Review Article

Anaesthesia and emesis. I: etiology

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One of the most distressing side effects following anaesthesia is the occurrence of nausea, often followed by retching and vomiting.

In a review of the subject in 1963 Riding felt that postoperative "sickness" was less frequent and severe than in the preceding twenty years.¹ However, examination of more recent literature suggests that the incidence of postoperative "sickness" has probably not changed although its severity may have. It is, however, difficult to compare these studies as there was little attempt by many authors to define nausea, retching, or vomiting, indeed postoperative "sickness" is often used interchangeably with these terms. The studies variably included one or all of these symptoms; population samples, anaesthetic conditions, and periods of follow up also differed (Table I).

This article will review our current understanding of the physiology of emesis (nausea, retching and vomiting) and evaluates the part played by anaesthesia in its aetiology. In a second review we will examine the methods and agents available to anaesthetists for prevention or treatment of emetic symptoms. Clearly there are several factors not directly related to anaesthesia that influence the incidence of emetic episodes (nausea, retching, or vomiting). These factors include metabolic, vestibular and psychogenic disturbances, gastro-intestinal and intracranial disorders and administration of agents with emetic potential such as ergometrine.

In these cases there is often little the anaesthetist

can do to influence the primary pathology and so brief mention will be made of these.

Physiology

The current concept of the mechanisms of vomiting is largely based on the work of Borison and Wang.² They defined vomiting as the forceful expulsion of gastrointestinal contents through the mouth; retching as the rhythmic activity of respiratory muscles usually preceding vomiting and nausea as a psychic experience which may or may not be associated with vomiting.

Emetic episodes are the response of the vomiting centre to stimuli arising from several areas; these include autonomic afferents from the gastrointestinal tract (GIT), and mediastinum, the vestibular component of the eighth cranial nerve, visual and cortical stimuli and an input from the chemoreceptor trigger zone (CRTZ). The vomiting centre lies in the dorsal part of the lateral reticular formation in the medulla. It is close to the tractus solitarius at the level of the dorsal motor nucleus of the tenth cranial nerve. The nuclei associated with vasomotor activity, salivation, respiration, and bulbar control are also close by. This proximity is consistent with the physiological reactions associated with vomiting. Stimulation of the vomiting centre results in reflex efferents travelling in the 5th, 7th, 9th, 10th and 12th cranial nerves, efferents also reach the diaphragm and abdominal muscles from the spinal nerves.³

The CRTZ is closely associated with the vomiting centre. It is a bilateral area located on the floor of the fourth ventricle above the area postrema. The CRTZ has a high density of dopaminergic receptors which are mainly stimulated by drugs and metabolic disturbances; impulses from the CRTZ are then relayed to the vomiting centre and a reflex initiated. Recently an antiemetic centre has also been proposed, the work relating to this was performed in

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TABLE I Selected studies on the incidence of emetic sequelae following anaesthesia

Investigator	Population size	Incidence (%)	Follow up in hours	NVR	Year
1. Waters, R.M. ²⁵	10,000	40.6	N/A	NV	1936
2. Dent, S. ³⁸	3,000†	27.2	24	VR	1955
3. Burtles & Peckett ⁹	1,702†	32	24-36	NV	1957
4. Bonica, J. ⁴⁰	1,561†	30.5	24	NVR	1958
5. Bellville, J.W. ¹⁰	748†	19.4	2.5	NV	1959
6. Adriani, J. ⁴⁸	2,230†	23	avg. 6	RV	1961
7. McKie, B.D. ⁴⁹	110†*	30.9	24	NV	1969
8. Korttila, K. ³⁰	40†*	55	24	NVR	1979
9. Mortensen, P.T. ²⁹	96†*	34.4	24	NVR	1982

N: Nausea; V: Vomiting; R: Retching; NA: Not available.

*Females only.

