Depression Modulates Autonomic Cardiac Control: A Physiopsychological Pathway Linking Depression and Mortality?

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Abstract

It is postulated that depressed patients have an increased risk of death due to cardiovascular disorders. Against this background, we investigated the heart rate variability (HRV) of untreated ambulatory patients complaining of depressive symptoms during their first visit to our department. Within this population, we can even demonstrate a reduced HRV in patients with a low level of depressive symptoms. These results lead us to speculate that altered autonomic cardiac control is a psychophysiological link between depressed mood and increased mortality (German J Psychiatry 2002;5:67-69)

Keywords: depression, heart rate variability

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Introduction

Depressed patients - with or without concomitant cardiac disease - may have an increased risk of dying due to cardiovascular reasons (Nemeroff et al., 1998; Wooley & Browner, 1998). Even though the clinical scores for depressive mood are not significant (screened by Beck’s Depression Inventory; BDI), it seems to affect the prognosis after acute myocardial infarctions (Bush et al., 2001). However, it still remains unclear what the underlying mechanisms are.

In patients with coronary heart disease or chronic heart failure, heart rate variability (HRV) is reduced, which indicates lowered vagal and/or increased sympathetic tone. This phenomenon is associated with an increased risk of suffering from sudden cardiac death (Bonaduce et al., 1999; Carnexy et al., 1995). Furthermore, in psychiatric patients treated with tricyclic antidepressants, HRV is dramatically reduced, although it remains unclear whether psychopharmacological treatment increases mortality (Rechlin, 1994). On the other hand, it is well known that e.g. social integration and/or physical fitness are associated with both lower cardiac risk and increased HRV (Horsten et al., 1999; Stein et al., 1999).

Patients and Methods

To clarify the question of whether depressive mood itself lowers HRV (heart rate variability), we investigated BDI (Beck Depression Inventory), blood pressure, heart rate and HRV in 22 drug-free patients who presented with depressive symptoms on their first visit to our psychosomatic outpatient clinic. None of these patients had a history of any other disease affecting autonomic control (e.g. diabe-
Physiopsychological data were also assessed on 11 age and sex matched healthy subjects (medical students/staff, BDI scores ≤ 8). ECG was assessed with a PowerLab® system and Chart® 4.6 software (ADInstruments Inc., Australia). Heart rate and short term HRV (quantified by calculating the root mean square of successive differences, RMSSD, over 3 min intervals) were calculated (a) during spontaneous breathing (RMSSDsb) and (b) during 6/min-metronomic breathing (RMSSD mb). RMSSD values are correlated to the vagal tone (i.e., the higher the RMSSD, the higher the vagal tone) and therefore this parameter has been considered to indicate vagal control of the heart.

Blood pressure was measured at the beginning of the registration procedure using cuff, manometer and stethoscope.

### Results

By BDI median split patients were divided into two groups, (a) BDI 9-19 (minor to moderate depression) and (b) BDI 20-33 (moderate to severe depression)(Table 1). Multivariate analysis (covariate age) with the independent factor group (low vs. high BDI score) and the autonomic indices (RMSSDsb, RMSSD mb, heart rate, resting blood pressure) as dependent variables revealed a significant group effect on the RMSSD not only of resting (F=3.60, p=0.025) but also of metronomic respiration (F=7.35, p=0.001): Subjects of the control group (BDI ≤ 8) had a significantly higher mean RMSSD compared to patients with moderate (BDI = 9-19) or high BDI (BDI = 20-33) scores. Additionally, there was a significant inverse correlation between the BDI scores and the RMSSD for resting (r=-0.34, p=0.055) as well as for deep respiration (r=-0.46, p=0.008).

### Discussion

Our results support findings of other researchers (Carney et al., 2000; Horsten et al., 1999; Yeragani et al., 2002) suggesting that major depression is associated with decreased cardiac vagal tone and/or a relative increase in sympathetic function. In addition, the equally reduced RMSSD values in both groups of depressed patients clearly demonstrate that even lower depressive symptoms may disturb the neurocardiac tone with measurable effects on the cardiovascular system (i.e., increased vascular tone, blood pressure, and heart rate as well as reduced HRV). As a consequence, those maladaptive autonomic and probable concomitant hormonal states (e.g. increased norepinephrine and/or cortisol levels) may increase the risk of suffering from or dying of cardiovascular disorders, e.g. by inducing arrhythmias or triggering traditional risk factors (e.g. arterial hypertension).

Regarding these findings, one can speculate that altered autonomic control of the heart is - at least in part - a link between depressed mood and increased cardiovascular risk. If further studies confirm our data, anti-depressant therapy - such as psychoeducation, psychotherapy, psychopharmacological treatment, and physical exercise programs (e.g. stretching, walking) - should be included in healthcare programs for the prevention and therapy of cardiovascular diseases even for patients with low-grade depression.

### References


Carney RM, Saunders RD, Freedland KE, Stein P, Rich MW, Jaffe AS. Association of depression with re-

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### Table 1: Epidemiological and cardiovascular parameters. Means and Standard Deviations. Analysis of variance

<table>
<thead>
<tr>
<th></th>
<th>BDI 0-8</th>
<th>BDI 9-19</th>
<th>BDI 20-33</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>N (female/male)</td>
<td>11 (8/3)</td>
<td>11 (8/3)</td>
<td>11 (8/3)</td>
<td>n.t.</td>
</tr>
<tr>
<td>BDI</td>
<td>4.4 (2.5)</td>
<td>13.9 (3.1)</td>
<td>25.5 (5.3)</td>
<td>p&lt;0.001</td>
</tr>
<tr>
<td>Age [years]</td>
<td>42.4 (25.4)</td>
<td>44.9 (13.2)</td>
<td>38.4 (14.4)</td>
<td>n.s.</td>
</tr>
<tr>
<td>Systolic blood pressure [mmHg]</td>
<td>119.2 (10.8)</td>
<td>123.4 (16.1)</td>
<td>120.3 (19.6)</td>
<td>n.s.</td>
</tr>
<tr>
<td>Diastolic blood pressure [mmHg]</td>
<td>73.5 (13.2)</td>
<td>83.1 (10.7)</td>
<td>82.3 (10.0)</td>
<td>n.s.</td>
</tr>
<tr>
<td>Heart rate [1/min] – spontaneous breathing</td>
<td>68.8 (9.2)</td>
<td>72.0 (7.5)</td>
<td>77.6 (15.1)</td>
<td>n.s.</td>
</tr>
<tr>
<td>RMSSD [ms] – spontaneous breathing</td>
<td>60.2 (39.4)</td>
<td>28.1 (11.2)</td>
<td>31.8 (19.3)</td>
<td>p&lt;0.05</td>
</tr>
<tr>
<td>Heart rate [1/min] – metronomic breathing 6/min</td>
<td>70.8 (8.9)</td>
<td>73.2 (7.7)</td>
<td>80.3 (11.5)</td>
<td>n.s.</td>
</tr>
<tr>
<td>RMSSD [ms] – metronomic breathing 6/min</td>
<td>73.2 (32.1)</td>
<td>41.3 (22.2)</td>
<td>41.7 (22.8)</td>
<td>p&lt;0.05</td>
</tr>
</tbody>
</table>

n.s., not significant; n.t, not tested. Other abbreviations see text.
duced heart rate variability in coronary artery disease. Am J Cardiol 1995; 76: 562-564.


