Pneumothorax in the newborn is possibly not a rare condition. Early recognition is important because correct treatment will provide immediate benefit and delay could be disastrous.

The reported incidence of pneumothorax in the neonate varies from 0.07 to 37.5 per cent. The figure of 37.5 per cent (Wieland, 1937) is surprising and it has been suggested, even by the author himself, that this high figure was largely accounted for by artefacts in the radiographs. By routine chest radiography Davis and Stevens (1930) detected six cases among 702 consecutive newborn infants; Howie and Weed (1957), however, suggest that this figure is unduly high and may be accounted for by the poorer radiographic techniques available at that time. Lubchenco (1959) reported an incidence of 0.7 per cent in premature infants and 0.1 per cent in full-term infants. Her results were based on clinical observation together with radiographic examination when indicated and, by similar means, Harris and Steinberg (1954) estimated the incidence to be 0.07 per cent. Obviously, the incidence of pneumothorax detected in any series will depend, at least partly, upon the vigour with which it is sought. It must be expected that the highest figure will be found in series in which all the neonates are examined by radiography, as by this means small collections of air, which give rise to neither respiratory distress nor physical signs, will be detected. It seems probable that the actual incidence of pneumothorax lies between 0.07 and 1 per cent; the clinically important incidence, however, probably lies in the lower part of this range.

**Case 1.**

The mother, aged 26 years and a primigravida, required a forceps delivery on account of foetal distress. The evidence of which was meconium staining and foetal bradycardia. At this stage the foetal head was in mid-cavity and the cervix was almost fully dilated. The baby (weight 3.2 kg) was delivered with moderate traction at 5.45 pm, on September 21, 1963. Atropine 0.6 mg had been injected intramuscularly approximately 20 minutes prior to induction of anaesthesia which was effected by thiopentone sodium 200 mg, suxamethonium chloride 75 mg, facilitated intubation with a No. 9 cuffed oral endotracheal tube, after which the lungs were inflated with a mixture of nitrous oxide (6 1./min) and oxygen (3 1./min), suxamethonium chloride (total 200 mg) being injected intermittently.

The baby cried slightly at birth but within 2 minutes became cyanosed and apnoeic, with a heart rate of 40 beats/min. At 2 minutes a 3-mm plain Magill endotracheal tube was passed and intermittent positive pressure ventilation with oxygen was instituted. The child's colour improved and spontaneous respiration commenced. Tracheobronchial toilet was performed with a soft No. 3 EG rubber suction catheter after which the endotracheal tube was then removed. The child's condition soon deteriorated again, cyanosis returning and the heart rate falling to 80 beats/min.
The endotracheal tube was replaced and intermittent positive pressure ventilation resumed. Because the mother had been given morphine sulphate 15 mg at 3 p.m., nalorphine 0.5 mg was injected at 6.5 p.m. into an umbilical vein. This had no effect. At 6.10 p.m., nikethamide 0.5 ml was similarly injected and this produced only temporary respiratory stimulation and wakefulness. At this stage it was appreciated that even with intermittent positive pressure ventilation the air entry on both sides of the chest was poor. The position of the endotracheal tube was altered, because of the possibility of right endobronchial intubation. This did not improve the situation. A pattern developed so that when the child breathed spontaneously it became unconscious and cyanosed and its heart rate fell to 80 beats/min; on the other hand, intermittent positive pressure ventilation with oxygen increased the level of consciousness, improved the colour, and increased the heart rate to 100-120 beats/min, but still the child did not appear fully oxygenated. At 6.20 p.m., in view of signs of bilateral diminished air entry to the lungs, it was decided to obtain a chest radiograph but, unfortunately, facilities for this did not become available until 8.30 p.m. Meanwhile the child's colour was kept reasonably pink by means of intermittent positive pressure ventilation with oxygen. The radiograph revealed a bilateral pneumothorax (fig. 1), and at 8.50 p.m. a No. 16 intravenous needle was inserted into each pleural cavity through the second interspaces anteriorly. The child's condition improved dramatically. It became pink and fully awake. The heart rate rose to 130 beats/min and the air entry to both lungs became satisfactory. A medium-sized flexible plastic intravenous cannula (Brathnil) was now inserted into each pleural cavity through the 7th intercostal space in the mid-axillary line and each of these was connected to underwater seal drainage bottles. Intermittent positive pressure ventilation was continued for a few minutes and it was noted that whilst air no longer escaped from the left side of the chest, it continued to escape profusely from the right side. Spontaneous breathing was now allowed and the condition remained satisfactory. The endotracheal tube was removed and the child was placed in an incubator where oxygen (2 l./min) at maximum humidity was administered.

