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A Case of Normalization of Wolff-Parkinson-White Syndrome Conduction during Propofol Anesthesia

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THE effects of intravenous[1,2] and volatile[3] anesthetics on the refractoriness of the accessory pathway in patients with Wolff-Parkinson-White (WPW) syndrome have been examined. Although propofol has not been reported to have any direct effect on the accessory pathway,[1] we report a patient with WPW syndrome whose electrocardiogram (ECG) was normalized during propofol infusion.

Case Report

A 29-yr-old woman was scheduled for dilation and curettage resulting from a missed abortion. She had experienced at least one attack of tachyarrhythmias per year during the preceding 6 yr and had been prescribed 10 mg oral propranolol for the attacks. The nature of the tachyarrhythmias was unknown. Her preoperative 12-lead ECG showed delta waves and short PR intervals in all leads. Because of appearance of a high R wave in lead V_1, she was diagnosed with Wolff-Parkinson-White (WPW) syndrome type A (fig. 1).

On the day of surgery, she received midazolam, 4 mg, intramuscularly 30 min before induction of anesthesia. On arrival to the operating room, an 18-gauge catheter was inserted in the forearm. ECG (lead II), blood pressure, and pulse oximetry were monitored continuously. Her preoperative blood pressure was 140/75 mmHg and heart rate was 85 beats/min. The ECG showed a delta wave and a short PR interval (0.08 s; fig. 2A). Anesthesia was induced with 1 mg/kg propofol intravenously after 0.1 mg fentanyl and maintained with propofol infusion of 6 $mg \cdot kg^{-1} \cdot h^{-1}$. Ventilation was assisted *via* a facemask using a mixture of oxygen and air ($F_{I_{O_2}}$, 0.4). One minute after the propofol injection, the delta wave disappeared, and the PR interval was normalized (0.2 s; fig. 2B). At this time, her blood pressure was 105/52 mmHg and heart rate was 86 beats/min. The surgery was started after the normalization of QRS complex. Nitrous oxide was added when she moved during operation. After curettage, 0.2 mg of ergometrine maleate was administrated intravenously, which did not affect the ECG. Hemodynamics were stable throughout the operative procedure with her blood pressure ranging between 105/52 and 118/65 mmHg and heart rate between 68 and 78 beats/min. The operation lasted 25 min. The normal ECG conduction remained until the end of anesthesia (fig. 2C). However, 5 min after the discontinuation of propofol, the delta wave returned suddenly (fig. 2D). The patient was discharged 6 h after the surgery. Her postoperative ECG did not show repeat normal conduction. There were no tachyarrhythmias throughout her hospital stay.

Discussion

In a typical WPW syndrome, the delta wave is the expression of preexcitation caused by an accessory pathway conduction, whereas the remaining normally inscribed QRS complex is the expression of normal atrioventricular conduction.[4] Consequently, there are two possible explanations for the delta wave disappearance in this case; decreased conduction time through the normal atrioventricular pathway or increased conduction time through the accessory pathway.

Decreased conduction time through the normal atrioventricular pathway is not a consideration in this patient because the PR interval during propofol infusion was prolonged. When the enhanced atrioventricular nodal conduction is responsible rather than true loss of preexcitation, QRS complex should have been normalized without PR prolongation.[5]

As discussed previously, depression of conduction through the accessory pathway was responsible for normalization of the QRS complex (*e.g.*, loss of delta wave) during propofol anesthesia. Propofol seems most likely

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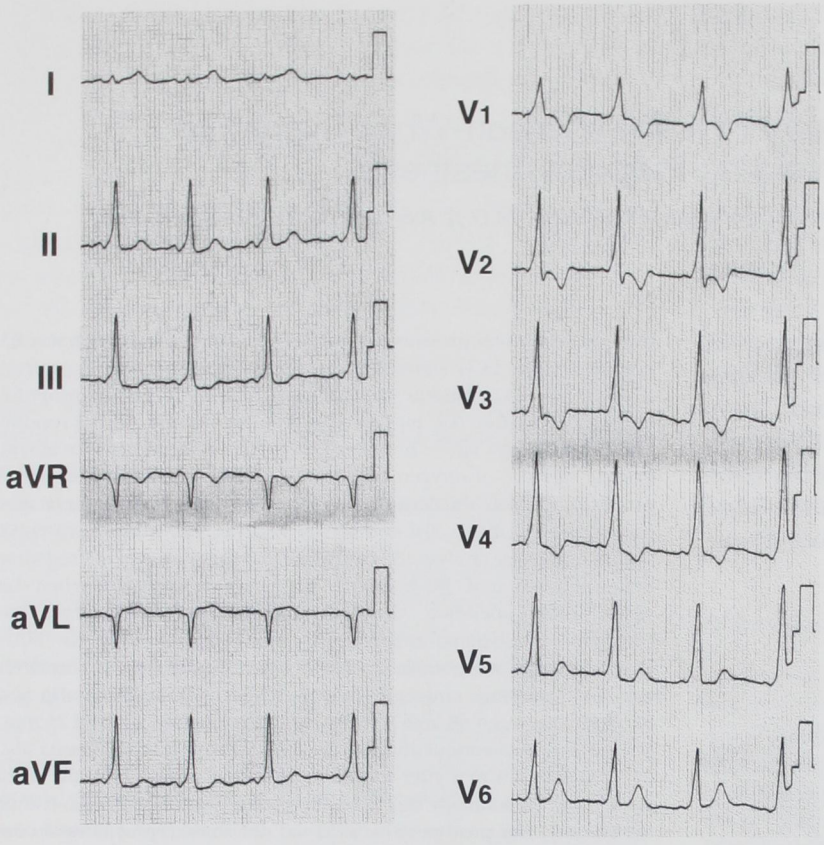

