PREVENTION OF ACID ASPIRATION: A REVIEW

Pages with reference to book, From 115 To 118

Nasir Khan Jakhrani (Anaesthesiology Department, Jinnah Postgraduate Medical Centre, Karachi.)

INTRODUCTION

Sir James Young Simpson recorded the first anaesthetic death from aspiration of gastric contents in 1848. The patient was a young girl being resuscitated from chloroform anaesthesia by having brandy poured into her mouth. In 1946 the New York obstetrician, Curtis Mendelson,[1] described the syndrome in obstetric patients receivinggeneral anaesthesia[2]. He noted an aspiration incidence in 1:668 deliveries. Recently Nimmo noted that aspiration syndrome formed a cause of death associated with anaesthesia in 25% of maternal, and 19% of all deaths totally attributable to anaesthesia. In our institute.

Acidity of stomach contents

The increased acidity is due to production of gastrin from placenta by twelfth week of pregnancy. Gastrin increases the acid, chloride and enzyme content of the stomach to levels above normal[3]. Aspiration follows passive regurgitation in 80% and active vomiting in 20% of parturients. Work by Teabeaut[4] (1952) in rabbits showed that the severity of the pulmonary lesions was related to the pH of the gastric aspirate, the critical value being 2.5. This value is accepted by most anaesthetists as being that below which severe pulmonary damage occurs[5]. Supporting evidence for this value comes from a small series of non obstetric patients reported by Lewis, Burgess and Hampson[6]. They related mortality following aspiration to the pH of aspirate :100% when the pH was <1.75,25% when the pH ranged from 1.75 to 2.4, but no deaths when pH was greater than 2.4. Crawford has suggested that pH 3 should be regarded as the crictical level.

Reduction of acidity

Because of the severity of acid aspiration, it is logical that attempts should be made to increase the intragastric pH above the so called crictical value and the methods of achieving this have recently been reviewed[8]. The main methods involve:

1. Regular Antacid Therapy.
2. Inhibition of Gastric acid production with H2 receptor antagonists.

Antacid Therapy

Work by many investigators have demonstrated that particulate antacids are effective in raising gastric fluid pH in a reasonably high percentage of both obstetrical and surgical patients in both elective and emergent situations[9]. Indeed, their effectiveness in raising gastric fluid pH depends on:-

1. The volume and pH of gastric contents present at the time of their administration.
2. The frequency and timing of antacid administration.
3. Type and amount of antacid given.
4. What maneuvers, if any are done to promote mixing of the antacid with the gastric contents.
5. The intrinsic gastric motility present at the time of antacid administration and,
6. The rate of ongoing gastric acid production.

Despite theft overall effectiveness in raising gastric fluid pH, particulate antacids have come under criticism in the last few years for two primary reasons. First, theft administration may increase gastric volume if 2-4 hourly dosage is adhered to. This practice has fallen into disfavour. Yet the impact of a single dose of an antacid on gastric volume in an individual patient is highly variable. In fact, in patients with reasonably normal gastric motility, antacids not only may not change volume but conceivably decrease it, because the duodenum can accept more rapidlx neutral gastric contents than highly acidic contents[10]. The second major criticism is that particulate antacid aspiration result not only

in as significant of initial pulmonary derangement as ahighlyacidic aspiratebut also in histologic abnormalities that were present as long as one month following aspiration[11]. Magnesium trisilicate B.P. also has the disadvantages of not mixing well with gastric contents and should be recently prepared[12]. Because of these concerns, an intense interest has been sparked in soluble antacids. Sodium citrate is as effective as particulate antacids at raising gastric fluid pH in both elective and emergency surgical patients if given within 15-60min of induction of anaesthesia[13]. It mixes with gastric contents more readily than particulate antacids. Despite its drawbacks, mist. magnesium trisilicate remains the most widely used antacid in UK[14].

