from Corfman, Diamond, DeCherney REF26

Venous Air Embolism During Operative Hysteroscopy

44

Philip G. Brooks

Introduction

A rare, but devastating complication of operative hysteroscopy is venous air entrapment and subsequent room-air embolization. While this phenomenon has been reported to occur during neurosurgical (1) and urological (2) surgery and at caesarean section (3), its occurrence during operative hysteroscopy has been cited only in obscure instances except as a complication of the inappropriate use of a dextran pump (4). Two cases were reported in letters to the editor of an anesthesia journal (5, 6), and one case was described in a letter to a British medical journal (7). Corson, in a recent publication on endoscopic air embolism in general (8), included several cases of hysteroscopic air embolism complications, some of which are included in this chapter. The author has been asked to review seven additional cases of this complication, either as a consultant for a hospital peer-review process or as an expert in a litigation. This chapter will describe these cases and the pathogenesis of this problem, with recommendations for its prevention and management when it occurs. Because some cases are still pending, they will be presented anonymously and with intentional minor changes.

Pathogenesis

In neurosurgery, when procedures are performed with the patient in the sitting position in order to gain access to posterior lesions, it is well recognized that, unless the brain is bathed in saline solution, opening venous sinuses in the calvarium or dura will permit venous air aspiration. This situation arises due to the fact that, because the heart is below the level of the brain, a negative intravenous pressure occurs with each diastolic relaxation of the heart (1). Such an event occurs in 25% to 50% of such neurosurgical operations, depending on the detection method. Once the air enters the venous circulation, foaming appears in the right side of the heart and blood outflow becomes obstructed, increasing the pulmon arterial pressure. Early in the development of problem, end-tidal CO_2 declines; ultimately, circulat collapse and cardiac arrest occur. Because the press increases in the right side of the heart to levels hig than those in the left heart, the previously closed foram ovale may open in more than 15% of adult patien resulting in paradoxical embolism to the brain and otl organs (1).

In gynecological procedures, the identical mechanis exists. Instead of the patient in the sitting positic however, his or her head is tipped down, putting t heart below the level of the uterus. If the surgeon ope some of the large sinuses deep in the myometrial wall a leaves an open passage to the outside, room air is as rated into the venous circulation.

Review of cases

In addition to the three cases cited earlier from letters the editors, seven cases of venous air embolism perso ally reviewed by the author are summarized in Tab 44.1. Five of the patients were undergoing hysteroscop for the management of abnormal uterine bleeding, one which was following a spontaneous abortion. The oth two were undergoing repair of intrauterine defects fc fertility reasons, one a congenital defect (septum) and or an acquired defect (uterine synechiae). Five of thes patients were stated to be in a Trendelenburg positio one was reported not to be tipped, and the other had n statement as to the degree of tip. Difficulty with dilatin the cervix was reported in three of the seven procedure two indicated no such difficulty, and no information wa available on the other two. No correlation was foun between the development of air embolism and the dis tention medium used, with three procedures employin carbon dioxide at appropriate flow and pressure, an three using different low-viscosity liquids; the remainin case had not yet started the hysteroscopy, as the first sig

Table 44.1. Summary of cases of venous air embolism occurring at operative hysteroscopy.

Case No.	Reason for Surgery	Trendel. Position?	Difficult Dilation?	Distention Medium	Time to Sign of Trouble	Clinical Signs	Result
1.	Uterine septum	Yes	n/s	CO_2	35 s	↓ E-T CO_2 ↓ PO_2	D.I.C., death
2.	Abnormal bleeding	n/s	yes	CO_2	9 min	Bradycardia; Doppler = bubbles, both sides of heart; septal defect	Coma, death
3.	Abnormal bleeding	yes	no	CO_2	10 min	Mill-wheel murmur; ↓ E-T CO_2 ↓ PO_2; Doppler = bubbles, both sides of heart	Hyperbaric chamber, survived
4.	Menorrhage	yes	yes	none	"after D&C only"	Bradycardia; ↓ E-T CO_2; CVP line withdrew bubbles	Death
5.	Menorrhage	no	yes	glycine	15 min	↓ E-T CO_2 ↓ PO_2; mill-wheel murmur.	Resuscitated, recovered
6.	Incomplete abortion	yes	no	Ringer's lactate	15 min	↓ PO_2 ↓ E-T CO_2; CVP line withdrew >50 cc bubbles	Death
7.	Synechiae	yes	n/s	saline	<10 min	↓ PO_2; bradycardia; inguinal crepitance; gas recovered from femoral vein and cardiac taps	Death