†Prospective study.

the cat which is a very good model for vomiting in the human.⁴ Figure 1 summarises the inputs to these centres. There are many consequences following vomiting that may concern the attending physician. Mild vomiting does not usually lead to electrolytic disorder, but is distressing to patients. Severe vomiting, however, may lead to hypokalaemic, hypochlorhaemic, hyponatraemic alkalosis and dehydration, but this is extremely uncommon after anaesthesia, as is a Mallory-Weiss tear or oesophageal rupture (Boerhaave's syndrome). However, in the presence of a depressed level of consciousness, or after oral surgery when the jaw is wired, aspiration of vomitus is not uncommon and carries a significant mortality and

morbidity. Wound disruption is a further concern, particularly following eye, abdominal or plastic surgery. In the case of the latter the associated rise in venous pressures in the head, neck and upper thorax leads to a significantly increased incidence of bleeding beneath skin flaps.⁵

Factors that may influence the incidence of emetic sequelae relating to anaesthesia have been grouped as those determined by the patient, by the anaesthetic, and the postoperative care.

Patient factors

Individual predisposition

This is a most difficult area in which to obtain conclusive data. There is no doubt that among the

FIGURE 1 Sites of action of stimuli related to anaesthesia.

general population there is a percentage that will suffer emetic symptoms at the slightest exposure to provocative stimuli. This has been considered to be psychological in origin. However, the authors are not wholly in favour with this view. We are more of the opinion that the vomiting reflex is variably developed. For example, it is well known by dentists that there is wide variability in the eliciting of a gag response with subsequent nausea and retching, suggesting a variation in threshold for emetic stimuli. This will certainly influence the number of patients that vomit on emergence from anaesthesia when a Guedel airway is in place.

Similarly, only some subjects may experience a feeling of nausea after a period of fasting during waking hours. We have recently conducted a small survey among 40 healthy adults between the ages of 20 and 50 years. Among the 20 females in this group, 56 per cent experienced nausea after an average period of fasting for seven hours. However among the 20 males only 38 per cent experienced nausea after an average period of 8½ hours. Patients who are prone to suffer with motion sickness also seem to experience more postoperative emetic problems.⁶ Furthermore, it is interesting to note that just as the susceptibility to motion sickness is high in young children and decreases with reaching adulthood, the same trend is seen in postoperative emesis.^{6,7} If patients with a low threshold for vomiting are predisposed to postoperative emetic sequelae, it is not then surprising that Purkis observed a threefold increased chance of vomiting in the first 24 hours among patients who had suffered previous postoperative vomiting.⁶

Sex

It is commonly stated that emetic symptoms are up to three times more common among females.⁸⁻¹¹ Dyrberg, on the other hand, found little difference between the sexes following extra-abdominal procedures, but females did have a higher incidence after abdominal surgery.¹² Bellville suggested that the increased incidence of nausea and vomiting among women was related to elevated gonadotrophin and progesterone plasma concentrations.¹³ This observation is possibly supported by the observation that emetic sequelae are seen in equal numbers among male and female children.¹⁴

Since most of the recent studies concerning postoperative vomiting have been conducted ex-

clusively among women, it is difficult to confirm the findings of earlier studies. However, a prospective study concerned with the incidence of nausea and vomiting in a plastic surgical unit in 1973 found that among adults having operations not involving the ear, a significantly higher number of females suffered from nausea and vomiting.¹⁴ This would confirm nearly all the observations made to date, that emetic episodes are two to three times more common among females.

Age

There is a correlation between increasing age and decreasing emesis as mentioned earlier. Children have about twice the adult incidence of postoperative nausea and vomiting.¹⁴ This is of some concern as children can develop dehydration more readily than adults. In a prospective study among children by Rowley and Brown there was a decreased incidence of vomiting below the age of three when observed for up to 18 hours after surgery.¹⁵ However, this apparent decrease may in part be due to different premedication in this group. In adults Burtles and Peckett⁹ and Bellville¹⁰ found a decrease in emetic symptoms with increasing age whereas Knapp and Beecher found a decrease only in the over 70's, but their numbers in this group were small.¹¹ Howat found no correlation between emesis and age.¹⁶ It would be interesting to see these studies repeated using current anaesthetic techniques, as elderly patients are now presenting more frequently for surgery than thirty years ago.

