**INTRODUCTION:** In a series of 518 consecutive adult/patients undergoing cardiac surgery bradycardia (heart rate less than 60 beats/minute) produced significant anesthetic management problems in 41 patients including 2 who developed cardiac arrest. This study is an analysis of the predisposing factors and hemodynamic changes associated with bradycardia (BC) in these patients.

**METHODS:** Patients were premedicated with diazepam, morphine and scopolamine. Monitoring included six standard EKG leads and lead V5, constant recording of heart rate (HR) with a cardiacthrometer, recording of intravascular pressures and flow with intra-arterial and pulmonary artery catheters (introduced prior to induction).

**RESULTS:** BC requiring treatment occurred in 41 patients who could be divided into 2 groups: Group I, patients who developed sudden BC; Group II, patients who presented with BC that persisted until treatment. Group I: There were 22 patients, 17 male, 5 female, mean age 55 ± 11 years. In figure 1, HR at baseline and at time of BC is presented and patients grouped in relation to total daily preoperative dosage of propranolol (P); dosage of P was not tapered before anesthesia. Four patients in this group developed cardiac arrest and were the only patients with this complication during the calendar year reviewed: two had not received P preoperatively but had been given intravenous P prior to induction and developed cardiac arrest which responded to closed chest massage, atropine (At) and vasopressor agent; two had received P preoperatively and presented with baseline HR of 40–one developed sinus arrest after skin incision and the other ventricular fibrillation following sternotomy.

Events immediately preceding BC were infusion of succinylcholine-1, laryngoscopy-6, skin incision-10, sternotomy-4, and in 1 patient administration of fentanyl 2 ml for sedation prior to induction with attendant hypotension. In the 22 patients of Group I, BC was associated with hypotension in 20 patients but in only 1 patient did hypotension precede BC. Ischemic EKG change occurred only in the patient who fibrillated. Hypotension was not productive of increase in HR in any of the patients, and HR responded only to pharmacologic treatment. In 12/20 patients hypotension involved fall in systolic pressure to less than 100 torr.

Group II: There were 19 patients, 18 male, 1 female, mean age 57 ± 7 years; initial HR was between 38 and 58 beats/minute; all patients received P preoperatively. HR responded to incremental doses of At except in 3 cases when the initial level of BC (46, 43, 53 beats/minute) was not corrected prior to induction; in these 3 cases systolic blood pressure fell 30 torr or more at induction and was then corrected by administration of At. In a fourth case, baseline HR 50, response to At was transient, with recurrence of BC and subsequent hypotension.

**DISCUSSION:** BC and hemodynamic instability occurred in 7.7% of 518 patients. When acute in onset, the event was usually related to a change in level of stimulation. In the entire series of 518 patients only 3 patients presented with a baseline HR of 40 or less and 2 of these 3 patients subsequently developed cardiac arrest. There was a 38% incidence of hypotension in the BC cases, although myocardial ischemia was apparent only in 1 patient and followed cardiac arrest. The two groups of BC patients accounted for all cases of severe hypotension in the total series of 518 patients. This experience indicates that patients presenting with baseline HR less than 50 beats/minute should benefit from elevation of HR by pharmacologic agent or by intracardiac pacing.

![Fig. 1: Group I: HR at baseline and at time of treatment in relation to total daily preoperative dose in mg/m of P (given as one of 4 ranges: A=0, B=40-120, C=160-180, D=200+)]