Anesthetic Modulation of Cancer Cell Biology: Convergent Roles of Lipid Rafts and Voltage-Gated Sodium Channels (A Narrative Review)

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Introduction. Anesthetic agents traditionally are recognized for reversibly suppressing neuronal excitability through ion channel modulation and membrane interactions. Emerging evidence suggests these agents also may influence cancer cell biology. Malignant cells exhibit increased cholesterol-rich lipid rafts and aberrant expression of voltage-gated sodium (NaV) channels, particularly neonatal splice variants, which together promote oncogenic signaling, invasion, metabolic reprogramming, and metastasis. Because anesthetics directly interact with both lipid membranes and NaV channels, they may modulate cancer-specific vulnerabilities.

Methods. Authors conducted a narrative using PubMed to identify experimental and clinical studies evaluating the effects of local, volatile, and intravenous anesthetics on lipid raft organization, NaV function, and cancer biology. Key search terms included “anesthetics,” “lipid rafts,” “voltage-gated sodium channels,” and “cancer.” Relevant mechanistic and preclinical data were synthesized.

Results. Preclinical evidence demonstrates that local anesthetics inhibit NaV activity, reduce persistent sodium influx, impair cytoskeletal remodeling, suppress extracellular acidification, and decrease cancer cell migration and invasion. They also induce mitochondrial dysfunction, apoptosis, autophagy, and immunogenic cell death. In contrast, volatile anesthetics often activate PI3K/Akt/mTOR and hypoxia-inducible factor pathways while suppressing anti-tumor immunity. Intravenous agents such as propofol exhibit context-dependent effects, with studies demonstrating both anti-proliferative and pro-migratory outcomes.

Conclusions. Anesthetics exert convergent effects on membrane lipid organization and NaV signaling, two interconnected regulators of malignant behavior. Local anesthetics demonstrate the most consistent anti-tumor profile, whereas volatile agents may promote survival signaling under certain conditions. Although clinical data remain largely neutral, these mechanistic insights provide a biologically grounded framework for interpreting perioperative oncologic outcomes and for designing anesthetic strategies that minimize tumor-promoting effects.