The condition remained satisfactory overnight although air continued to escape from the right side of the chest.

On September 22 the child was transferred to the Royal Hospital for Sick Children, Glasgow. Later in the day the plastic cannulae came out and were not replaced. Subsequent portable chest radiographs showed that the left lung was fully expanded but the right lung took about one week to reach this stage. The child made a complete recovery. It should be noted at this stage that intermittent positive pressure ventilation with oxygen during resuscitation was carried out with a manometer in the circuit, and a pressure of 30 cm H$_2$O was not exceeded at any time. The circuit consisted of an oxygen cylinder, a reducing valve and a flowmeter leading, through the manometer, to one limb of the Y-piece inserted into the endotracheal tube. Intermittent occlusion of the second limb of the Y-piece resulted in ventilation of the lungs.

**CASE 2.**

This child (birth weight 3.4 kg) was born at home on April 9, 1963, being three weeks post-mature. Delivery was normal and the child cried at birth. A lumbar meningomyelocele was found and on the same day the infant was admitted to the Royal Hospital for Sick Children, Glasgow. At the time of admission the house surgeon made particular note that the extremities were cyanosed and that crepitations and rhonchi were present all over the lung fields. It was decided, nevertheless, to proceed with operation for treatment of the meningomyelocele. The child was premedicated with atropine sulphate 0.2 mg, given intramuscularly 30 minutes prior to induction of anaesthesia. While the infant was conscious the trachea was intubated with a 3-mm flexible metal Magill endotracheal tube. Anaesthesia was then administered in the form of nitrous oxide (3 l./min), oxygen (3 l./min) and halothane using intermittent positive pressure ventilation. At no time was inflation of the lungs difficult. It was apparent, however, that the child was not fully oxygenated despite receiving 50 per cent oxygen. The position of the endotracheal tube was carefully checked but there was no evidence of the tube having entered a bronchus. Anaesthesia was continued and the lumbar meningomyelocele was excised, with the child in the prone position. Anaesthesia lasted 45 minutes and the
operation 30 minutes. At the close, when the endo-
tracheal tube was removed, the child was slightly
cyanosed. It was placed in an incubator and oxygen
administered at a rate of 3 l./min. The colour then
improved. However, the nursing staff reported that the
baby had become cyanosed on several occasions
during the night after operation and a portable chest
radiograph taken the following morning revealed
small bilateral apical pneumothoraces with partial col-
lapse of the upper lobes and a pleural adhesion at the
left apex (fig. 2). The radiologist was of the opinion
that the air had probably entered the pleural spaces
from the upper mediastinum.

It was decided not to aspirate the air from the
pleural cavity as the child appeared normal when
undisturbed and showed only minimal signs of res-
piratory distress when being handled and fed.

On April 11 oxygen was discontinued. The child's
colour was good and no respiratory distress was
present. Lateral and antero-posterior chest radiographs
were taken and demonstrated improvement with ex-
pansion of the lungs, but there was still a loculated
pneumothorax in the anterior and superior medias-
tinum.

FIG. 2
Antero-posterior radiograph of chest. There is a small
bilateral apical pneumothorax with partial collapse
of the upper lobes and a pleural adhesion at the left
apex.

During the following weeks this child showed
increasing clinical evidence of hydrocephalus. On
May 17 right ventriculo-atrial drainage was carried
out; general anaesthesia was employed, the technique
being identical to that on the previous occasion. There
was no recurrence of pneumothorax. On June 15 the
child was discharged home.

DISCUSSION

The classical work on the aetiology of pneumo-
thorax was carried out by Macklin (1937), who
described the histological appearances of the lungs
and mediastinum of cats following deliberate
over-inflation with air. He was able to show that
the probable mechanism was over-distension of
alveoli followed by the rupture of their walls and
by the passage of air into the sheaths of the pulmo-
ny arteries. The air then
passed along the line of least resistance, namely
into the sheaths of the larger pulmonary arteries
(where the interstitial space is greater) and then
into the mediastinum. Macklin then showed that
if the volume of air in the mediastinum was large
enough, the mediastinal pleura might rupture in
one or more places, giving rise to a pneu-
mothorax on one or both sides. The mediastinal air
also spread along the aorta and oeso-
phagus into the retroperitoneal tissues or even
enter the peritoneal cavity, giving rise to a pneu-
moperitoneum. If the air dissects into the sub-
cutaneous tissues of the neck, subcutaneous
emphysema results. It is now known that the air
may not leave the mediastinum at all and may
persist as an isolated pneumomediastinum. This
account of the pathological basis of pneumothorax
is still accepted thirty years later, although some
authors believe that in the case of over-dis-
tended subpleural alveoli the visceral pleura
may rupture, giving rise to a pneumothorax
directly (Deaton and Doyle, 1960).

