Transcoronary Chemical Ablation of Atrioventricular Conduction

Pedro Brugada, MD, Hans de Swart, MD, Joep Smeets, MD, and Hein J.J. Wellens, MD

In seven patients with symptomatic atrial fibrillation and uncontrollable ventricular rates, selective catheterization of the atrioventricular (AV) nodal artery was performed to chemically destroy the AV node. Ethanol at a concentration of 96% and a dose of 0.5–2 ml was used after selective catheterization of the AV nodal artery had demonstrated temporary AV block after the administration of isotonic iced saline. Complete AV block was produced in five patients and AV conduction was sufficiently modified to control symptoms in the remaining two patients. A minimal enzyme rise occurred in six patients. A severe complication in the remaining patient occurred when, after 2 ml ethanol in the AV nodal artery, occlusion developed in the midright coronary artery that led to an inferior wall myocardial infarction. It is concluded that the AV nodal artery can be selectively catheterized using presently available angioplasty techniques. Ethanol can be used to destroy the AV node and block AV conduction. (Circulation 1990;81:757–761)

We have recently shown that it is possible to identify and selectively catheterize a small coronary artery providing blood supply to an arrhythmogenic area or pathway.1–2 Using this technique, we have successfully ablated, with 96% ethanol, ventricular tachycardia in patients with incessant ventricular tachycardia after myocardial infarction.2 In our previous report,1 we had selectively catheterized the atrioventricular (AV) nodal artery in one patient having circus-movement tachycardia using the AV node–His pathway and an accessory AV pathway. Administration of iced saline resulted in termination of the arrhythmia by block in AV nodal conduction.

The AV nodal artery arises in most humans from the right coronary artery and is a readily identifiable vessel.3 Electrical ablation of the AV conduction system has been successfully used for several years to control ventricular rates in patients with atrial fibrillation not controllable with antiarrhythmic drugs.4–5 In some patients, however, electrical ablation of the AV conduction system can fail to create AV block or sufficiently modify AV conduction. Using the same techniques as previously described,1–2 we undertook chemical ablation of AV conduction in seven patients with symptomatic uncontrollable ventricular rates during atrial fibrillation. The feasibility of this procedure, but also the occurrence of a severe complication in one patient, are the reasons for this report.

Methods

Seven patients were studied. Their clinical characteristics are summarized in Table 1. All patients had failed multiple antiarrhythmic drug trials to control ventricular rates during atrial fibrillation. In four patients, three to seven attempts of electrical ablation of the AV system were performed without lasting success. Three patients had a permanent pacemaker implanted. Possible damage to the pacemaker by renewed attempts of electrical ablation was an additional reason to undertake chemical ablation in these patients. The last two patients gave preference to chemical ablation above electrical ablation or surgery. The last patient had undergone successful surgical ablation of an accessory pathway. All patients had normal coronary arteries and were free from any structural heart disease. The experimental nature of this procedure was explained to all patients, and they gave informed consent. The protocol was approved by the Medical Ethical Committee of our University.

Coronary angiography using the Judkins technique was first performed in all patients. All seven had normal coronary arteries. The AV nodal artery originated from the right coronary artery in all patients except in patient 3 in whom it originated from a dominant circumflex coronary artery. The AV nodal
artery was a discrete single vessel in five patients but
 two branches next to each other were observed in
 patients 2 and 3. Selective catheterization of the AV
 nodal artery was performed using previously
 described methodology.1–2 In summary, a 0.014-in.
 angioplasty guide wire was advanced through an 8F
 guiding catheter as far as possible into the AV nodal
 artery. Thereafter, a 2.2–4.0F lumen catheter with a
 metallic marker at the tip was advanced over the
 guide wire as far as possible into the AV nodal artery.
 The guide wire was, thereafter, removed. Contrast
 material was given to confirm catheterization of the
 vessel identified as the AV nodal artery. During the
 procedure, an electrode catheter was inserted in the
 right ventricular apex and connected to an on-
demand external pacemaker in all patients. Nitro-
glycerin was continuously infused intravenously dur-
ing the procedure. Heparin was also given at a dose
 of 1 mg/kg body wt (equivalent to 100 units heparin
 per kilogram body weight). Confirmation of selective
 catheterization of the AV nodal artery was done by
 observing the occurrence of transient AV block on
 selective catheterization of the artery, administration
 of contrast material, and administration of iced
 saline. An example is shown in Figure 1.

 After confirmation of the identification of the AV
 nodal artery, 96% ethanol was given at a dose of
 0.5–1 ml in 2–4 seconds except in patient 4, who
 received a total of 3 ml in 2 sessions. The reasons
 for the larger dose in the last patient will be discussed
 later.

 After AV block was created, catheters with the
 exception of the pacing lead were removed, and the
 patient was transferred to the coronary care unit for
 48 hours. A permanent pacemaker was implanted
 2–3 days later when required.

 Results

 Transient complete AV block occurred on cathe-
terization of the AV nodal artery in all patients.
 Conduction recovered after a few seconds to minutes
 in all patients, although ventricular rates remained
 below the initial heart rates when the catheter was
 maintained in place. Iced saline and contrast mate-
rial (Figure 1) resulted in transient complete AV
 block in all patients.

