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Does Prophylactic Lidocaine Control Cardiac Arrhythmias Associated with Pulmonary Artery Catheterization?

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The pliable balloon tip of the Swan-Ganz catheter (SG catheter) attenuates cardiac arrhythmias associated with endocardial contact.[1] Ventricular arrhythmias still occur frequently during SG catheterization.[2,3] Administration of lidocaine (50–100 mg, iv) has been advocated prior to the floatation of the catheter tip through the right heart to electrically stabilize the myocardium.[4] However, after routine administration of prophylactic lidocaine, we were unable to observe any distinct diminution in catheter-induced ectopic activity. This double-blind comparison of lidocaine against saline was performed to examine our impression in a more controlled manner.

METHODS

One hundred seven patients scheduled for cardiac surgery were allocated randomly to three study groups receiving either 1 mg/kg or 2 mg/kg of 1 per cent or 2 per cent lidocaine, respectively, or an equal volume of 0.9 per cent saline, iv, in a double-blind fashion. Only patients with sinus rhythm and devoid of ectopic activity for at least two minutes before catheterization were included in the study. The three groups were comparable with respect to their sex distribution, age, weight, and type of surgical approach (table 1). The medications were continued up to and including the preoperative day. The proportion of patients receiving digitalis preparations or β-blocking agents was not different among the study groups.

Anesthesia was induced with 5–10 mg droperidol followed by 0.5–1 mg fentanyl and 0.1 mg/kg pancuronium, iv, to facilitate endotracheal intubation. Ventilation was controlled while administering 50 to 60 per cent nitrous oxide. Access to the venous circulation was via the right internal jugular vein or when this was unsuccessful, via the right innominate vein. After the introducer had been inserted into the vein, lidocaine or saline was injected into an arm vein. Two minutes later, a 7-Fr SG catheter was inserted, the balloon inflated with 1.5 ml of air, and the catheter advanced to the right heart. If the catheter failed to reach the wedge position in the pulmonary artery within two minutes the patient was excluded from the series. The floatation times in the respective study groups are indicated in table 1.

The ECG was recorded via a precordial lead throughout the catheterization procedure, as were the pressure tracings from the distal lumen of the SG catheter and the radial artery catheter. The concentrations of lidocaine in the arterial blood following the 2 mg/kg dose of lidocaine were determined according the gas chromatography method described by Mather and Tucker.[5] This was done to confirm that an antiarrhythmic concentration of lidocaine had been reached.

Incidence and frequency of ventricular ectopic beats were recorded. The occurrence of any ventricular tachycardia (defined as three or more successive beats of ventricular origin with a frequency of more than 150 bpm) was also noted. Appropriate modifications of the t test and the chi-square test were used for statistical comparisons. $P < 0.05$ was considered significant.

RESULTS

Catheter-induced ectopic ventricular beats were observed in 65 per cent (35/54) of the control patients. These occurred in a frequency exceeding five beats per minute in 52 per cent (28/54) of the cases. Ventricular

TABLE 1. Patient Characteristics and Floatation Times (Mean ± SD)

Group	Operation C/V*	Age (yr)	Weight (kg)	Floatation Time† (s)
Saline	28/26	52.3 ± 8.0	74.4 ± 12.6	63.9 ± 22.7
Lidocaine 1 mg/kg	16/13	54.3 ± 7.2	74.0 ± 11.0	79.2 ± 20.8
Lidocaine 2 mg/kg	14/10	74.1 ± 9.1	54.1 ± 20.7	75.7 ± 26.2

* C/V = coronary bypass surgery/valvular surgery (those with combined surgery scored in the coronary bypass surgery group).
† Floatation time = (s) of catheter tip passage from venous entry to wedge position.