More controversy surrounds the question of
how alveolar over-distension occurs. Various
aetiological factors have been described and are
now discussed.

Pneumothorax occurs frequently among new-
born infants who have been subjected to resus-
citative measures, and Nelson (1959) believes it
likely that the most common cause is over-infla-
tion of the lungs with resulting alveolar rupture.
This, however, is not evidence that intermittent
positive pressure ventilation is responsible for
PNEUMOTHORAX IN THE NEONATE

pneumothorax in any particular instance. In some cases pneumothorax may have been the cause of the respiratory distress which resulted in the employment of intermittent positive pressure ventilation. It must be rare for chest radiographs to be obtained during the first few minutes of the infant's existence, but only in this way could it be shown whether a pneumothorax preceded or followed intermittent positive pressure ventilation. Not only has endotracheal intermittent positive pressure ventilation been incriminated; according to Emmert (1930) and Craig et al. (1962) direct mouth-to-mouth respiration, if applied too vigorously, can over-distend and rupture alveoli.

Most clinicians accept the need for endotracheal intubation and intermittent positive pressure ventilation in certain cases of neonatal respiratory distress, although the range of indications for such treatment varies very widely. The question then arises as to what inflating pressure can be used safely. Donald, Kerr and Macdonald (1958) have shown, by the measurement of oesophageal pressures (which are believed to correlate closely with intrapleural pressures), that pressure swings of up to 90 cm H₂O can occur in the newborn. On the other hand, Donald and Lord (1953) have shown, using immediately excised lungs of fresh human stillbirths, that it is possible to produce macroscopic evidence of damage at positive pressures exceeding 70 cm H₂O.

In the past, authors have recommended different safe upper limits of pressure which can be applied. Examples are as follows: Wylie and Churchill-Davidson (1960), 15–30 cm H₂O; Rees (1961), up to 25 cm H₂O; Hodges and associates (1960), up to 40 cm H₂O; Donald (1960), up to 35 cm H₂O. The positive pressure applied, however, is not the only factor involved. Day and colleagues (1952) have, as the result of animal experiments, thrown much light on this problem. They state that injury to a lung arises from distension, not from pressure, and that distension is proportional not only to pressure but also to the time during which pressure is applied. A given pressure applied for a longer time will produce greater expansion of the lungs than the same pressure for a shorter time. When the lungs of a neonate require to be inflated at birth, usually some parts are atelectatic whereas other parts are at least partially expanded. Care is required if the atelectatic areas are to be expanded without over-distending the partially expanded areas. These authors state that low pressures (of the order of 15 cm H₂O) are safe, but ineffective, in expanding atelectatic lungs; on the other hand, high pressures applied over a long period will expand the atelectatic areas but will over-distend and rupture the already expanded areas. In their experience atelectasis of foetal lungs can be safely and effectively corrected by application of pressures of the order of 40 cm H₂O applied for a time interval of 0.15 sec or less; however, they also state that the difference between 0.15 sec and 0.2 sec may represent the difference between safety and danger. Obviously, it is impossible without special apparatus to measure accurately such small time intervals when ventilating the lungs of the neonate, as Crawford (1959) has pointed out. The work of Day and colleagues is very important because it stresses the pressure-time relationship.

Crawford (1959) suggests that a pressure of 25 to 35 cm H₂O should be applied for 0.5 to 1 sec with an interval of 1 sec between applications. At pressures of 35 cm H₂O he believes that the duration of inflation is not quite so critical in relation to alveolar rupture as is the case with higher pressures, the margin of safety, on the basis of animal experiments, being very considerable. Above the level of 35–40 cm H₂O the duration of application assumes a significance which increases greatly with each successive rise in pressure exerted.

