Supplementary material:
Sympathetic vasoconstrictor activity before and after left ventricular assist device implantation in patients with end-stage heart failure

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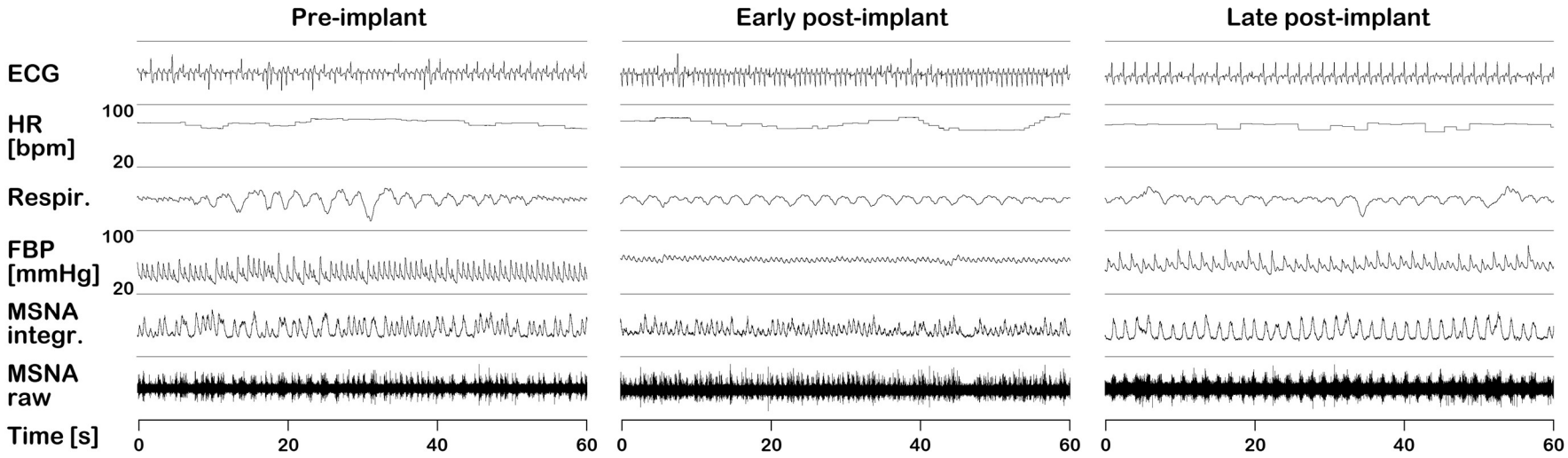
Figure legend

Longitudinal original recordings of the same patient (patient #5) at pre-, early, and late post-implantation stages. LVAD rotational frequency was 2480 rpm on both post-implantation occasions. Note the Cheyne-Stokes breathing before implantation which disappeared afterwards. The recordings show, side by side, how arrhythmia severity levels incrementally confound the sympathetic neural bursting pattern and its assessment from low (early post-implant), moderate (pre-implant), to severe manifestation (late post-implant). Eleven months post-implantation, the patient presented a bigeminal rhythm with hemodynamic consequences that have to be considered when assessing sympathetic activity. Prolonged cardiac filling time caused a higher preload which led to increased pulse pressure (early 5.8 vs late 15.6 mmHg) and aortic valve opening while cutting the number of diastolic events that are able to generate sympathetic bursts. Thus, the specious reduction in sympathetic burst frequency and burst incidence is misleading (see also Figure 1B in the main text, patient #5). Only every second cardiac *electrical* event generates a diastolic pressure that is low enough to sufficiently silence baroreflex receptor afferents, thereby disinhibiting sympathetic nerve traffic. The central neural 'conclusion' is that *hemodynamic* pulse interval has doubled and sympathetic bursts are generated appropriately, with lower frequency, broader base, and taller amplitude, indicating more sympathetic action potentials per burst. This pattern is also present before LVAD implantation and has been described previously as 'sympathetic alternans' in synchrony with pulsus alternans.[1,2]

ECG, electrocardiogram; HR, heart rate; Respir., respiration; FBP, finger blood pressure; MSNA, muscle sympathetic nerve activity.



References

1. Ando S, Dajani HR, Senn BL, Newton GE, Floras JS. Sympathetic alternans: evidence for arterial baroreflex control of muscle sympathetic nerve activity in congestive heart failure. *Circulation* 1997;**95**:316–319.

2. Floras JS. Arterial baroreceptor and cardiopulmonary reflex control of sympathetic outflow in human heart failure. *Ann N Y Acad Sci* 2001;**940**:500–513.