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TABLE 2. Occurrence of Ventricular Ectopic Beats and Ventricular Tachycardia Induced by Swan-Ganz Catheterization after Saline or Lidocaine (1 and 2 mg/kg, iv)

Pretreatments (Number of Patients)	Number (and Per Cent) of Patients with Catheter-Induced	
	Ventricular Ectopic Beats	Ventricular Tachycardia
Saline (54)	35 (64.8)	10 (18.5)
Lidocaine 1 mg/kg (29)	22 (75.9)	5 (17.2)
Lidocaine 2 mg/kg (24)	16 (66.7)	3 (12.5)

TABLE 3. Frequency Distribution of Ventricular Ectopic Beats While the Swan-Ganz Catheter Tip Was Moving Through the Right Ventricle to Wedge Position

Ventricular Ectopic Beats/Min	Number of Patients		
	Saline	Lidocaine 1 mg/kg	Lidocaine 2 mg/kg
0	19	7*	8†
1–5	7	5	8
>5	28	17	8

* χ^2 test (lidocaine 1 mg/kg *vs.* saline); $\chi^2 = 1.13$; ($P > 0.75$).
† χ^2 test (lidocaine 2 mg/kg *vs.* saline); $\chi^2 = 4.84$; ($P > 0.10$).

tachycardia was seen in 19 per cent (10/54) of the control patients. In 24 per cent (13/54) of the patients ventricular extrasystoles were observed while the catheter tip passed through the right ventricular outflow tract. There was no difference in the incidence of these arrhythmias between patients undergoing coronary artery bypass and those undergoing valvular surgery.

The number of patients experiencing catheter-induced ventricular ectopic beats or tachycardia was unaffected by lidocaine (1 or 2 mg/kg) pretreatments (table 2). The intensity of ventricular ectopy tended to decrease after the higher lidocaine dose as revealed by the frequency distribution of ventricular extrasystoles and the incidence of ventricular tachycardias (tables 2 and 3). However, these decreases were not significant statistically. This dose resulted in a concentration of 10.0 ± 3.9 μg/ml (mean $\pm$ SD, n = 24) of lidocaine in arterial blood at the time of catheterization. Two minutes after completion of injection the arterial blood pressures were unaffected after 1 mg/kg of lidocaine. Systolic blood pressure decreased from 114.8 ± 12.3 to 109.3 ± 16.1 mmHg (mean $\pm$ SD) ($P < 0.01$ when compared to the saline group) after the 2 mg/kg dose of lidocaine.

DISCUSSION

Several patients undergoing open-heart surgery had arrhythmias when the SG catheter was inserted. Our incidence of 65 per cent rhythm disturbances is clearly higher than previously reported but is in accordance with recent prospective studies also using continuous ECG recording throughout the catheterization procedure.[3,6,7] In none of our patients was the arrhythmia sustained after the desired position in the pulmonary artery was reached. Nevertheless, persisting ventricular tachycardia, ventricular fibrillation, and even death because of failure of subsequent resuscitation are reported complications of SG catheterization.[7-9]

We failed to show that 1 mg/kg lidocaine given iv 2–4 minutes before the SG catheter was passed through the right ventricle had any significant beneficial effect. This contradicts the results of Shaw,[6] who reported a decrease in catheter-induced ventricular ectopy from 39 to 5 per cent after the iv administration of lidocaine. We find no obvious explanation for the different results. The dose and timing of drug administration and the patient series were essentially the same.

A trend and certainly clinically insignificant effect of lidocaine was seen only after the dose of 2 mg/kg. Perhaps with a larger number of patients, this trend (tables 2 and 3) would have become statistically significant. However, this 2 mg/kg dose resulted in blood levels well in excess of those normally considered to be adequate for antiarrhythmic activity in patients suffering from myocardial infarction.[10]

This suggests that the arrhythmias resulting from mechanical events during catheter passage are clearly more resistant to lidocaine than those provoked by myocardial ischemia. Lidocaine up to 2 mg/kg did not markedly deteriorate the cardiovascular state of our patients. It is, however, possible that CNS symptoms would have occurred in some of the patients, if not masked by anesthesia, since toxic symptoms have been shown to occur with venous lidocaine concentrations of 4–5 μg/ml.[11]

We conclude that because of the benign and reversible nature of the catheter-induced ventricular ectopy, the ineffectiveness of lidocaine in suppressing it, and the potential detrimental effects associated with its use, prophylactic lidocaine bolus is not warranted before SG catheterization of patients scheduled for open-heart surgery.