The position is that if too high an inflating pressure is applied there is risk of a pneumothorax developing; on the other hand, if the pressure is too low, expansion of collapsed lung will not occur. The currently accepted view appears to be that it is safe to employ pressures up to 30 and possibly 35 cm H₂O provided that the positive pressure phase does not exceed 0.5–1 sec. Once the lungs have expanded, much lower pressures are sufficient. When the lungs are being ventilated it is important to note whether the movement of the chest and air entry is satisfactory on both sides so that ventilation...
of one lung only is avoided. If the position of the endotracheal tube is satisfactory, and air entry to a lung or part of a lung is diminished despite previous tracheobronchial aspiration of secretions, the positive pressure applied should not be increased above 30–35 cm H₂O. Air entry to part of the chest may be diminished as a result of conditions such as diaphragmatic hernia or agenesis of a lung; in these conditions an increase in positive pressure above 30–35 cm H₂O will be of no benefit and may indeed be harmful, as a pneumothorax may result, thus adding to the difficulties. The correct course is to obtain a chest radiograph and by this means endeavour to discover why the air entry is diminished.

While intermittent positive pressure ventilation has been discussed as a possible cause of neonatal pneumothorax there is evidence that it occurs most frequently in the absence of any form of resuscitation. The National Survey of Perinatal Mortality (Butler, 1961) showed that endotracheal intubation or mouth-to-mouth respiration had been employed in less than half the neonatal deaths due to pneumothorax. What, then, is the alternative mechanism? It is believed that over-distension of alveoli may occur as the result of partial airway obstruction as in these circumstances air may enter the lungs during inspiration (when there is a relative bronchodilatation) but does not pass the obstruction during expiration (when there is a relative bronchoconstriction). Various factors have been incriminated as the cause of the bronchial obstruction, including aspirated mucus or amniotic fluid and congenital abnormalities (Lubchenco, 1959). According to Deaton and Doyle (1960) predisposing causes include prematurity, difficult delivery, breech delivery and Caesarean section. Butler (1961) describes the underlying picture as usually one of intrapartum asphyxia. Obviously asphyxia, if it produces respiratory stimulation, will favour aspiration of amniotic fluid and also an exaggerated respiratory effort which will tend to force air past a respiratory tract obstruction, thus facilitating the development of a pneumothorax. Less commonly, other causes are found to account for pneumothorax in the neonate. Butler (1961) described a case of neonatal pneumothorax resulting from a laceration of the oesophagus caused by the insertion of rubber tubing into the stomach for the gastric administration of oxygen. The rupture of a subpleural congenital pulmonary cyst has also been suggested as an uncommon cause of pneumothorax (Nelson, 1959). A more rare cause of pneumothorax in the neonate is injury to the chest wall from obstetric instruments (Holt and McIntosh, 1953).

It is difficult to be certain of the aetiology of the bilateral pneumothorax in the two cases described here. In case No. 2 pneumothorax was probably present prior to anaesthesia. On admission to hospital there was peripheral cyanosis together with bilateral rhonchi and crepitations; a clinical picture compatible with a diagnosis of pneumothorax and pulmonary collapse. Unfortunately, a radiograph was not taken prior to anaesthesia. No form of resuscitation was necessary at the birth of this baby; it appears likely that the pneumothorax resulted from the child's own respiratory efforts, the mechanism having been described earlier.

Case No. 1 is more problematical. Two main possibilities must be considered. The infant may have developed a pneumothorax immediately after it was born; at this stage it cried and could conceivably have developed alveolar over-distension and rupture as a result of partial bronchial obstruction by inhaled amniotic fluid. Since vigorous respiratory activity was not noted at this stage, this possibility is unlikely. The other possibility is that intermittent positive pressure ventilation resulted in alveolar over-distension although at no time did the inflating pressure exceed 30 cm H₂O. The endotracheal tube used was long, and study of the chest radiograph shows that its tip lay within the right main bronchus. The effect of right endotracheal intubation would be that a greater volume of gas would enter the right lung initially but a pressure of 30 cm H₂O would build up more rapidly than normal. Release of pressure at this stage would mean that gas would have entered the right lung at a greater rate for a shorter time and the net change in the volume of gas entering the lung would not be great. For this reason the presence of the tip of the endotracheal tube in the right main bronchus should not predispose to the development of the pneumothorax.
The diagnosis of a pneumothorax.

