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NEW EVIDENCE FOR VAGAL CONTROL OF THE LEFT HEART

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The role of the parasympathetic innervation of the left ventricle remains controversial, although there is some evidence that vagus nerve stimulation decreases left ventricular contractility in vivo in the human and pig heart. The aim of this study was to identify the anatomical location of vagal preganglionic neurones that control ventricular contractility using an anaesthetised rat model. Male Sprague-Dawley rats (350–400 g, n=6) were anaesthetised with pentobarbitone sodium (induction 60 mg/kg i.p.; maintenance 15–20 mg/kg/hr i.v.) and artificially ventilated. The left femoral artery and both femoral veins were cannulated for the measurement of arterial pressure and administration of fluids and drugs. The right carotid artery was cannulated for left ventricular pressure recording using a Millar pressure probe. Lead II ECG was also monitored. The animal was placed in a stereotaxic frame and the dorsal surface of the brainstem was exposed. To activate vagal preganglionic neurones, an excitatory neurotransmitter glutamate (10 mM, 40 nl, pH 7.4) was microinjected into three discrete locations (separated by 0.5 mm) along the rostro-caudal extent of the left and right dorsal motor nuclei of the vagus nerve (DVMN). Responses were measured in a second group (n=3) with transoesophageal atrial pacing (TAP) to abolish chronotropic changes. A third group (n=2) underwent a spinal transection at C1 to remove sympathetic influences followed by gelofusine and vasopressin infusion to maintain mean arterial blood pressure at ~100mmHg. Values are means±S.E.M., compared by paired student's t-test. Glutamate microinjections into the most caudal part of the left DVMN caused a significant (p