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Coronary Air Embolism during Venous Cannulation

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Venous air embolism during insertion of intravascular catheters rarely presents a significant hazard in most patients. When intracardiac shunts are present, systemic air embolism may occur. We report a case of venous air embolism leading to air in the coronary arteries.

REPORT OF A CASE

A 60-year-old woman was scheduled for total correction of a tetralogy of Fallot. She had no previous surgery. She suffered from decreasing exercise tolerance over many years and was admitted following an episode of hemoptysis. Several days following recovery from this episode, physical examination revealed a cyanotic woman with arterial blood pressure 110/80 mmHg, heart rate 95 beats/min, and respiratory rate 28/min. While breathing room air, Pa_{O_2} and Pa_{CO_2} were each 35 mmHg, and pH_a was 7.51. The hematocrit was 46 per cent. Cardiac catheterization demonstrated a 4.2 l/min right-to-left shunt at the ventricular level, infundibular pulmonic stenosis, overriding aorta, and normal coronary anatomy without intraluminal obstructions.

The patient was brought to the operating room without premedication. Oxygen was administered nasally at 4 l/min. A five lead electrocardiogram monitor was used. A 1-mV calibration pulse in the diagnostic mode spanned 22 mm on the recorder (fig. 1*A*). Peripheral intravenous and radial artery cannulae were inserted. With the patient tilted head down 10°, a 20-gauge catheter was inserted into the right internal jugular vein. Visualization of venous waveforms by pressure transduction confirmed venous cannulation,[1] an important feature in patients with cyanotic heart disease. A 0.025-inch diameter spring guide wire passed through the catheter easily and an 8-Fr dilator-introducer was inserted over the wire into the vein.

The patient was instructed to hold her breath and the dilator was removed and replaced with a 16-gauge 5¼-inch catheter. However, she inspired deeply while the introducer was open to atmosphere. At that instant, an assistant at the patient's side heard a churning "mill wheel" sound coming from the patient's precordium. No attempt was made to change the patient's position. Within a minute, the patient complained of heavy precordial pressure. Mean arterial blood pressure then dropped from 90 to 45 mmHg. ECG lead V_5 demonstrated severe ST segment elevation and intermittent second degree AV block (fig. 1*B*). Her chest pain resolved over several minutes, accompanied by an increase in mean arterial pressure to 90 mmHg and improvement in the ECG (figs. 1*C*, 1*D*, and 1*E*). Her mentation and her sensory and motor functions were assessed throughout this episode; they did not change from baseline. Anesthesia and surgery proceeded without difficulty. High-dose fentanyl with 100 per cent oxygen provided acceptable anesthetic conditions. Several postoperative visits disclosed no myocardial, neurologic, or other sequelae of the embolic episode.

DISCUSSION

This woman sustained venous air embolism massive enough to allow detection without ultrasound or even a stethoscope. This occurred despite several precautions which minimize the likelihood of air entrainment into the circulation: head-down tilt to increase central venous pressure (CVP), occlusion of the introducer to prevent continuity with the atmosphere, and asking the patient to hold her breath while the introducer was exposed to air. Conahan[2] detected air embolism in one of twelve patients monitored with precordial Doppler ultrasound units during placement of 8-Fr pulmonary artery catheter introducer sheaths. In that patient, there were no hemodynamic alterations. The same study[2] showed that a pressure gradient of 4 mmHg causes flows of 90 ml/s, enough to result in fatal venous air embolism.[3] Great

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