A small pneumothorax will often escape notice unless a radiograph is taken, as only large collections of air produce classical physical signs. Furthermore, physical signs are often difficult to interpret in tiny infants and on the basis of these signs it may not be possible to differentiate pneumothorax from other conditions causing a fairly similar clinical picture. A chest radiograph should, therefore, always be obtained when abnormal physical signs are present in the chest; this will reveal a pneumothorax when this is present. Tension pneumomediastinum is being recognized increasingly as an occasional cause of respiratory distress in the newborn (Ibrahim, 1964). The aetiology is fairly similar to that of pneumothorax, the condition resulting from alveolar rupture and the passage of air, along the sheaths of the pulmonary blood vessels, to the mediastinum. The mediastinal air does not rupture into the pleural cavity or into the subcutaneous tissues of the neck, but remains trapped, compressing the venae cavae and the pulmonary veins (and even the trachea and bronchi on occasion), thus causing circulatory and respiratory embarrassment. According to Jewett, Adler and Taheri (1962) it is very difficult to differentiate tension pneumomediastinum from the other causes of respiratory distress of the newborn, on the basis of clinical examination. The condition can only be diagnosed with certainty by means of radiography. A pneumomediastinum is often not apparent on a postero-anterior film, and therefore, a lateral view is required. This will show a pocket of air in the mediastinum if the condition is present. The subsequent progress of the condition, in the absence of treatment varies. The mediastinal air may gradually be absorbed with complete recovery. Spontaneous decompression may occur by rupture into the pleural space or the subcutaneous tissues of the neck. Finally, death may occur from progressive respiratory embarrassment. The treatment is described later in this paper.

Differential diagnosis of pneumothorax.

In differential diagnosis of pneumothorax it is necessary to exclude other conditions which cause respiratory distress in the neonate. Briefly, these are:

1. central depression of respiration (for example, as the result of sedation of the mother);
2. respiratory obstruction (for example, micrognathia or subglottic stenosis);
3. compression of the lung (for example, congenital diaphragmatic hernia);
4. intrapulmonary pathology (for example, atelectasis or neonatal pneumonia);
5. congenital heart disease.

Often only by means of chest radiography can some of these conditions be diagnosed with certainty.

Treatment of pneumothorax in the neonate.

The general principles of treatment are similar to those used in the management of pneumothorax in the adult. The severity of the condition must first be assessed clinically and radiologically. If the pneumothorax is small and is producing no signs of respiratory distress, no active treatment is indicated, but the child should be observed closely as sudden worsening of the condition may occur. If the pneumothorax is large, or if respiratory distress is present, the air should be aspirated from the pleural cavity by means of a needle (inserted through the 2nd or 3rd intercostal space anteriorly) and syringe. If the air re-accumulates or if a tension pneumothorax is present it becomes necessary to employ underwater seal drainage. A small skin incision is made and a self-retaining catheter (such as a Malecot pattern) is inserted with the aid of forceps into the appropriate side or sides of the chest through the 2nd or 3rd intercostal space anteriorly and is connected to an underwater seal drainage bottle. In an emergency an intravenous needle or preferably the blunter Braüinule (which is less liable to produce pulmonary damage) may be used. The underwater seal drainage system used should be similar to that used for adults. Since the intrapleural pressures developed by a neonate are similar to those of the adult, it is a fallacy to suppose that a scaled-down bottle and tubing should be used for the neonate.
The treatment of tension pneumomediastinum can be conveniently considered here. If respiratory distress is minimal, conservative treatment with oxygen and increased humidity may suffice. The infant must be observed closely, however, in case sudden deterioration occurs. If the infant shows increasing signs of respiratory distress, surgical decompression of the pneumomediastinum should be carried out. This can usually be achieved by insertion of a needle through the 3rd anterior intercostal space (preferably on the side on which air is seen on a postero-anterior chest film); the needle should be inserted just lateral to the sternum and directed towards the anterior mediastinum until air is aspirated; this should result in immediate improvement in the infant's condition. If air re-accumulates, it may be necessary to repeat the procedure, but if re-accumulation of air occurs repeatedly this is an indication for underwater seal drainage of the mediastinum as described for pneumothorax. Rarely, if needle aspiration is unsuccessful, a more radical approach may be necessary. A collar incision should be made over the suprasternal notch and the loose tissue behind the upper end of the sternum opened up by blunt dissection to permit escape of the mediastinal air.