Histamine 112 Receptor Antagonists

It is well documented that the H2 blockers decrease nocturnal, basal and meal stimulated gastric acid production by competitively inhibiting the action of histamine of the H2 receptor of the gastric parietal cell. In contrast they have no apparent effect on gastric emptying time or lower oesophageal sphincter pressure. The preoperative administration of cimetidine decreases acidity of gastric contents in a dose related manner[15]. Timing of administration is important. Oral administration takes about 60- 90 minutes before a significant effect can be demonstrated and this effect seems to be waning by four to six hours. In contrast intravenous administration takes 45-60 minutes although the durationS of action is not necessarily any longer than following Po administration[16]. Intramuscular administration results in earlier and higher initial blood levels and results in a slightly higherpercentage of patients with pH values >2.5 at induction of anaesthesia as compared with Po administration. Multiple dose regimen (a dose the evening prior to surgery and morning of surgery) are some what more effective than single morning doses. Cimetidine is associated with a number of side effects, and in particular inhibits drug metabolizing enzymes in the liver involving anticoagulants, barbiturates, benzodiazepines, propranolol and theophylline[17]. It may result in significant hypotension when given by the I.V. route[18]. Ranitidine has greater potency longer duration of gastric antisecretory effect (six to eight hours), lower incidence of side effects and lesser degree of inhibition of the mixed function oxidase system. Its onset of action is no more rapid than that of cimetidine and therefore it suffers from the same limitations in the emergency situation. Its efficacy in raising gastric pH has proven to be similar to or slightly better than that of cimetidine. The prolonged duration of action is well documented and does offer an advantage. A dose on the eve of surgery should suppress acid production throughout the night while a morning dose should be of long enough duration to provide good conditions well into emergence/recovery period. It may, therefore be concluded that efforts should continue, to increase the intragastric pH in obstetric patients, but the most effective and reliable method and most appropriate agents of doing this have yet to be found. Gillet et al[19] have advocated the use of ranitidine, metoclopramide and 0.3M Sodium citrate with the woman in àstablished labour, solid food is avoided and ranitidine 150mg is administered orally six hourly until delivery. If the operation is required patient is given 10mg meto-clopramide intramuscularly and asked to lie on her left side. Immediately before the induction of anaesthesia 30ml of 0.3M sodium citrate is given by mouth. In elective C. Sections, the ranitidine is given 2 hours preoperatively and the metoclopramide 1 hour later.

INTRA GASTRIC VOLUME

In modern obstetric practice, prolonged labour is avoided and hence there is no longer any need to give fluids by mouth. Among the most potent factors delaying gastric emptying are opioid analgesics. Repeated doses of opioids should be avoided in labouring women and other forms of analgesia should be made available. Mean intragastric volume with extradural analgesia has been found to be: 22ml as compared to 50ml in those without[20]. Mean gastric volume should not be more than 0.3-0.4ml/kg Factors favouring regurgitation include increased intragastric pressure, gastric distension, airway

obstruction, anticholinergics (4 LES tone), altered oesophagocardic angle in the late pregnancy. Diaphragmatic activity during respiratory obstruction, gastric inflation, the pregnant uterus, the lithotomy position and fundal pressure. All raise . intragastric pressure. Narcotics markedly decrease gastric emptying during labour. Excitement during induction/emergence, the stimulation during light anaesthesia and pain and or hypotension during epidural or spinal anaesthesia may induce vomiting. Prevention of inhalation of regurgitated material Prevention offers the best protection against aspiration. Avoid general anaesthesia when possible. I.V. induction of anaesthesia is almost universal m obstetrics, so that active vomiting is not a problem. In all obstetric patients, induction must be accompanied by efforts to prevent any regurgitated material from entering the lungs. The danger period is the time from loss of consciousness until the airway is secured by a cuffed tracheal tube.

Lower oesophageal sphincter (LOS) pressure

One approach to the prevention of regurgitation is to increase the tone of LOS. Metoclopramide increases the sphincter pressure[21]. Cisapride has been shown to increase LOS in both conscious volunteers and patients[22] . It increases the rate of gastric emptying and reverses the morphine induced delay in gastric emptying in patients before anaesthesia. However, the rate of production and composition of gastric secretions are not affected by cisapride. The drug also increases the motility of both small and large intestines. Its parenterál use would cause vasodilation[23]. Atropine, which is frequently given I.V. at induction, consistently decreases LOS tone, but the effect is not evident until 3 minutes after administration, when tracheal incubation would already have been performed. Furthermore it does not increase the incidence of reflux. It is very difficult to envisage that administration of a drug will result in such an increase in LOS pressure that it will prevent regurgitation at induction of anaesthesia. Reliance should not be placed on this method of preventing aspiration of gastric contents.

Position at Induction

All anaesthetists should be proficient at induction of anaesthesia and tracheal incubation in the head down, left lateral position. This ensures that any regurgitated material cannot enter the lungs but, this position is not practical. Induction in the lithotomy position should be avoided because of the associated increase in intragastric pressure makes regurgitation more likely.