Weight

Obesity has been reported to increase postoperative vomiting possibly, it is reasoned, due to a longer desaturation period for anaesthetic agents.^{10,17} The correlation between weight, nausea, retching and vomiting were recently studied by McKenzie *et al.* among patients undergoing first trimester abortions.¹⁸ They found a higher percentage of patients with these emetic episodes in the heaviest group. The patients were under anaesthesia for no longer than a mean of 25 minutes. It is hard to believe that the difference in anaesthetic uptake between the patients in the light and heavy group during 25 minutes of anaesthesia accounts for the differing emetic sequelae. We feel that the current explanation for the increased incidence in emetic symptoms among the obese is not clear.

Concurrent illness

Patients presenting for surgery may often have nausea or be actively vomiting due to an underlying condition. Perhaps the most common is a patient with acute appendicitis or gastrointestinal obstruction. However any patient with a full stomach (including swallowed blood) is likely to vomit prior to anaesthesia. Some of the appropriate measures that may be undertaken in these circumstances will be considered in Part II of this review. A less obvious, but equally important surgical cause of emesis prior to anaesthesia is the semicomatose patient with rising intracranial pressure. The prevention of vomiting here acquires special significance if the resultant straining is not to exacerbate the underlying condition. Metabolic disturbances and toxicity due to drugs including alcohol are other common conditions that may warn the anaesthetist of possible vomiting pre- or postoperatively.

Anaesthetic considerations

Premedication

Premedication with morphine leading to an increase in the incidence of postoperative emetic symptoms is well known.¹⁹ Riding, in a study among women undergoing uterine curettage observed that only 22.4 per cent of them suffered from nausea, retching or vomiting when unpremedicated. However, when morphine was added those affected rose to 67 per cent. When atropine was combined with morphine as a premedicant the incidence of emetic symptoms then fell to 37 per cent suggesting that atropine had significant antiemetic properties. This was confirmed by the administration of atropine 0.6 mg alone which reduced the incidence from 22.5 per cent seen in unpremedicated patients to 11.5 per cent.¹⁹ Unfortunately trying to unravel the issue of which opiate premedicants cause most postoperative emetic problems is difficult.

In an early study Burtles and Peckett noted more emetic problems after morphine or papaveretum premedication than with meperidine;⁹ Didier *et al.*²⁰ and Bellville¹⁰ on the contrary found morphine was a less emetic agent than meperidine. Dundee *et al.* in a later study obtained a significantly increased incidence of emetic symptoms in the first six hours postoperatively when morphine and atropine were used in preference to meperidine and atropine as premedication.²¹ However, in their discussion they

acknowledged that 50 mg of meperidine and 10 mg of morphine were not equi-analgesic and so were not necessarily comparable. To add further confusion, Bellville in his review described both positive and negative correlations between meperidine administration and emesis, dependent on dosage.¹³ Meperidine given in doses of less than 1 mg per kg was found to be antiemetic (Fig. 2). Similar antiemetic effects have been noted when morphine is given after apomorphine induced vomiting.

In fact the antiemetic properties of opiates has led to the proposal of an antiemetic centre by Costello and Borison; enkephalins are thought to be the transmitters at this centre.⁴ Harris suggested that the emetic and antiemetic effects of opiates may be explained by the presence of different opiate receptors at these centres.²²

Alternatively, in order to accommodate Bellville's findings one must suppose that the emetic centre has more opiate receptors, so that emetic symptoms prevail at larger doses.¹³ The concurrent use of anticholinergic agents with opiates for premedication has been thoroughly studied by Clarke *et al.*, who concluded that hyoscine was preferable to atropine when combined with morphine for avoiding nausea and vomiting.²³ Recently Mirakhor and Dundee concluded that glycopyrrolate a possible alternative to hyoscine had no antiemetic properties which they reasoned was due to its inability to cross the blood brain barrier.²⁴ A fuller discussion of anticholinergic agents and their anti-emetic properties will follow in part II of this review.

