

TXA₂ receptor-binding affinity at the 2 different concentrations. Consequently, 40 µM propofol might affect the TXA₂ pathway through enhancing the activity of the Gq protein or PLCβ. (Fig. 1).

In summary, volatile anesthetics such as halothane and sevoflurane have been found to inhibit platelet aggregation. Enflurane, isoflurane, and desflurane appear to have minimal or negligible effects on platelets. Barbiturates, benzodiazepines, and etomidate do not seem to affect platelet function. One intravenous anesthetic, propofol, has dual effects on platelet aggregation. High concentrations of propofol suppress platelet aggregation, but low concentrations of propofol enhance platelet aggregation.

These anesthetics appear to have similar effects on platelet aggregation and bleeding time just like other antiplatelet drugs. Therefore it is possible that these anesthetics may affect the incidence of intraoperative bleeding or thromboembolic complications in a similar manner to other antiplatelet drugs. However, the clinical significance of the inhibitory effect of these anesthetics on platelet function in vivo awaits further investigations.

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