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Title : HEART RATE CONTROL IN MAN DURING NITROPRUSSIDE HYPOTENSION

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Introduction: A method has been described for assessing the baroreceptor reflex in man[1]. The principle of the method is to use a drug which alters systolic pressure (SP) by producing vasoconstriction or vasodilation without a direct effect on cardiac pacemaker, so that the observed changes of pulse interval (PI) are mediated reflexly through baroreceptors. It is generally agreed that reflex bradycardia associated with hypertension is caused primarily by vagal enhancement. The mechanism of reflex tachycardia during induced hypotension, however, is not clearly understood. The present investigation was designed to study the roles of sympathetic and vagal activities on SP-PI relationship in man during hypotension induced by sodium nitroprusside (SNP).

Methods: Ten informed patients were premedicated with morphine and secobarbital. Following induction and intubation with thiopental and succinylcholine, anesthesia was maintained with halothane(0.3-0.5%, inspired)-N_2O(60%)-O_2 mixture and metocurine (0.3 mg/Kg) throughout the study. P_aCO_2 was controlled at 25-30 mmHg with a respirator. Direct arterial pressure (via a radial artery), EKG and airway pressure were recorded continuously. A small dose of SNP (3-6 ug/Kg, I.V.) was given to produce a transient decrease in SP by about 30 mmHg, and SP was allowed to recover spontaneously to its resting level (within 3-4 min). This period of hypotension served as the control SNP test on baroreceptor reflex. Five of these patients were then treated with atropine (0.01-0.02 mg/Kg, I.V.), while the other five received propranolol (0.05-0.1 mg/Kg). Two min later, the post-treatment SNP test on baroreceptor reflex was performed with the same dose of SNP as in the control test. The SP and its succeeding PI (R-R on EKG) were sampled every 3-5 sec during the entire course of SNP test. Those heart beats falling on inspiratory phase were excluded in order to avoid possible sinus arrhythmia related to ventilation. The relationship between SP and the succeeding PI was evaluated on a beat to beat basis.

Results: Following the administration of SNP, PI usually continued to fall when SP had already reached its maximal response. The lowest value of PI (PI_{min}) was attained later than that of SP (SP_{min}). This phase shift between responses of PI and SP was 20.0 ± 2.5 sec (mean ± SEM) for ten control tests. As a result of the phase shift, there was a hysteresis on SP-PI plot (Fig 1). A linear relationship was found between SP and PI during the initial phase of pressure decrease (A-B) as well as during the recovery phase (C-D). Correlation coefficients for all regression lines obtained were greater than 0.88. The PI corresponding to SP_{min} was ascertained and a horizontal line was drawn to intercept the regression line for the initial phase (Point B) and that for the recovery phase (Point C). The pressure difference between points B and C was used as an index of hysteresis, which averaged 17.8 ± 2.4 mmHg for the control test.

Following atropine treatment, the slopes of SP-PI regression lines decreased by 65% below the control values for the initial phase and by 60% for the recovery phase. Atropine also significantly increased phase shift and hysteresis by 70% and 176% over the control, respectively. In contrast, propranolol caused marked decreases in phase shift and hysteresis. The slopes of SP-PI regression lines during both the initial and recovery phases remained unaltered after propranolol treatment.

Discussion: The findings that slopes of SP-PI regression lines were depressed by atropine but unaffected by propranolol indicate a predominance of vagal influence on the heart rate response to changing pressure. Since the phase shift and hysteresis were markedly decreased by propranolol and significantly increased by atropine, it seems that sympathetic activity also plays a prominent role when the arterial pressure reaches a relatively low level. It is concluded that the autonomic control of heart rate in man in response to changing arterial pressure is dominated by vagal influence and that sympathetic activity becomes evident as vagal influence subsides more quickly when the arterial pressure stays relatively constant at a new level.

Reference:

1. Smyth HS, Sleight P, Pickering GW: Reflex regulation of arterial pressure during sleep in man: A quantitative method of assessing baroreflex activity. Circ. Res 24:107-121, 1969

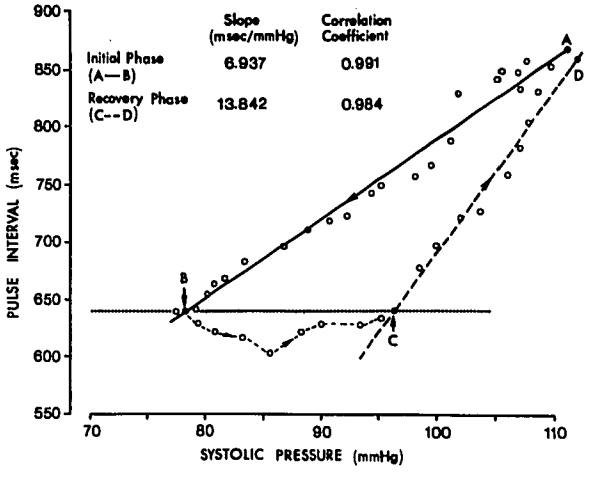

Fig. 1 Correlation of SP with the succeeding PI during the entire course of a SNP test.