n/s = not stated.
E-T = end-tidal.

of trouble occurred at the end of a preceding dilation and curettage.

All of the complications emerged within 15 minutes after beginning the actual operative procedure. In each case the earliest signs of problems were dramatic changes noted by the anesthesiologist. These signs included either a sudden fall in end-tidal CO_2 measurements, bradycardia, a fall in oxygen saturation, or the presence of the classic sign of air in the heart, a mill-wheel type of murmur auscultated over the precordium. Confirmation of the presence of gas in the circulation was achieved in two cases with the use of emergency echocardiography and by needle or central venous pressure catheter aspiration in three others.

Five of the seven patients died from the complication, with the other two recovering. One recovery was attributed to the brilliance of the anesthesiologist in turning the patient onto her right side, rapidly infusing physiologic saline solution intravenously, confirming the condition with a transesophageal echocardiogram, and ordering the transfer of the patient to a hyperbaric chamber.

Discussion

Drawing from the understanding of the development of venous air embolism during other surgical procedures, a fairly clear pattern emerges as to how this complication happens during operative hysteroscopy. Very often—if not for every procedure—the patient's position on an operating table places the level of the heart and vena cava below the level of the uterus; this differential is often accentuated by requesting that the head of patient be tipped even more downward (Fig. 44.1), mainly so that weighted specula stay in place better. With the current use of video cameras for hysteroscopic procedures, almost no other reason exists to tip the patient.

When operative instruments are to be used, their larger caliber requires that the cervix be dilated more than with diagnostic instruments. This process can result in either occult lacerations and false passages at the level of the internal cervical os, or in partial penetrations into the myometrial wall at the top of the fundus from a partly blunt dilator "popping" through the cervix after considerable force is applied to dilate the canal. At that point, while the surgeon is assembling and readying the operative instruments, the cervix and vagina are left open to room air and the negative pressure in the vascular tree literally sucks the air in.

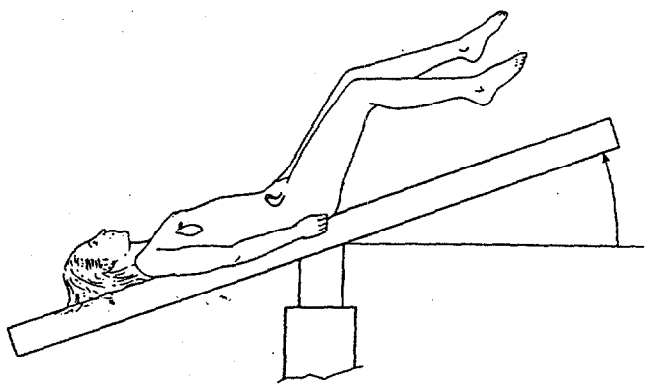

Fig. 44.1 Hysteroscopy in the Trendelenburg position.

