hypotension.

- Does the incidence of orthostatic hypotension or tachycardia decrease?
- Do standing time on a tilt-table increase?
- Does cerebral blood flow and cerebral autoregulation stabilize?
- To what extent is the RAAS affected?

Table 1. Difference in central and cerebral haemodynamics from tilt session 1 to tilt session 2.

	Baseline	HUT 30°	HUT 60°	HUT 80° ^a	HUT0	Posttilt
MAP (mmHg)	5 (3)	8 (4)	12 (3)	10 (5)	11 (3)	4 (3)
HR (bpm)	-2 (7)	-3 (3)	-4 (3)	-4 (3)	1 (3)	-1 (3)
CPPe (mmHg)	5 (3)	10 (4)	13 (3)	11 (4)	12 (4)	4 (3)
MCA Vmean (cm/s)	2 (2)	2 (2)	5 (2)	5 (4)	1 (2)	3 (2)
rScO ₂ (%)	-1 (1)	-1 (1)	-2 (1)	-2 (1)	-3 (1)	1 (2)

Positive numbers represents higher values at tilt session 2. HUT: Head up tilt; MAP: Mean arterial pressure; HR: Heart rate; CPPe: Cerebral perfusion pressure; MCA Vmean: Middle cerebral artery velocity; rScO₂: Near infrared spectroscopy determined regional frontal lobe oxygenation. Values are presented as mean with SE in brackets. a: n = 8 reached HUT 80° during tilt session 1 and 2.

Methods

Equipment:

Continuous blood pressure: photo-plethysmography measuring beat-to-beat blood pressure (mmHg).

Cerebral blood flow: Trans Cranial Doppler (2 Mhz) middle cerebral artery flow velocity (MCAV) (cm/sek).

Heart rate: Three lead ECG (bpm).

Regional cerebral oxygenation (rScO₂): Near Infrared Spectroscopy (NIRS) (%)

Estimated cerebral perfusion pressure (CPP): established through an equation taking into account the hydrostatic pressure during head-up tilt.

$$CPP = MAP_{heart\ level} - 30\ cm \times 0.7 \frac{mmHg}{cm} \times \sin(\text{tilt angle})$$

Procedure:

- Blood samples were taken in the morning before the experiment and at the end of the experiment.
- Patients were secured to the tilt table and after 5 minutes baseline measurements in supine, they were elevated head-up to 30°, 60° and 80° with one minute intervals. When at 80° the patients stood for up to 18 minutes. If orthostatic reactions occurred the patient was lowered to 0° (supine).
- After the first tilt table session patients were given 1L isotonic saline intravenously over 1 hour.
- Afterwards a second tilt table session was conducted.

