

Title: PERIPHERAL VASCULAR VERSUS CARDIAC RESPONSE TO  $\text{CaCl}_2$  INJECTION IMMEDIATELY FOLLOWING CARDIOPULMONARY BYPASS IN MAN

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**Introduction.** Whether calcium chloride ( $\text{CaCl}_2$ ) increases systemic blood pressure (SBP) by enhancing myocardial contractile state<sup>1</sup>, or by increasing systemic vascular resistance<sup>2</sup>, (SVR) remains controversial. To clarify this, we studied the hemodynamic response to injection of  $\text{CaCl}_2$  in 18 patients upon conclusion of extracorporeal circulation.

**Methods.** The protocol was approved by the institutional human studies committee and signed informed consent was obtained from each patient. After termination of bypass and initial hemodynamic stabilization control measurements were made and  $\text{CaCl}_2$  10 mg/kg injected into the right atrium over 10 seconds. Hemodynamic responses were recorded continuously for the next 5 minutes, after which a second injection of the same dose was given and measurements repeated. Contractile element velocity (Millar transducer) - peak measured value (Vpm), aortic blood flow (electromagnetic flow-probe), ECG, systemic arterial (SBP), left ventricular, pulmonary arterial and right atrial pressures were continuously recorded. Ionized calcium was measured prior to injection and one, three and five minutes thereafter.

**Results.** Control ionized Ca was  $3.6 \pm 0.6$  mg/100 ml, increased to  $5.4 \pm 0.8$  mg/100 ml one minute after injection, and was  $4.7 \pm 0.6$  mg/100 ml in five minutes. Hemodynamic data are summarized in the graph. Early hemodynamic enhancement was consistently observed - maximum at  $23.7 \pm 11.5$  seconds ("Max CI") - and included an increase in Vpm, cardiac index, SBP and in stroke volume index (SVI), while heart rate (HR) and SVR remained unchanged. At  $69.8 \pm 27.1$  seconds, CI returned to control level while HR declined. SVR gradually increased and was significant at six minutes. SBP remained elevated throughout the period of study.

**Discussion.**  $\text{CaCl}_2$  exerted an immediate and a sustained enhancement of myocardial contractile state during the immediate post-bypass period in man. This was accompanied by an initial increase in CI and SVI. The subsequent decline in heart rate was associated with a return of CI to control levels. The SBP remained elevated throughout the study period and appeared to be sustained by a rise in SVR after the initial increase in CI. Injection of a second dose of  $\text{CaCl}_2$  (acute hypercalcemia) was followed by a similar pattern of response: Rapid and sustained rise in contractile state and an initial but transient increase in CI and SVI, followed by a slowing of heart rate, return of CI to control, and a rise in SVR. Thus in this clinical setting acute hypercalcemia did not modify the response to  $\text{CaCl}_2$ , in contrast to observations of others.<sup>3</sup> We conclude that  $\text{CaCl}_2$  injection produces 1) a rapidly developed and sustained enhancement of myocardial contractile state, 2) a transient increase in CI, followed by a slowing of heart rate and return

of CI to control levels, 3) persistent elevation of SBP related to a rise in SVR, and 4) a pattern of hemodynamic response that is similar during both hypocalcemia and acute hypercalcemia.

**References.**

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