ACKNOWLEDGMENTS
I wish to express my thanks to Mr. W. M. Dennison, Mr. J. F. R. Bentley and Mr. H. Stirling for permission to present the two cases; to Dr. C. H. Hodge and Dr. J. D. Muir for assistance in the management of Case No. 1; to Dr. H. H. Pinkerton and Dr. J. R. Munro for the very considerable help and encouragement which they gave me during the preparation of this paper; and to Mr. J. Devlin, who prepared the photographs. The two cases of pneumothorax were encountered while on rotational duties from my main hospital.

REFERENCES
PNEUMOTHORAX IN THE NEONATE

de 30–35 cm H₂O pendant 0,5 à 1 sec. La radiographie du thorax est très importante pour le diagnostic de la détresse respiratoire chez le nouveau-né. Suivant la sévérité du pneumothorax en cause le traitement peut aller des mesures conservatrices jusqu’au drainage intercostal sous l’eau.

PNEUMOTHORAX BEIM NEUGEBORENEN

ZUSAMMENFASSUNG


CORRESPONDENCE

RE-ANAESTHETIZING CASES OF TONSILLETOLOGY AND ADENOIDECTOMY BECAUSE OF PERSISTENT POSTOPERATIVE HAEMORRHAGE

Sir,—There are certain points in Dr. Denison Davies’ article on this subject (Brit. J. Anaesth., 36, 244) which merit challenging!

With reference to the advocated removal of clots before inducing anaesthesia, I find that clots which are really loose are expectorated or swallowed by all but heavily narcotized patients, while the usual semi-adherent clots in tonsil fossae remain in position during anaesthetic induction and have to be swabbed away by the surgeon: when this is done, fresh and maybe brisk bleeding from the vessel concerned (there is invariably only one such) occurs. Surely the last thing desirable would be fresh and fast bleeding during induction of anaesthesia?

Having personally so far effected upwards of 10,000 tonsillectomies with or without adenoidectomy, and likewise dealt with something like forty cases of postoperative haemorrhage, it seems most remarkable that over 200 cases should have been quoted as returned to the operating theatre for postnasal bleeding: I have known only one such case where another operator had, at a guillotine operation, left half the adenoids behind. Such bleeding, to my mind, implies imperfect adenoidectomy, which can be avoided altogether in the open "dissection" operation which is nowadays, one hopes, the routine procedure elsewhere as hereabouts, where anaesthetists feel quite free to use nasally introduced intratracheal tubes.

By all means empty the stomach of swallowed blood if possible; but blood-volume diminution of serious degree should never occur in primary postoperative haemorrhage if promptly dealt with, and in this connection I think the surgeon's opinion ought always to prevail. I teach juniors that primary haemorrhages should never be allowed to reach the stage of having to contemplate blood replacement. Secondary haemorrhages admitted from outside hospital may, of course, be in a condition to need blood replacement before operation.

M. HAYDON-BAILLIE

Worksop

A copy of this letter was forwarded to Dr. Denison Davies, who replied as follows:

Sir,—I should like to comment on one or two points raised by Mr. Haydon-Baillie.

Respiratory obstruction by inhaled blood clots is a very real hazard in these cases and one cannot rely on all loose clots being expectorated or swallowed before induction of anaesthesia. Naturally one does not want to start fresh bleeding, and therefore only clots which are obviously loose should be removed.

It is certainly remarkable that over 200 cases should be quoted as having returned to the theatre because of postnasal bleeding. These cases were from a series of 21,500 children, the majority of whom had had adenoidectomy performed; this, certainly, must represent an abnormally high return rate. In this connection it is significant that out of a total of five deaths in this series of 21,500 cases, four were directly related to postnasal bleeding. This high return rate is probably related to the fact that most of the tonsillectomies and adenoidectomies in this series were performed by junior house surgeons in training. It is possible that postoperative bleeding is occasioned by imperfect adenoidectomy, but one wonders whether the too radical use of an over-sharp curette may not be a more common cause.

Mr. Haydon-Baillie states that in cases of primary postoperative haemorrhage serious blood volume depletion can always be avoided. This, most certainly, is an unattainable ideal and must represent a dangerously unrealistic approach to the problem. Blood can be swallowed by the patient and a considerable blood loss may have occurred before the ward staff are aware that the patient is bleeding. In fact in many cases postoperative bleeding may be actually heralded by the patient, quite unexpectedly, vomiting a dangerously large quantity of blood.

One wonders what is meant by a "serious" degree of blood-volume diminution. A deficit which is not serious in a patient lying in bed in a ward may be very serious if that patient is anaesthetized and his airway becomes temporarily obstructed by blood clots. Naturally the anaesthetist must accept repon-