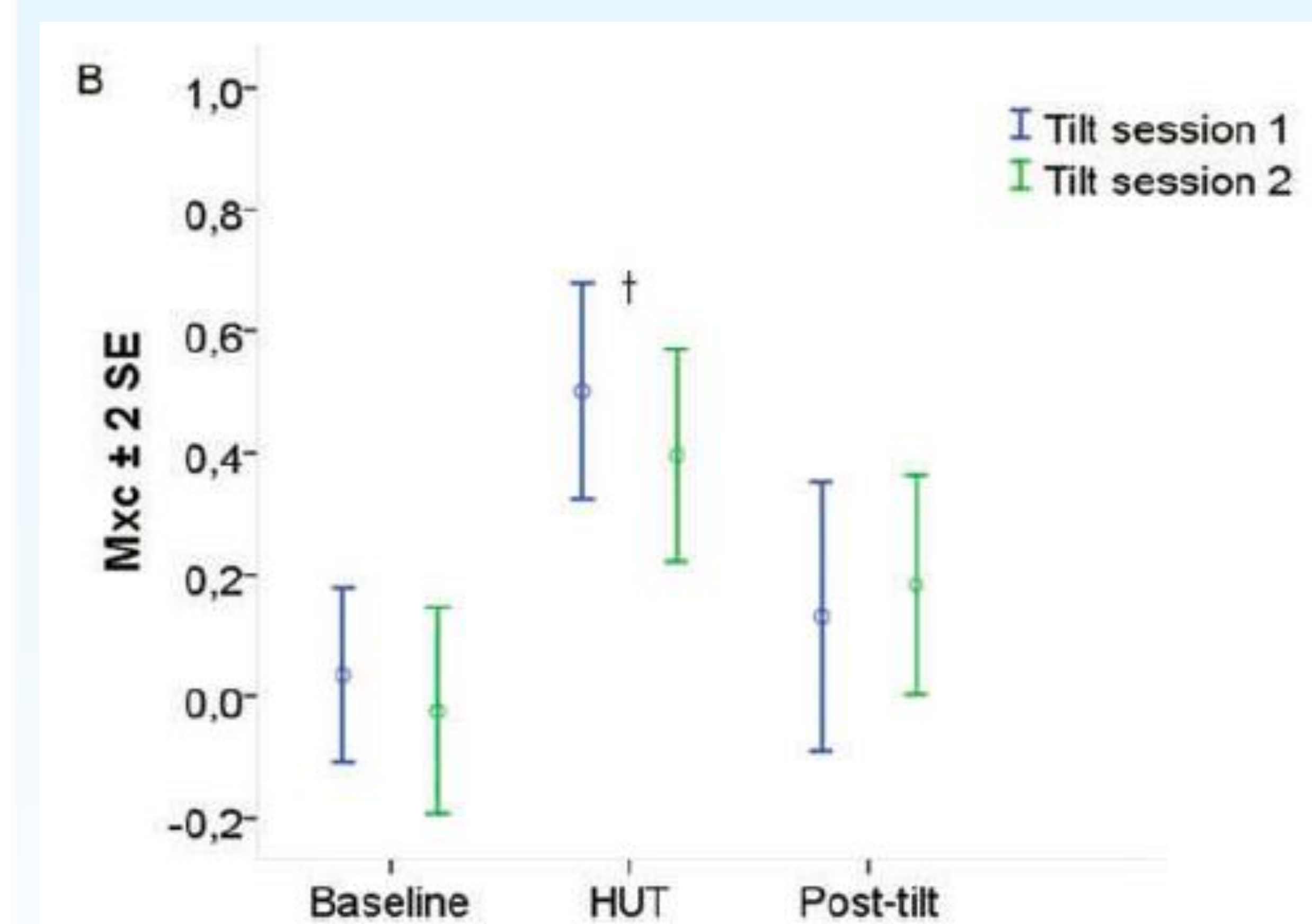


Figure 1. Cerebral autoregulation of blood flow before, during and after HUT. Mxc: Flow index based on CPPe; HUT: head-up tilt. † indicates significant change in both HUT sessions from baseline to timepoint ($P < 0.05$).

Subjects

Fourteen patients (age 56.6 ±SD 17.6) with TBI (n=8), hemorrhage (n=5) and anoxia (n=1) were consecutively included. Patients had a median GCS of 9 (IQR:8;13) and average days since injury of 41 (±SD 11.66)..