Cricoid Pressure

Application of cricoid pressure as described by Sellick[24] is the lynch pin of physical prevention of aspiration. There are however, many reports of deaths after either failure to apply cricoid pressure or its release before passage of a cuffed tracheal tube.

Before induction the cricoid is palpated and lightly held between the thumb and second fmger; as anaesthesia begins pressure is exerted on the cricoid cartilage mainly by the index finger. Sellick also stressed that the neck should be extended bringing the cervical vertebrae forward so that it is easier to occlude the oesophagus by backward pressure on the cricoid. He showed radiologically that contrast media maintained at a pressure upto 94 cmH2o could be prevented from passing beyond the point of application. This method effectively prevents regurgitated material from entering the pharynx but also during cricoid pressure the lungs may be ventilated by intermittent positive pressure without the risk of gastric distension. Cricoid pressure must be applied before loss of consciousness and although not comfortable is well tolerated by the majprity of patients. It has been shown that a force of 44N is required to protect the majority of adults from regurgitation[25].

Failed Tracheal Intubation

When confidential enquiries commenced, tracheal intubation was rarely performed during obstetric anaesthesia. It was the work of Hodges and colleagues[26] that routine intubation after thiopentone and suxamethonium became acceptedpractice and it is noteworthy that in those years the percentage of true maternal deaths from anaesthesia more than doubled. Inability to intubate the trachea in an apnoeic patient is a life threatening situation that demands immediate skilled action, since that time difficulty with tracheal intubation has been increasingly implicated as a factor contributory to maternal deaths.

There are number of reasons for that. The mothers usually have full dentition and the thorax has been lifted into an unusual position by a wedge. Some degree of laryngeal edema maybe present. Incorrect application of cricoid pressure may distort and displace the larynx, while the pressure of large breasts and a hand applying cricoid pressure may make introduction of a laryngoscope difficult. Cormack and Lehane[27] have classified difficulty of intubation into four grades according to the view obtained at laryngoscopy. They suggested that the main cause of trouble was grade 3, in which the epiglottis can be seen, but not the cords. It is not always possible to predict from usual clinical observations whether tracheal intubation will he difficult. Mallampati and colleagues[28] (1985) have suggested a relatively simple sign to predict difficult intracheal intubation, which involves the ability to visualize the pillars of the fauces, soft pallate and base of the uvula when the patient opens the mouth and protrudes the tongue maximally while in sitting position. They found that the degree of difficulty in visualizing these three structures was an accurate predictor of each of direct laryngoseopy. Every one who practices obstetric anaesthesia is going to meet a patient whose trachea cannot be intubated. Thus it is essential that under these circumstances a failed mtubation drill is instituted which is, understood by all labour staff. Failed intubation drill suggested by Thnstall[29] make a sound form. The most important factor in this situation with an apnoeic patient is a prompt decision that a tracheal tube cannot be passed and the lung must be ventilated. Scott[30] has stressed that patients do not die from failure to intubate, they die from failure to stop trying to intubate. All anaesthetists must be capable of ventilating the lungs using a mask, airway and bag; correctly applied cricoid pressure will prevent inflation of the stomach during the procedure. If ventilation with a mask is difficult, then deliberate passage of a tracheal tube or some form of specially designed tube into the oesophagus may allow the lungs to be ventilated until spontaneous breathing returns. Anaesthesia with spontaneous ventilation should continue using a volatile agent with which the anaesthetist is familiar and in a concentration which allows safe completion or surgery.

