SUCCESSFUL TREATMENT OF LIFE-THREATENING VENTRICULAR TACHYCARDIA WITH HIGH-DOSE PROPRANOLOL UNDER EXTRACORPOREAL LIFE SUPPORT AND INTRAAROTIC BALLOON PUMPING

MITSURO KURose, M.D., KAZUFUMI OKAMOTO, M.D., TOSHIHIDE SATo, M.D.
RIICHIRO YATSUDA, M.D., KENICHI OGATA, M.D., MASANOBu YASumoto, M.D.
HIDENORI TERASAKI, M.D., HIRAAM GOTO, M.D.
AND KEN OkUMURA, M.D.

The use of propranolol is generally contraindicated in patients with congestive heart failure. However, we successfully used a continuous high-dose infusion of propranolol, in combination with the use of extracorporeal life support (ECLS) and intra-aortic balloon pumping (IABP), to treat refractory life-threatening ventricular tachycardia in a patient with postoperative congestive heart failure. The early combined use of ECLS and IABP before irreversible myocardial damage contributed to the successful outcome.

(Jpn Circ J 1993; 57: 1106–1110)

Refractory ventricular tachycardia (VT) is a life-threatening arrhythmia, especially in patients with heart failure! We describe here a patient who developed refractory VT with congestive heart failure just after ventricular aneurysmectomy and cryosurgery. Treatment with a continuous high-dose infusion of propranolol under extracorporeal life support (ECLS) plus intraaortic balloon pumping (IABP) was successful.

CASE REPORT

A 48-yr-old man weighing 73 kg with a left ventricular aneurysm and frequent syncope induced by refractory VT or ventricular fibrillation following broad anterior myocardial infarction underwent ventricular aneu-

rysmectomy and cryosurgery. Preoperative examinations showed 75% stenosis of the proximal left anterior descending artery in the coronary arteriogram and dyskinesis of the apex with an ejection fraction of 29% by left ventriculography.

Ventricular aneurysmectomy was performed uneventfully. However, because VT could not be provoked by programmed ventricular stimulation, intraoperative pace-mapping was performed. By pacing at various ventricular sites during sinus rhythm, the site where pacing produced a similar QRS complex configuration to that of the patient's previous premature ventricular contraction was identified. Cryosurgery (−60°C for 2 min) was then performed at both the epicardial and endocardial sites of this presumed source of VT.

Postoperative cardiac function was severely depressed and circulatory assist using

Key words:
Ventricular tachycardia
Congestive heart failure
Propranolol
Extracorporeal life support and intraaortic balloon pumping

(Received December 8, 1992; accepted April 16, 1993)
Division of Intensive & Critical Care Medicine, the Department of Anesthesiology, the 1st Department of Surgery and the Division of Cardiology, Kumamoto University School of Medicine, Kumamoto, Japan
Mailing address: Mitsuuro Kurose, M.D., Division of Intensive and Critical Care Medicine, Kumamoto University School of Medicine, Kumamoto City 860, Japan

1106 Japanese Circulation Journal Vol. 57, November 1993
IABP was required. Although there were no ischemic ST segment or T wave changes in the 12-lead electrocardiogram (ECG), ventricular arrhythmias, including premature ventricular contractions (PVC) and VT, were frequent. Direct current cardioversion (DC) was frequently required because the VT was resistant to pacing and various antiarrhythmic agents, including lidocaine, mexiletine, disopyramide, phenytoin, verapamil, and magnesium (Figs. 1 and 2). About 60 h after surgery, the systolic blood pressure and urine output had decreased to 80 mmHg and 15 ml/h, respectively. The arterial oxygen tension (PaO₂) also fell to 50 mmHg with an inspired oxygen fraction (FiO₂) of 1.0. Therefore, ECLS was initiated using a venoarterial bypass and a membrane oxygenator (Carmede Maxima, Sweden). Blood was withdrawn from the right atrium and reinfused into the right subclavian artery, with a pump flow of 3 L/min.

After starting ECLS, the systolic blood pressure and urine output rose to 110 mmHg and 115 ml/h, respectively. The PaO₂ increased to 160 mmHg, the frequency of VT gradually decreased and DC was not required for a period of time. However, 2 days after the start of ECLS, uniform VT emerged again and its frequency gradually increased. DC was required more frequently than before ECLS (Fig. 2). Five days after starting ECLS, the decision was made to conduct another cardiac cryosurgery to treat this refractory VT.

This time, VT with the same electrocardiographic configuration as that of the spontaneous VT was successfully provoked during surgery. Sequential site epicardial mapping was performed during one episode of
VT and the earliest activation site was found at the lateral aspect of the left ventricle close to the obtuse marginal branch of the circumflex coronary artery (Fig. 3). This site was located in the border of the scar tissue left after myocardial infarction. Cryosurgery of the endocardium opposite the epicardial site of earliest activation was performed and appeared to be successful.