 Administration of 96% ethanol led to complete
 AV block in all patients. All patients showed ventricu-
 lar extrasystoles before the development of AV
 block as illustrated in Figure 1. AV block remained
 complete in patients 1, 2, 4, 6, and 7, after follow-up
 of 1–7 months (mean, 4 months). In patients 3 and 5,
 conduction recovered within 24 hours but the ven-
tricular rate during atrial fibrillation decreased from
 a mean of 170 to 100 beats/min and from 160 to 80
 beats/min, respectively, with pronounced symptom-
atric improvement after 2 and 5 months’ follow-up.

 Complications and Course of Events in Patient 4

 As previously described,2 all patients complained
 of short-lasting (a few seconds) chest pain during
 administration of ethanol. The oxalacetyl transami-
nase rose to 85, 96, 65, 37, 29, and 220 units (normal
 at our laboratory, <40 units) in patients 1, 2, 3, 5, 6,
 and 7, respectively. No other complications occurred.
 Echocardiograms were normal in all patients except
 patient 4, before and after ablation. Because of the
 severe complication occurring in patient 4, the course
 of events in this patient will be described in detail.

 Selective catheterization of the AV nodal artery
 was performed without any particular problem in this
 patient. Administration of iced saline and contrast
 material resulted in transient complete AV block, as
 in the other patients. A dose of 1 ml ethanol was
 given, and complete AV block was created. The
 patient was transferred to the coronary care unit.
 Twelve hours later, AV conduction resumed with the
 same ventricular rates as before the procedure.
 Selective catheterization of the same AV nodal artery
 was repeated 1 day later using the same approach.
 Complete AV block was again easily produced by
 administering iced saline or contrast material.
 Because of the experience during the first attempt, a
 dose of 2 ml 96% ethanol was given in the AV nodal
 artery. Complete AV block occurred, associated with
 chest pain. Chest pain, however, persisted and was
 accompanied by marked ST segment elevation in the
 inferior leads during pacemaker rhythm. An angiog-
 raphy of the right coronary artery showed that the
 contrast material stopped in the proximal part of the
 right coronary artery (Figure 2) but, farther on, the
 artery was patent as shown by injection of contrast
 material more distally using a perfusion catheter (Fig-
 ure 2). The vessels distal to the main right coronary
 artery could not be visualized (Figure 2), suggesting
 occlusion of the peripheral branches. Retrospective
 analysis of the data suggested the following sequence
 of events: At the time of the first procedure, no chest
 pain developed during administration of ethanol, no
 enzyme rise occurred, and complete AV block did not
 persist. This suggests that, for whatever reason, the
 alcohol did not reach the distal end of the selective
 catheter. During the second procedure, a dose of
 ethanol was given that in retrospect was probably too
 large. This was followed by the development of an

| Table 1. Clinical Characteristics of the Patients Studied |
|-----------------|-----------------|-----------------|
| Patient | Age/sex | Indication for TCA-AVC |
| 1 | 70/F | Failed electrical ablation |
| 2 | 64/F | Failed electrical ablation |
| 3 | 64/M | Failed electrical ablation, permanent pacemaker |
| 4 | 69/F | Permanent pacemaker |
| 5 | 60/M | Failed electrical ablation, permanent pacemaker |
| 6 | 69/M | Patient preference |
| 7 | 54/M | Previous arrhythmia surgery, patient preference |

F, female; M, male; TCA-AVC, transcoronary chemical ablation of atrioventricular conduction.
Figure 1. Panel A shows right coronary angiogram in left anterior oblique projection. Atrioventricular nodal artery is indicated by arrow. Panel B shows selective catheterization of atrioventricular nodal artery with 2.5F catheter (arrow). Contrast is given showing myocardial staining and no backflow. Panel C shows effects of contrast material on ventricular rate during atrial fibrillation. Transient complete atrioventricular block and pacemaker rhythm is observed in right part of electrocardiographic strip. Leads I, II, and III are shown. Panel D shows effects of administration of 0.5 ml 96% ethanol. After series of premature ventricular beats, complete atrioventricular block and pacemaker rhythm occur. Panel E shows escape rhythm after interruption of pacing.
inferior wall myocardial infarction with a maximal oxalacetyl transaminase rise to 363 units. The left ventricular ejection fraction was 50%. The right ventricular ejection fraction was 25%, indicating that infarction of the right ventricle also occurred. One week later, this patient's course was complicated by late ventricular fibrillation from which she was successfully resuscitated. Treatment with amiodarone was started, and the patient discharged in complete AV block 2 weeks later. At 5 months' follow-up, the patient remains in complete AV block, and her functional class for dyspnea is I.

Discussion

The results of this study indicate that transcoronary chemical ablation of the AV conduction system can be done successfully; however, the complications occurring in patient 4 warn us about the possible risks of this new technique. Although the procedure was uneventful and successful in six patients, the complications in patient 4 indicate the dangers of manipulation of the coronary arteries and the dangers of a too-large volume of ethanol. Transcoronary chemical ablation with ethanol of the AV node can be done uneventfully. To avoid complications like the ones observed in patient 4, however, several precautions seem necessary. The catheter should be fully wedged in the artery to prevent any backflow. Catheters of different diameters and with a distally located balloon might be helpful for that purpose.

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