Fig. 1. Preoperative 12-lead electrocardiogram tracing showing Wolff-Parkinson-White syndrome type A.

to be responsible for the normalization of the QRS complex because the delta wave disappeared immediately after propofol induction and returned 5 min after discontinuation of propofol. However, whether this was a result of a direct effect of propofol on accessory pathway conduction cannot be determined in this case. This determination would require an electrophysiologic study with application of propofol to determine its effect. Further, propofol has been shown not to have any significant effect on accessory pathway conduction in those with WPW syndrome during alfentanil and midazolam anesthesia.[1] This finding suggests that depression of accessory pathway conduction in this case was the result of the natural physiologic expression of this patient's particular pathway (*e.g.*, intermittent preexcitation). It has been reported that a patient with intermittent loss of delta wave has a longer refractory period in the accessory pathway and poorer conduction over the pathway than one with constant preexcitation.[6] Our patient, therefore, might have the accessory pathway of poorer conduction, so that it was more susceptible to be depressed. We could not have estimated the conduction of the accessory and normal pathways using electrophysiologic technique before surgery because of the emergency nature of her operation. In addition, there is a limitation to evaluation of QRS normalization with a single lead. It is possible that only partial leads of ECG may show apparent QRS normalization.[6]

Previous work has indicated that propofol has no effects on the refractory period of accessory pathway[1] and therefore is an appropriate anesthetic agent to use during ablative procedure. On the other hand, for patients with accessory pathways requiring general anesthesia for nonablative procedures, the anesthetics that increase refractory period of the accessory pathway should be of choice because most common tachyarrhythmia in WPW syndrome is reentrant tachycardia using accessory pathway for one limb of reentry circuit.[7] For example, droperidol, which increases refractory period of the accessory pathway, may prevent reentrant tachyarrhythmia.[2] Enflurane also is the agent of choice to suppress intraoperative tachyarrhythmias because it not only increases the refractoriness of the accessory pathway, but it also has no effect on the coupling interval.[3] Nevertheless, we chose propofol for this patient instead of enflurane because enflurane may stimulate the sympathetic

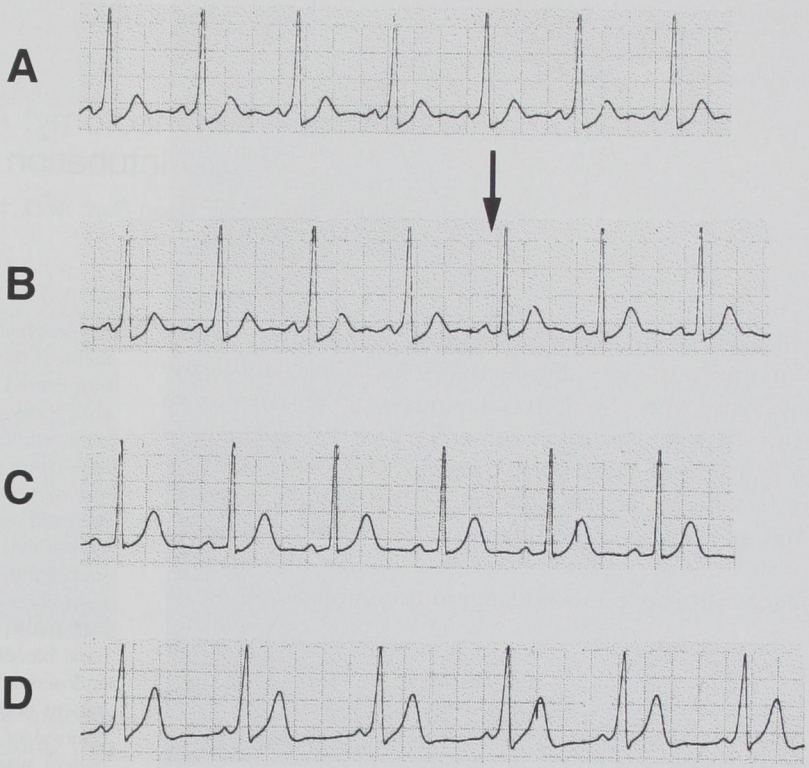

Fig. 2. Lead II electrocardiogram tracing obtained before induction of anesthesia (*A*), 1 min after the propofol injection (*B*), during operation (*C*), and 5 min after the discontinuation of propofol (*D*). Arrow indicates sudden normalization of QRS complex and PR interval after induction of anesthesia with propofol.

nervous system when inhaled through a facemask.[8] Further, the patients given propofol showed better recovery and less incidence of postoperative nausea and vomiting than those given enflurane in day-case gynecologic surgery.[9]

In conclusion, we encountered a patient with WPW syndrome whose delta wave disappeared during propofol infusion, although propofol has been shown not to have any significant effect on accessory pathway conduction in WPW syndrome.

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