Altered Baroreceptor Sensitivity and Heart Rate Variability after Stroke Predict Increased Blood Pressure Variability

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Introduction

Beat-to-beat blood pressure variability (BPV) is increased after stroke and is associated with poor prognosis.¹ Reduced cardiac baroreceptor sensitivity (BRS) and heart rate variability (HRV) have also been demonstrated after stroke.²⁻⁴ Given that cardiac baroreceptors regulate short-term blood pressure (BP) stability, impaired BRS may be a causative mechanism for increased beat-to-beat BPV after stroke.

Methods

- Adults with TIA/stroke (NIHSS<15) were recruited between 72 hours and 12 weeks post-event.
- Baseline BP was taken as the mean of three clinic readings, seated at rest, using a semi-automated monitor (Omron 705IT).
- Beat-to-beat BP was measured for 10 minutes, supine at rest, using a Task Force® Monitor (APC Cardiovascular Ltd, Southport, UK). This device also measures HRV and BRS in the time domain.
- Subjects with <5 minutes valid recording and those taking beta blockers were excluded.
- Variability of systolic BP (SBP), diastolic BP (DBP), and mean arterial pressure (MAP) were calculated as the standard deviation (SD), coefficient of variation ((CV) 100 x SD/mean), and average real variability ((ARV), mean of the absolute difference between successive BP measurements).
- Data were analysed using SPSS (v.23.0). Relationships between BPV indices, BRS and HRV were assessed using Pearson’s correlation coefficient. Hierarchical multiple regression was used to assess if increased BPV was predicted by BRS or HRV after controlling for age, baseline BP, antihypertensive treatment, and stroke diagnosis.

Results

- N=97 after exclusions for beta blocker therapy and inadequate beat-to-beat BP data.
- High frequency (HF) HRV was positively correlated with ARV of SBP, DBP and MAP (r=0.3/0.5/0.4 respectively, all p<0.0001).
- Low frequency (LF) HRV was positively correlated with ARV of DBP (r=0.3, p=0.005).
- BRS was positively correlated with ARV of DBP and MAP (r=0.4/0.2 respectively, all p<0.05).
- Correlations with SD and CV were either weak or non-significant.
- Regression models for ARV of SBP, DBP and MAP are presented in the tables.
- Regression models for SD and CV of SBP, DBP, and MAP were non-significant (data not presented).

Age (years)	73.4 (9.8)
Sex (male)	78 (65%)
Diagnosis (TIA)	79 (67%)
Time to recruitment (weeks)	9.2 (3.2)
Clinic SBP (mmHg)	151.3 (18.5)
Clinic DBP (mmHg)	83.8 (11.9)
Antihypertensive treatment	109 (91%)
LF HRV (ms ²)	410.9 (784.3)
HF HRV (ms ²)	759.8 (1825.4)
LF:HF HRV ratio	2.4 (3.8)
BRS (ms/mmHg)	11.2 (8.5)

Table 1: Demographics, presented as mean (SD) or count (%).

Dependent variable	Step 1 R ²	F (df)	P value	Step 2 R ²	F change (df)	P value
SBP ARV	0.11	1.91 (6,90)	0.09	0.29	5.57 (4,86)	<0.0001
DBP ARV	0.12	2.06 (6,90)	0.07	0.42	11.32 (4,86)	<0.0001
MAP ARV	0.12	2.12 (6,90)	0.06	0.34	7.16 (4,86)	<0.0001

Table 2: Summary results of hierarchical multiple regression models for prediction of increased beat-to-beat BPV calculated as ARV.

Independent variable	Beta (SBP model)	P value	Beta (DBP model)	P value	Beta (MAP model)	P value
Baseline SBP	0.09	NS	0.04	NS	0.05	NS
Baseline DBP	-0.06	NS	0.18	NS	0.13	NS
AHT* treatment	0.06	NS	0.09	NS	0.09	NS
Stroke	0.29	0.003	0.26	0.003	0.29	0.002
Age	-0.08	NS	0.05	NS	0.01	NS
LF HRV	-0.03	NS	-0.12	NS	-0.14	NS
HF HRV	0.51	0.001	0.56	<0.0001	0.57	<0.0001
LF:HF ratio	-0.16	NS	-0.10	NS	-0.09	NS
BRS	-0.29	0.02	0.08	NS	-0.07	NS

Table 3: Influence of predictor variables in the second step regression model for ARV of SBP, DBP, and MAP.

*Antihypertensive

Conclusions

- Cardiac BRS and HRV were positively correlated with beat-to-beat ARV.
- Both predict increased beat-to-beat BPV in patients with a recent cerebrovascular event, suggesting a possible causal link.
- The therapeutic potential for improving BRS and reducing BPV after stroke should be investigated further.

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