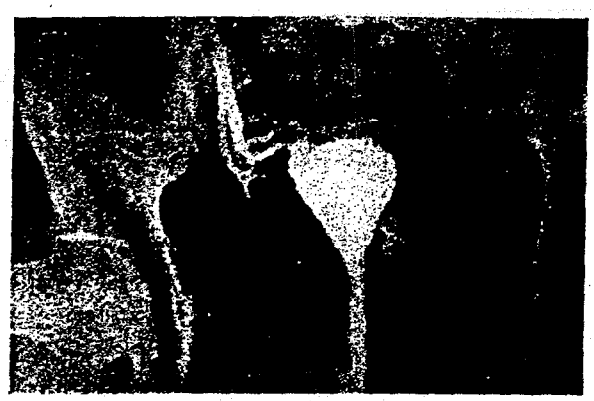

Fig. 44.2 Hysterogram showing a fistulous communication between the uterine cavity and a major uterine blood vessel, created by a deep biopsy. (Photo courtesy of Ray Garry, M.D.)

Evidence for large sinus communication with the venous circulation has been shown brilliantly by Garry (9). In this research, following a biopsy deep into the myometrial wall, dye instilled at intrauterine pressure higher than venous pressure was seen radiographically to flow into the venous system at flow rates sufficient to give a clear venogram (Fig. 44.2). That cardiorespiratory function is altered so soon after the beginning of the procedure attests to the fact that the bubbles noted must be room air, as carbon dioxide (used in three of the cases) has a wide margin of safety at the flow rates and pressures used in hysteroscopy, even if insufflated directly into the venous circulation (10). Because of its high solubility in plasma, it would take much more time or much higher flow rates to create this condition using carbon dioxide.

Detection and management

Figure 44.3 shows the sensitivity of methods used to detect the presence of gas in the heart and great vessels and to monitor the subsequent physiological changes (8). Echocardiography, either by transesophageal probe or by precordial Doppler, may be the most sensitive technique to detect as little as 0.5 mL of gas bubbles in the heart (1). Neither method is widely used by anesthesiologists, especially for "low-risk" gynecological procedures, because of the high false-positive readings obtained. As noted in the case summaries, however, both techniques were used to define and document the event.

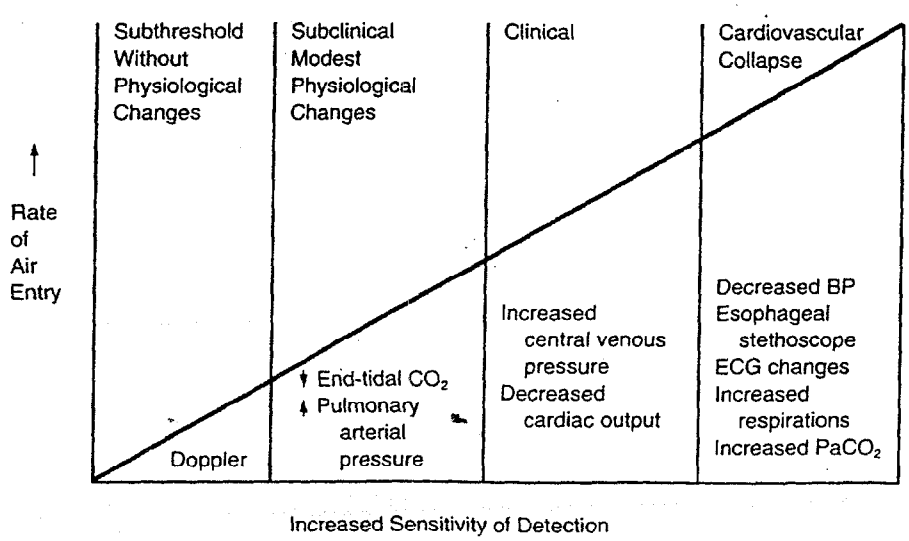

Fig. 44.3 Venous air embolism monitoring, showing the development of abnormal cardiorespiratory parameters as more air enters the vascular system. [Courtesy of Stephen L. Corson, M.D. (8).]