Anaesthetic agents

Despite the lack of strict controls and methods in early studies, most authors agree that cyclopropane and diethyl ether were the most potent emetics among the inhalational agents available. Waters reported incidences for nausea and vomiting of 56.6 and 39 per cent for ether and cyclopropane respectively.²⁵ Burtles and Peckett observed incidences of nausea and vomiting of 33 and 24 per cent for ether and cyclopropane respectively.⁹ Bellville *et al.*, however, reported an incidence of only 16.1 per cent for ether and 27.4 per cent for cyclopropane.¹⁰ It is reassuring, then, that these agents are less commonly used today.

Jenkins and Lahay speculated that the higher incidence of emetic problems among patients re-

FIGURE 2 The relationship between dose of meperidine premedication and incidence of postoperative nausea and vomiting. From Bellville,¹³ with permission.

ceiving diethyl ether or cyclopropane was related to raised cerebral spinal fluid (CSF) concentrations of endogenous catecholamines.²⁶ In their study with cats, norepinephrine and epinephrine were injected through cannulae placed into the cerebral ventricles. They noted that a vomiting response followed the injection of agents with alpha agonist properties whether or not the agent also had beta agonist properties. Vomiting was not observed when predominantly beta agonists or alpha or beta antagonist agents were injected. We may speculate that nausea and vomiting associated with conditions that result in elevated catecholamine concentrations such as pain or hypotension may be the result of the same mechanism. Halothane, an agent that tends to act as an adrenergic antagonist, on the other hand, is thought to have both emetic and antiemetic effects, the latter in subanaesthetic doses.²⁷ Riding observed only a five per cent incidence of emetic episodes with halothane but 7.5 per cent and 35 per cent incidences for trichlorethylene and ether respectively.¹ Heal, however, noted a 40 per cent incidence of nausea retching and vomiting after halothane among women who had also received

meperidine and atropine premedication.²⁸ The apparent discrepancy between these studies is impossible to evaluate as Riding gives no details of his anaesthetic technique or patient selection. The newer inhalational agents, enflurane and isoflurane have not been compared to halothane in a prospective study specifically to evaluate emetic sequelae. Although inhalational agents contribute significantly to postoperative sickness, the avoidance of these agents has not resulted in a dramatic reduction of emetic symptoms.^{29,30} This is presumably due to the current use of potent narcotics.

An interesting study conducted by Janhunen and Tammisto attempted to compare the emetic sequelae that followed a relaxant-meperidine anaesthetic with those following a relaxant-halothane technique.³¹ Nausea retching and vomiting were scored and conditions were standardised. They found that there was no difference between the groups. Even more interesting was their finding that when meperidine was omitted from the relaxant technique leaving only nitrous oxide as the anaesthetic agent there was no decrease in the emetic sequelae.

However, the dose of meperidine administered in this study was $1 \text{ mg} \cdot \text{kg}^{-1}$, this is the dose above which Bellville suggests meperidine becomes an emetic. The new opiates now available such as fentanyl, alfentanil and sufentanil have not been subjected to controlled prospective trials designed to evaluate their influence on vomiting. Most studies to date concerning these drugs have concentrated on their haemodynamic and respiratory effects and any data concerning emesis has not been strictly comparable to earlier studies on the subject.

