Diltiazem for Ventricular Fibrillation

To the Editor — Diltiazem, a calcium blocker, suppresses ventricular fibrillation induced by myocardial ischemia and improves resusci-
tability in animal experiments. Inhibition of the Ca** influx into
the myocardial cell, reduction of the heart rate, and enhancement
of the electrical stability may contribute to the improved resusci-
tation rate by diltiazem. Despite these encouraging results in animal
experiments, there has been no clinical evaluation of the effects
of diltiazem on ventricular fibrillation as of yet. We report a case of
successful resuscitation by diltiazem from ventricular fibrillation,
which resulted from acute myocardial infarction and was refractory
to standard therapy.

The patient was a 75-year-old man. He had a percutaneous translum- nal coronary recanalization for acute myocardial infarction of the
right coronary artery 2 yr ago from this April, but he has been well
since then. On admission to the emergency room because of chest
pain in this May, his consciousness was clear but ECG showed a
complete AV block and ST segment elevations at II, III, and aVF leads.
The patient suddenly developed ventricular fibrillation. After the
endotracheal intubation and the initiation of external chest compression
of cardiac massage, defibrillation was sequentially attempted at 5 times
with the energy of 200 J. Each defibrillation resulted in transient
sinus tachycardia (heart rate was 100–120 beats/min), which lasted
only for less than 1 min and ventricular fibrillation recurred (fig. 1).
Because of tachycardia, epinephrine was withheld. Lidocaine, 50–
100 mg, was administered intravenously before the last three defibri-
lation attempts (total dose of lidocaine was 200 mg). Then, diltiazem,
5 mg, was administered intravenously before the sixth defibrillation
at 200 J. Ventricular fibrillation disappeared immediately after de-

fibrillation (sinus rhythm with AV block, fig. 2). He recovered without
any complications after the treatment with temporal ventricular pacing
and tissue-type plasminogen activator. Although administration of
diltiazem to a patient in complete AV block may be controversial,
we administered diltiazem to a patient who was already in ventricular
fibrillation. It is assumed that restoration of any type of sinus rhythm
is of paramount importance. Although restoration of sinus rhythm
might have been due to the doses of lidocaine, we assume that
successful resuscitation was due to diltiazem because of the previous
failure of the lidocaine alone. The dramatic response to diltiazem
after unsuccessful efforts with lidocaine and defibrillation suggests
that diltiazem might be worth trying in resuscitation from recurrent
ventricular fibrillation.

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