As more air enters the bloodstream, the fall in end-tidal CO_2 is highly sensitive and specific for embolism. Most patients undergoing general anesthesia today are monitored by capnography, making this one of the most important early signs. If a central venous pressure catheter is placed, either prior to the procedure (for high-risk patients) or during the procedure (as difficulties arise), elevation of intracardiac and pulmonary arterial pressures can be detected and monitored. Aspiration of the bubbles, when detected, can assist in the correction of the problem. As more gas is entrapped, the increasing resistance to blood flow results in hypoxia, decreased cardiac output, hypotension, tachypnea, and cardiac arrest. Resuscitative efforts at this point might be aided by stopping the source of the air inflow, turning the patient onto her right side, attempting to aspirate the bubbles as much as possible and flushing the circulation with a large saline bolus.

Prevention

The events in venous air embolism are so sudden and so severe as to make their management extremely difficult. Consequently, this complication often results in death or severe disability. It is obvious that prevention is the most important lesson from the review of this problem.

To begin with, avoid putting the patient in the Trendelenburg position. As noted earlier, the use of video cameras essentially eliminates the need to tip the patient head-down. Dilate the cervical canal with care, attempting to avoid lacerations and partial penetrations into the myometrial wall. After the dilation, do not leave the cervix and vagina open to room air for any length of time. If the instruments were not prepared before the dilation is performed, allow the vagina to close or place a wet gauze sponge or packing against the cervix while they are assembled. Another suggestion would be to leave the last dilator in the cervical canal until the surgeon is ready to insert the hysteroscope and start the distention gas or liquid flowing. The best protection against a complication due to venous air embolism is understanding how it develops.

REFERENCES

1. Shapiro HM, Drummond JC. Neurosurgical anesthesia and intracranial hypertension. In: Miller RD, ed. Anesthesia, 3rd ed. New York: Churchill Livingstone, 1990:1737–1789.
2. Albin MS, Ritter RR, Reinhart R, Erickson D, Rockwood A. Venous air embolism during radical retropubic prostatectomy. Anesth Analg 1992;74:151–153.
3. Lowenwirt IP, *et al.* Nonfatal venous air embolism during caesarean section: a case report and review of the literature. Obstet Gynecol Surv 1994;49:72.
4. Nachum Z, Kol S, Adir Y, Melamed Y. Massive air embolism—a possible cause of death after operative hysteroscopy using a 32% dextran-70 pump. Fertil Steril 1992;58:836–838.
5. Michael A. Endometrial ablation and air embolism (letter). Anaes Intens Care 1993;21:475.
6. Eugster D. Cardiac arrest during endometrial ablation (letter). Anaes Intens Care 1993;21:891–892.
7. Wood SM, Roberts FL. Air embolism during transcervical resection of endometrium (letter). Br Med J 1990;300:945.
8. Corson SL, Brooks PG, Soderstrom RM. Gynecologic endoscopic gas embolism. Fertil Steril 1996;65:529–533.
9. Garry R. Endometrial laser ablation. References Gynecol Obstet 1994;2:188–195.
10. Corson SL, Hoffman JJ, *et al.* Cardiopulmonary effects of direct venous CO_2 insufflation. J Reprod Med 1988;33:440–445.

Complications of Laparoscopy and Hysteroscopy

Second Edition
Edited by

Randle S. Corfman, PhD, MD
Director
Midwest Center for Reproductive Health, P.A.;
Assistant Clinical Professor of Obstetrics and Gynecology
Department of Obstetrics and Gynecology and Family Practice
University of Minnesota
Minneapolis, Minnesota

Michael P. Diamond, MD
Professor of Obstetrics and Gynecology
Wayne State University School of Medicine;
Director of Reproductive Endocrinology and Infertility
Hutzel Hospital
Detroit, Michigan

Alan H. DeCherney, MD
Professor and Chairman
Department of Obstetrics and Gynecology
UCLA School of Medicine
Los Angeles, California

b
Blackwell
Science

FERTILITY AND STERILITY
Vol. 65, No. 3, March 1996
Printed on acid-free paper in U. S. A.

JW 4/01

Gynecologic endoscopic gas embolism

1 saline
2 CO_2
1 N_2O
1 nothing?