Nitrous oxide is an agent known to cause vomiting but it seems to have never attracted much attention from investigators. The relaxant and nitrous oxide technique used by Janhunen and Tammisto³¹ among out-patients undergoing vein stripping operations resulted in a 56.8 per cent incidence of emetic problems. Riding using a similar anaesthetic technique noted "sickness" incidences as high as 80 per cent after major abdominal gynaecological operations, or thoracoplasty in females, but as low as 11 per cent after pneumonectomy in males.¹ Unfortunately he did not give details of how patients were assessed. Nitrous oxide probably causes emetic symptoms by central and peripheral effects although Adriani claims that nitrous oxide does not stimulate the vomiting centre.³² The peripheral effect of nitrous oxide leading to gastrointestinal distension as a cause of emesis has been little investigated.

Scrager and Ivy demonstrated in the unanaesthetised dog that distension of the gallbladder and biliary passages produced nausea, vomiting and distress in proportion to the degree of distension.³³ Goldberg later showed that distension of pyloric pouches with pressures of 30 to 35 mmHg invariably resulted in vomiting.³⁴

The gastrointestinal tract is commonly distended during anaesthesia in two ways. Perhaps the most important is by manual ventilation with a mask following the administration of a non-depolarising muscle relaxant. Secondly, nitrous oxide transfer to the gastrointestinal tract will also result in distension during anaesthesia. Eger estimated that the bowel volume will increase by about half a litre per hour in the presence of an alveolar nitrous oxide concentration of 75 per cent.³⁵ This was only half the predicted maximum increase because equilibrium was not reached and nitrogen was escaping

from the gastrointestinal tract into the blood. Eger notes that under normal circumstances the volume of gas in the gut is about 100 ml and a modest increase would probably have no effect.³⁵ However, he suggests that in the anxious patient or perhaps the patient expecting to vomit postoperatively, air swallowing leads to a far higher initial gut gas volume. Nitrous oxide transfer in these cases will result in greater distension. Eger suggested that air swallowing occurred in ten per cent of cases.³⁵

From what is known of nitrous oxide uptake, clearly a reduction in intestinal distension would be predicted if shorter exposure and lower concentrations of nitrous oxide are used. Parkhouse has associated the incidence of nausea with the concentration of nitrous oxide used, little nausea was reported at concentrations of 20 per cent, whereas at 40 per cent and 60 per cent more subjects became nauseated.³⁶ However, Kvisselgaard using an opiate, nitrous oxide, relaxant technique showed that the duration of anaesthesia was unimportant with regard to nausea and vomiting.³⁷ Our clinical impression is that gastric inflation by manual ventilation is a major contributor to vomiting. This is based on our observation that vomiting appears less frequent if inflation is avoided by a period of preoxygenation; however, this has never been subjected to clinical trial. Some anaesthetists may prefer to compress the abdomen or pass a stomach tube after inadvertent gastric inflation, but this does not guarantee complete emptying of gas.

Muscle relaxants are not thought to influence postoperative vomiting; however, in the study by Janhunen and Tammisto when reversal of muscle relaxation was omitted and patients were allowed to reverse spontaneously a much higher incidence of vomiting was noted. Whether this implies that relaxants are emetics or that the reversal agents with atropine are antiemetics is difficult to decide but the latter is more likely even though the half life of atropine is shorter than that of neostigmine.³¹

Among non-anaesthetic drugs with potent emetic properties, is ergometrine; fortunately many centres have replaced this with infusions of syntocinon, a shorter acting but more tolerable agent.

