Best Practice & Research Clinical Anaesthesiology
Vol. 19, No. 3, pp. 485–500, 2005
doi:10.1016/j.bpa.2005.03.001
available online at http://www.sciencedirect.com

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PONV: A problem of inhalational anaesthesia?

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Even nowadays every third or fourth patient suffers from postoperative nausea and vomiting (PONV) after general anaesthesia with volatile anaesthetics. There is now strong evidence that volatile anaesthetics are emetogenic and that there are no meaningful differences between halothane, enflurane, isoflurane, sevoflurane, and desflurane in this respect. However, when propofol is substituted for volatile anaesthetics the risk for PONV is reduced by only about one fifth, indicating that there are other even more important causes for PONV following general anaesthesia. A main causative factor might be the use of perioperative opioids, but their impact—relative to other factors including volatile anaesthetics—has never been quantified. Patient-specific risk factors have also been shown to be clinically relevant; they are therefore included in the calculation of simplified risk scores that allow prediction of a patient's risk independent of the type of surgery. Although controversial, the well-known different incidences following certain types of surgery are most likely caused by patient-specific and anaesthesia-related risk factors. There is a common consensus that prophylaxis with anti-emetic strategies is rarely justified when the risk of PONV is low, while it is warranted in case of imminent medical risk associated with vomiting or in a patient with a high risk for PONV. A recently published large multicentre trial of factorial design, IMPACT, has demonstrated that various anti-emetic strategies are associated with a very similar and constant relative reduction rate of about 25–30% and that the main predictor for the efficacy of prophylaxis is the patient's risk for PONV. Interestingly, all anti-emetics (dexamethasone, droperidol and ondansetron) work independently, so that their combined benefit can be derived directly from the single effects. The effectiveness of the anti-emetics was also independent of a variety of risk factors, including volatile anaesthetics.

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1521-6896/$ - see front matter

This means that any anti-emetic prophylaxis for PONV induced by volatile anaesthetics is equally effective. Of course, the most logical approach for prevention would be the omission of volatile anaesthetics and nitrous oxide using a total intravenous anaesthesia with propofol. However, since volatile anaesthetics are probably not the most important risk factors, it might be even better—if appropriate—to avoid general anaesthesia by using a regional, opioid-free anaesthesia if PONV is a serious problem.

Key words: volatile anaesthetics; postoperative nausea and vomiting; pathophysiology; risk factors; predictive models.

Approximately 75 million operations are performed annually throughout the world, mainly with the patient under general anaesthesia with volatile anaesthetics. Unfortunately, every third patient suffers from mild to severe postoperative nausea, vomiting, or both (PONV).[1–3] Furthermore, PONV may be a more frequent reason for patient discomfort than postoperative pain.[4,5] It is not surprising, therefore, that preventing PONV improves satisfaction among patients likely to experience it.[6] Nausea is uncomfortable for the patient, and vomiting increases the risk of aspiration and has been associated with suture dehiscence, oesophageal rupture, subcutaneous emphysema, and bilateral pneumothoraxes.[7,8] PONV frequently delays discharge from post-anaesthesia care units and is the leading cause of unexpected hospital readmission after planned ambulatory surgery.[9] The annual cost of PONV in the United States is thought to approach a billion dollars.[10,11]

Numerous factors influence whether a patient develops PONV.[12–16] However, a critical review of the literature reveals that strong evidence, which comes from prospectively designed surveys with multivariable analysis, is only available for a few risk factors.[4,17]

The trigger for the complex act of vomiting after general anaesthesia is still not understood completely.[18,19] Those mechanisms that are well studied and well known are based on vomiting induced by apomorphine, cisplatin or radiation in animals. The lack of an adequate experimental animal model makes transfer of these known mechanisms to human beings difficult.[18] Although there are some studies on volunteers investigating the effects of hyperbaric nitrous oxide[20,21], haloperidol[22], ondansetron[23], propofol or midazolam[24], there are very few studies about the actual pathological mechanisms of PONV in humans.

Despite the enormous number of clinical trials, the incidence of PONV varies from 10 up to 80% in very special circumstances, the variation being due largely to differences in individual and anaesthetic risk factors.

This review seeks to describe the role of volatile anaesthetics in causing PONV. To this end, it will review the physiology of PONV (as far as it is understood), describe the risk factors known to be associated with PONV, and put the causative role of volatile anaesthetics into perspective. Finally, predictive models based on these factors will be discussed along with anti-emetic strategies to prevent or treat PONV.

PHYSIOLOGY OF PONV

The pathophysiology of vomiting is complex, and several organs are involved.[18] The bilateral vomiting centre is located in the medulla oblongata and lies near the tractus solitarius at the level of the dorsal motor nucleus of the vagus.[25] Afferents from