Stephen L. Corson, M.D.*†‡§
Philip G. Brooks, M.D.‖
Richard M. Soderstrom, M.D.¶

Thomas Jefferson University School of Medicine, Pennsylvania Hospital, Graduate Hospital, and the Philadelphia Fertility Institute, Philadelphia, Pennsylvania; University of California Los Angeles School of Medicine, Cedars-Sinai Hospital, Los Angeles, California; and University of Washington School of Medicine, Seattle, Washington

Objective: To alert gynecologic surgeons to the risk of room air embolism during endoscopy.
Design: Case reports.
Setting: Medico-legal consultations.
Patients: Five women having endoscopic procedures.
Interventions: Endoscopy followed by emergency resuscitative measures.
Results: Morbidity and mortality.
Conclusions: The risk of room air embolism may be lessened by attention to the operative technique and by monitoring the end tidal carbon dioxide levels.
Fertil Steril 1996;65:529–33

Key Words: Gynecologic endoscopy, hysteroscopy, laparoscopy, room air embolism

Risks of air embolism during obstetric and/or gynecologic operative procedures were documented before the advent of endoscopic technology. Even nonsurgical procedures, such as entry of air into the placental sinusoids during orogenital sexual activity during pregnancy, can result in death (1). The famous gynecologic surgeon, C. Lee Buxton (2), described patient mortality in a case where room air was used to test tubal patency during surgery. Death has been described from misconnected suction machines when the exhaust tubing was placed into the uterine cavity (3). There also is an extensive literature on cesarean section-related room air emboli (4–7) as well as room air embolus occurring during hysterectomy (8).

Gynecologic endoscopic procedures introduce the insufflating gas as another possible source of embolic phenomenon. The increased solubility of carbon dioxide, which is the commonly used gas for peritoneal distension and the only gas used for hysteroscopy, is expected to give a wide margin of safety compared with room air.

Functioning as medico-legal consultants and reviewers, the authors have collected a series of gas embolic accidents occurring during gynecologic endoscopy. These cases are presented to alert the gynecologist to these risks and to mention methods of early diagnosis and some maneuvers designed toward possible prevention of these catastrophic occurrences. Because specific therapy usually is within the provence of the anesthesia team, aspects of acute management will not be discussed.

CASE REPORTS

Case 1

A 27-year-old woman had sustained postpartum hemorrhage and a curettage was performed on two separate occasions. Subsequently, she developed amenorrhea. A hysterosalpingogram was diagnostic for severe uterine cavitary adhesions. Of note was significant intravasation of the dye during the study.

Received June 26, 1995; revised and accepted September 21, 1995.
* Department of Obstetrics and Gynecology, Thomas Jefferson University School of Medicine.
† Division of Reproductive Endocrinology, Department of Obstetrics and Gynecology, Pennsylvania Hospital.
‡ Department of Obstetrics and Gynecology, Graduate Hospital.
§ Reprints are not available.
‖ Department of Obstetrics and Gynecology, University of California Los Angeles School of Medicine, Cedars-Sinai Hospital.
¶ Department of Obstetrics and Gynecology, University of Washington School of Medicine.

A few weeks afterward, she was taken to the operating room, given general inhalational anesthesia, and her cervix was dilated to 7 mm in order to accommodate a hysteroscope. Uterine distention was created with saline delivered by gravity flow with the gradient of the pressure being approximately 1 m between the bag and the patient. She was in a "moderate" Trendelenburg position. The hysteroscope was removed to change over to an operating sheath; before reinsertion, the anesthesia team noted a sudden and marked decrease in heart rate, falling oxygen saturation levels, falling blood pressure, and cyanosis. Crepitance was detected in both inguinal areas and a femoral venous tap produced frothy blood. Gas was aspirated from both cardiac ventricles. Despite resuscitative efforts the patient died.