Gastric suction

Smeesaert reduced the incidence of postoperative nausea and vomiting by decompressing the stomach

with a gastric tube.¹¹ However, Dent in an earlier study did not find a reduced incidence of symptoms using a nasogastric tube, but her population samples varied not only in numbers, 179 patients with tubes and 1821 without, but also in the types of operation performed.³⁸ Holmes on the other hand reported a reduction in vomiting but an increased frequency of nausea and retching when a nasogastric tube was present, which is understandable in view of the likely pharyngeal irritation.³⁹ In the most recent study on the subject gastric suction peroperatively was most effective among a group of patients having cholecystectomies. However, in these patients only the incidence of moderate to severe vomiting (as described by Bellville¹⁰), was reduced; the nasogastric tubes were removed immediately after surgery, thus avoiding pharyngeal stimulation during the postoperative period.³¹

We believe that gastric suction may be a valuable method of reducing vomiting in those situations where the cause is likely to be of gastrointestinal origin, for example following upper abdominal surgery. However, when other causes of vomiting are more likely, e.g., following opiate administration, a nasogastric tube probably has no value.

Duration of anaesthesia

The duration of anaesthesia has long been considered an important factor in the aetiology of emesis. Bellville *et al.* noted an increase in nausea and vomiting from 17.5 per cent for anaesthesia lasting from 30 to 90 minutes to 46.4 per cent for anaesthesia lasting from 150 to 210 minutes.¹⁰ Bonica also reported a greater frequency of emetic symptoms among those who had longer operations.⁴⁰ Longer operations would lead to longer exposure to potentially emetic agents used preoperatively; however, at the same time the effect of preoperative agents should diminish. We have already mentioned that a number of subjects feel nauseated after a wakeful period of fasting. Is it possible that these same subjects are more likely to suffer from postoperative vomiting? Patients may be regaining consciousness after anaesthesia when the mechanism for this feeling of starvation nausea is still active. If this is a possibility, then we should not only concern ourselves with the duration of the anaesthetic but also the total post-prandial period. We do not suggest this to be anything other than speculation, and as no investigator has ever ex-

amined this, future study including this factor would be very interesting.

Site of operation

There is controversy over the importance of the operative site as a factor in postoperative nausea and vomiting. Knapp and Beecher, Burtles and Peckett concluded that with the exception of fenestration operations the operative site was unimportant.^{7,5} Both Dent and Smessaert found that head and neck operations had the highest incidence of nausea and vomiting; Dent quoted 38.1 per cent for head and neck versus 30 per cent abdominal operations as being significantly different.^{38,17} Bonica, however, observed a 70 per cent incidence of emesis among patients who had operations on the stomach, duodenum or gall bladder, and 15 per cent among abdominal wall operations.³¹ Haumann and Foster noted the highest incidences of emetic problems among ear, nose, and throat (ENT) and abdominal operations and lowest after head, neck, and chest operations.²⁷ Unfortunately, all these studies are not comparable, not only have patients not been standardised for known influencing factors such as sex but the premedication and subsequent anaesthetic techniques vary. However, the most consistent findings have been the higher incidences of vomiting among patients undergoing ear and gastrointestinal surgery. A further interesting effect of operative site was noted by Dundee *et al.*; in a controlled study they observed that dilatation and curettage of the uterus resulted in a higher incidence of "sickness" than after evacuation of the uterus not requiring cervical dilatation.⁴¹

Miscellaneous

Among the other factors that may be influences on postoperative vomiting are: instrumentation of the pharynx and the use of oral airways, the inexperienced anaesthetist, prolonged postoperative sleep, peroperative hypoxia, hypotension and carbon dioxide retention.

None of these factors have been strictly evaluated.

Spinal and regional anaesthesia

One would imagine that removing the majority of the known causes of emesis associated with general anaesthesia by employing spinal or regional anaesthesia would reduce the problem of postoperative emesis considerably.