REFERENCES

1. Mendelson, C.L. Aspiration of stomach contents in the lung during obstetric anaesthesia Am. J. Obstet. Gynaecol., 1946; 52: 191.
2. Nimmo Med., W.S. Aspiration of gastric contents. Brit. 3. Hosp. M., 1985; 34: 176-179.
3. Debring, D.J., Mcdonald, 3.5. Intubation, aspiration propbylsxis in midtrimester abortions. Anesthesiology, 1984; 61: 223.
4. Teabeaut, J.R. Aspiration of gastric contents - experimental study. Am.). PathoL, 1952;28:51.
5. Vandam, Lfl Aspiration of gastric contents in the operative period. N. EngI. 3. Med.,1965; 273: 1206.
6. Lewis, R.T., Burgeaa, i.E., Hampson, LC Cardioreapiratoty studies in critical illness. Changes in aspiration pneumonitis. Arch. Surg., 1971; 103: 335.
7. Crawford, J.S. The anaesthetièts contribution to maternal mortality. Br.J. Anaesthesia,1970; 42:70.
8. Morgan, M. Control of intragastric pH and volume. Brit. 3. Anaesthesia, 1984; 56:47.
9. Taylor, 0., Pryse-Davies, 3. The prophylactic uae of antacids in the prevention of acid pulmonary aspiration syndrome. Lancet, 1966; 1:288.
10. Hunt, iN., Knoz M.T. The slowing of gastric emptying of four strong acids and three weak acids. J> Phyaioi, 1972; 222: 187.
11. Gibbs, C.P., Schwartz, K.J., Wynne, J.W. stat Antacid pulmonary aspiration in the dog. Ansesthesiology, 1979 51: 380.
12. Crawford, J.S. Potter, Sit Magnesum triailicate mixture B.P. Its physical characteristics and effectiveneaa aa a prophylactic. Anaesthesia, 1984; 39: 535.
13. Gibbs, CF., Banner, T.C Effectiveness of bacitra as preoperative antacid. Anaeathesiology, 1984;

61: 97.
14. Sweeney, B. and Wright, I. The use of antacida aa a prophylaxis againat Mendelson's syndrome in the United Kingdom. A survey. Anaesthesia, 1986; 41: 419.
15. Huaemeyer, R.P., Davenport, H.T., Raja Senkram, T. Cimetidine as a single oral dose for propbylaxis against Mendelson;s syndrome. Anaesthesia, 1978; 33: 775.
16. Weber, L, Hirabman, CA. Cimetidine for prophylaxis of aspiration pneumonitis. Anaesthesia and Analgesia, 197% 58: 426
17. Manchikanti, L, Krans, J.W., and Edds, S.P. Cimetidine and related drugs in anaesthesia. Anaesthesia and Analgeaia, 1982 61:595.
18. Hammond, i.E and Ware, R. Hypotenaion following intravenous cimetidine. Anaesthesia, 1983; 38: 701.
19. Gillet, G.B., Watson, J.D., Langford, RM. Ranitidine and single dose antacid therapy a prophylaxi against acid aspiration syndrome in obstetric practice. Anaesthes., 1984; 30 63t
20. Holdaworth, JO. Relationship between stomach contents and analgesia in labour. Br. Anaesthesia, 1978; 50: 1145.
21. Brock-Utne, 3.0., Downing. J.W., Moahal, M.G. and Naicker, M. The administration. metoclopramide with atropine. A drug interaction effect on the gastro oesophage. sphincter in man. Anaesthesia, 1976; 31: 1186.
22. Smout, A.). P.M., Boogard, J.W., Grade, A.C., Ten Thije, O.J., Akkerman's MA Witiebol, P. Effects of ciaapride, a newgastro intestinal prokineticsubstance on interdiger tive and postprandial motor activity of the distal oeaophagus in man. Out, 1986; 26:246
23. Rowbotham, D.J> Cisapride and Anaesthesia. Editorial. Br.). Anaesthesia, 198% 62: 12:
24. Sellick, B.A. Cricoid pressure to control regurgitation of stomach contents during indut tion of anaesthesia. Lancet, 1961; 2: 404.
25. Wraight, W.J., chamney, AS. and HoweHa, T.H. Thedetermination of aneffective crico pressure. Anaesthesia, 1983; 38: 46t
26. Hodges, R.J.H., Bennett, iS., Tunstall, MS. and Knight, R.F. General anaesthesia fo operative obstetriewith special reference to the use of thiopentone and sunmethonum Br.). Anaesthesia, 195% 31: 152
27. Cormack, R.S. and Lehane, J. Difficult tracheal intubation in obstetrics. Anaesthesis 1984; 39: 1105.
28. Mallampati, S.R, Gall, &P., Ougina, LD., Desai, S.P., Warakaa, &, Freiberger, D. Un, P1. A clinical sign to predict difficult intubation a prospective study. Can. Anaesth Soc., J., 1985;32:429.
29. Tunstall, M.E. Failed intubstion drilL Anaesthesia, 1976; 31: 850.
30. Scott, DR Endotracheal intubation friend or foe? Br. Med. J., 1986; 292: 157.