However, VT occurred frequently even after the second cryosurgery procedure and was resistant to pacing and the previously mentioned antiarrhythmic drugs. Only an intravenous bolus injection of propranolol was temporarily effective. Therefore, continuous infusion of propranolol was begun. To suppress VT, the infusion rate of propranolol was titrated and cautiously increased from 1 to 4.8 mg/h. As a result, the serum concentration of propranolol increased to 242 ng/ml.

The frequent episodes of ventricular arrhythmias, which had continuously disturbed synchronization of IABP with the ECG or blood pressure, disappeared after initiation of the continuous infusion of propranolol (Figs. 1 and 2), and the blood pressure became stable. Blood lactate level, which was 3.93 ng/ml just after the second cryosurgery procedure, decreased gradually to within the normal range and oxygenation also gradually improved.

Eleven days after the start of ECLS, it was discontinued uneventfully and IABP was also discontinued 3 days later. Although ventricular arrhythmias were observed just after the cessation of IABP, these disappeared within a few days. Twenty-four days after starting the continuous infusion of propranolol, it too was discontinued without the recurrence of any life-threatening arrhythmias. Oral administration of atenolol (50 mg) was begun. An electrophysiologic study was performed about 3 months after the second cryosurgery procedure. VT was no longer induced despite programmed ventricular stimulation using up to 3 extrastimuli and ventricular burst pacing. The patient was discharged ambulatory from hospital and has had no further episodes of syncope for 16 months.

DISCUSSION

Left ventricular depression commonly occurs following cardiac surgery. However, this postoperative myocardial dysfunction is often reversible if the circulation can be appropriately assisted for a few days?

VT itself causes severe left ventricular de-
pression and may also finally degenerate into ventricular fibrillation. Thus, when VT markedly reduces the blood pressure, DC should be performed immediately. Furthermore, when VT is resistant to pacing or various antiarrhythmic agents, DC must be performed repetitively. However, repeated DC may cause functional and morphological changes of the myocardium. Thus, to treat recurrent life-threatening VT in a patient with heart failure, an alternative method is required.

ECLS, utilizing a pump and an external oxygenator, is one of the most effective methods of supporting the failing heart and lung. Veno-arterial bypass provides exact hemodynamic support and improves oxygen delivery to the main organs, including the brain, the heart, the liver and the kidney. Improvement of hemodynamics by the use of ECLS following IABP may eliminate certain causes of VT, such as myocardial ischemia, hypoxia, metabolic acidosis, or sympathetic stimulation! This may explain the temporary disappearance of ventricular arrhythmias in our patient during the combined use of ECLS and IABP.

The therapeutic efficacy of propranolol has been well established in patients with VT. However, propranolol may cause congestive heart failure in patients with impaired myocardial function. Thus, propranolol should be administered with great care in patients with left ventricular failure.

The myocardial depressant effect of propranolol is dose-dependent and the usual therapeutic serum level is 25 to 150 ng/ml. In the present case, an extraordinary dose of propranolol was required to suppress the ventricular arrhythmias, which resulted in a very high serum concentration! However, despite this large dose of propranolol, the patient's hemodynamics stabilized without congestive heart failure after the complete suppression of VT along with the combined use of ECLS and IABP. The hemodynamic improvement was probably due to improvement in the effect of IABP and ECLS after the suppression of frequent ventricular tachyarrhythmias by the propranolol infusion, as well as to improvement of left ventricular dysfunction by the aneurysmectomy procedure.

It has been demonstrated that electrical instability of the heart can be induced by stimulating the stellate ganglia and that reflex reduction of sympathetic activity decreases cardiac vulnerability. Thus, the frequent episodes of life-threatening ventricular tachyarrhythmia in our patient may have been due to increased ventricular electrical instability related to ventricular depression and cardiac damage just after the cardiac surgeries.

In summary, we have described a patient with refractory VT and heart failure following ventricular aneurysmectomy and cryosurgery who was successfully treated by a continuous high-dose infusion of propranolol combined with the use of ECLS and IABP. ECLS plus IABP enabled us to use high-dose propranolol despite the existence of postoperative heart failure, and the combined use of ECLS and IABP before irreversible myocardial damage contributed to successful outcome.

REFERENCES

8. HUIKURI HV, COX M, INTERIAN A, KESLER KM, GLICKSMAN F, CASTELLANOS A, MYERBURG RJ: Efficacy of intravenous propranolol for suppression of inducibility of ventricular
tachyarrhythmias with different electrophysiologic characteristics in coronary artery disease. *Am J Cardiol* 1989; 64: 1305–1309