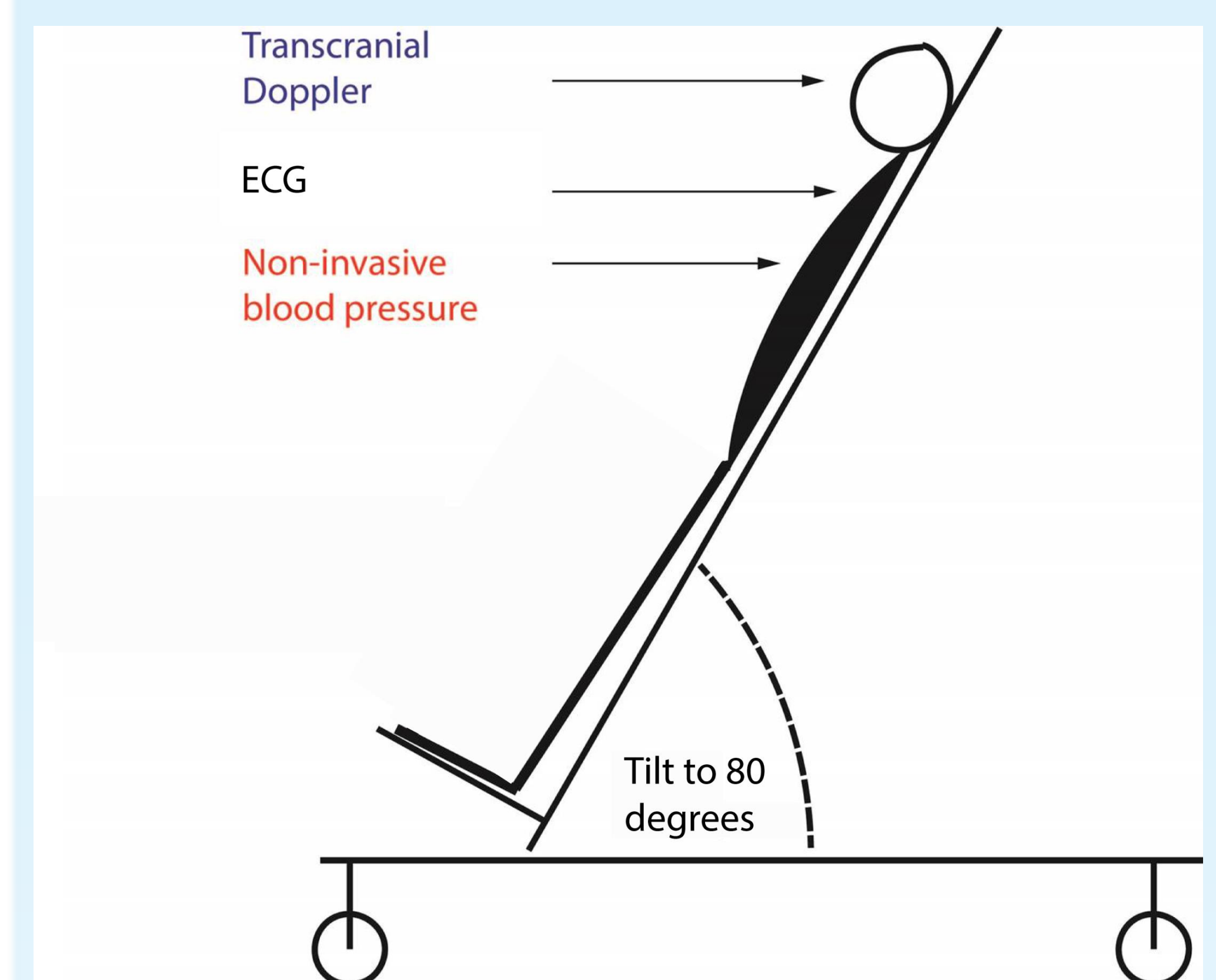


Table 2. Plasma renin, angiotensin II and aldosterone levels before and after two HUT sessions.

	Before HUT	After two HUT sessions	P-value
Renin (mIU/l)	24 (5)	15 (3)	0.0027*
Angiotensin II (pmol/l)	2.4 (IQR: 1.1-5.8)	1.2 (IQR: 0.8-3.7)	0.0359§
Aldosterone (pmol/l)	167 (IQR: 99-176)	137 (IQR: 85-230)	0.5935§

Results are given as a mean ±SE or median with inter quartile range (IQR). *twosided paired t-test. § Wilcoxon signed rank test.

Results

- No difference was found in the incidence of orthostatic hypotension. There were a tendency for higher baseline MAP, CPPe and MCA Vmean and lower HR at baseline. There was a tendency for a positive respond at the second tilt which was not significant.
- Patients did not stand for longer time on the tilt table (193 (IQR: 117–596) and 321 s (IQR: 127–526), respectively ($P = 0.51$, Wilcoxon signed rank test)). Four patients stood for a shorter period of time during the second HUT session whereas eight patients stood for a longer time and two remained unchanged.
- There was a tendency towards higher MAP and CPPe in tilt session 2 at baseline ($P = 0.054$) (table 1).
- No difference in the Mx index (cerebral autoregulation) between sessions (fig. 1) $P=0.730$ (figure 1).
- Plasma renin and angiotensin II decreased after infusion of isotonic saline and two tilt sessions (table 2).

Conclusion

This study in patients with severe ABI, disorders of consciousness and orthostatic hypotension did not demonstrate an improvement in standing time following rehydration and showed paradoxical reductions in plasma renin and angiotensin II in response to HUT. Further investigation in the RAAS for patients with severe ABI is warranted.