Crocker and Vandam in a retrospective study found that the incidence of emetic symptoms during spinal anaesthesia was 19 per cent.⁴² They found no correlation with age, sex, or physical status and came to no conclusion regarding the part played by premedication. Intraabdominal procedures had higher emetic incidences and Crocker and Vandam argued that this was due to lack of autonomic blockade especially of the vagus; hypotension below 80 mmHg systolic and pain were also significantly associated with a higher incidence of emetic symptoms. Ratra in his prospective study of retching and vomiting during spinal anaesthesia concluded that premedication did not significantly alter emetic events but that hypotension below 80 mmHg increased emesis significantly.⁴³ Further, when 100 per cent oxygen was administered by a Magill circuit perioperative retching and vomiting was significantly reduced. Ratra also recorded a reduction in emesis among patients receiving oxygen despite blood pressures below 80 mmHg and concluded that vomiting centre hypoxia was a further stimulus to vomiting.⁴³ In an earlier prospective study among both sexes conducted by Dent, retching and vomiting were recorded for the first 24 hours. She observed that spinal anaesthesia was followed by an 11.1 per cent incidence of retching and vomiting, whereas regional anaesthesia was followed by a 4.3 per cent incidence.³⁸ Bonica in his prospective 24-hour postoperative survey also included nausea; he found a 21.1 per cent incidence of emesis for spinal anaesthesia and only 8.8 per cent after local anaesthetic blocks of the extremities.⁴⁰ Other than the roles played by hypotension and vagal stimulation, other factors are the administration of opiates as premedicants, possible psychological and visual stimuli may further contribute to pre- and postoperative emesis among these patients. Bonica also noted that in the event of regional anaesthesia being supplemented by general anaesthesia the incidence of emetic problems rose to greater than the combined values of the two techniques separately.⁴⁰

Postoperative factors

Few factors related to the postoperative period have been investigated. Parkhouse suggested that visceral or pelvic pain was a common cause of nausea.⁴⁴ Andersen and Krohg in a study during the first 12 to 24 postoperative hours noted that only ten

per cent of patients had episodes of pain without nausea, whereas 58.6 per cent of patients experienced pain with nausea.⁴⁵ Perhaps more surprising was their finding that opiates given intravenously relieved both nausea and pain in 80 per cent of these episodes, they concluded "relief of pain and persistent nausea was uncommon and if pain relief was inadequate nausea was unabated." Opiates in this study were thought to induce nausea in only 3.4 per cent of occasions; this figure is probably lower than expected. Their patients were kept in a postoperative ward and were probably not subject to the occasionally violent movements of a transporting trolley, that tends to provoke nausea; however, no details of this are available. Evidence that pain and nausea are related is supported by the observation that excessive doses of naloxone not only results in an abrupt return of pain but also nausea and vomiting.⁴⁶ Bellville suggested there was a vestibular component to postoperative sickness since agents that controlled vestibular induced nausea and vomiting were useful.¹³ Opiates are thought to sensitise the vestibular apparatus and so postoperative movements have an important influence on the frequency of opiate induced emesis.⁴⁶ The authors' personal experience after visiting a large group of postoperative dental patients within 24 hours was that the majority of patients who had emetic symptoms said that they had occurred while getting out of bed. This observation could also have been the result of hypotension although this was not recorded at the time.

Parkhouse suggested that "any hypotensive patient is liable to vomit or feel nauseated and that this nausea is characteristically relieved by lying down."⁴⁴

Conclusion

The most recent studies suggest that in the absence of antiemetics the incidence of emetic problems associated with anaesthesia is still around 30 per cent. Although not strictly comparable, these observations are little different from those in the last two or three decades. In this review we have discussed the part played by age, sex, obesity, operative site and duration of anaesthesia and unfortunately these are all predetermined. Postoperative vomiting, however, is rarely the result of a single factor and it is for this reason that management may not be successful. The anaesthetist has therefore been

forced to adopt a practice reliant on antiemetics for symptomatic control. Of the factors under our control the evidence available would indicate that large doses of opiates, and over-enthusiastic manual ventilation by mask may adversely affect the incidence of postoperative vomiting. However, prompt pain relief, normotension and gentle care during the postoperative period are preventative measures that may also help reduce the distressing symptoms of emesis. In part II of this review we will discuss the general and specific measures available for the prevention and treatment of emesis during the postoperative period.

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