Case 2

A 27-year-old nulligravida was taken to the operating room for hysteroscopic examination because of abnormal uterine bleeding resistant to hormonal therapy. Under general anesthesia, cervical dilatation to 5 mm was achieved with some difficulty, after which the hysteroscope was inserted with carbon dioxide at 70 mm Hg pressure (flow rate unknown) delivered with a standard hysteroscopic gas source. Before any surgical manipulation other than inspection of the cavity, bradycardia developed at 9 minutes into the case (total anesthesia time) and atropine was given IV. Cardiac asystole ensued. Transesophageal ultrasonography demonstrated gas in all four chambers of the heart and a large atrial septal defect was noted. The vagina was packed to prevent further entry of room air (the presumed cause). She was comatose for 7 days after resuscitative efforts. The family and her medical attendants agreed on removal of life support systems but were over-ruled by the Medical Ethics Committee. Subsequently, she made a good recovery but sustained permanent brain damage.

Case 3

A 32-year-old patient was brought to the operating room for diagnosis and possible therapy after three consecutive pregnancy losses in the late first and early second trimesters. Laparoscopy, performed without apparent incident, disclosed a broad uterine fundus. After cervical dilatation, a hysteroscope with an operating sheath for optical scissors was introduced into the uterine cavity with the patient in the "usual Trendelenburg attitude." Carbon dioxide delivered at a maximum of 75 mL/min and at a maximum of 90 mm Hg pressure was used as distention. The septal incision was begun and the real-time video recording documents that at 35 seconds from the initial incision there was considerable bleeding. The hysteroscope was removed in order to clear the lens as the vision became obscured. Within 2 minutes, anesthesia alarm systems were activated; end tidal CO_2 plummeted, oxygen saturation decreased, and bradycardia was noted. Cardiac puncture produced gas but it was not analyzed. She was resuscitated and then developed disseminated intravascular clotting. The patient was transferred from the community hospital to a university center but then developed renal shutdown. Brain waves ceased and at 10 days life support systems were removed. Autopsy showed no thrombus formation in the heart and no atrial septal defects. No uterine perforation or laceration was seen.

Case 4

A 32-year-old patient complaining of chronic pelvic pain and abnormal uterine bleeding unresponsive to hormonal therapy was taken to the operating room for dilatation and curettage (D&C) and laparoscopy. She was 152.5 cm tall but weighed 109 kg. Under general anesthesia with intubation, a dilatation and curettage was performed without incident. An open-ended intrauterine cannula-manipulator was inserted into the uterus and the peritoneal cavity was distended with N_2O with a Verress needle placed via the umbilicus. The laparoscopy then was done in the usual fashion as a diagnostic procedure. Because of her obesity, the patient was placed in a steeper Trendelenburg position than usual. Vigorous manipulation with the uterine manipulator was necessary to help the surgeon visualize pelvic structures. At the end of the procedure, the gas was released from the abdominal cavity and the patient then was placed in the supine position. A suture was used to close the umbilical wound. As the patient was about to be extubated, the cardiac monitor alarm was activated, blood pressure fell, and a convulsion occurred. A millwheel murmur over the heart was detected and the patient was placed immediately on her left side. Needle aspiration of the heart showed frothy blood. Despite resuscitative efforts, the patient died within minutes. The eye grounds showed small bubbles in the retinal arteries.

At autopsy the heart was filled with gas in all four chambers and an atrial septal defect was seen. The meningeal arteries also had gas within the lumen. Before the heart was opened, a sampling of the gas was made and sent for analysis; the gas proved to be room air.

During the postmortem discussions, attention was paid to the fact that because of the patient's habitus, which necessitated a steep head-down position, the

handle of the uterine cannula was at least 26 cm above the level of the heart. It was proposed that with the active manipulation a passage was created such that room air was sucked into the uterine cavity and through a venous sinus opened by the D&C procedure and then into the major vessels leading back to the heart. Because of the low venous pressure and the Trendelenburg position, the bolus of air may not have traveled to the heart until the patient was returned to the supine position.

Case 5

The patient was a 48-year-old with a history of abnormal uterine bleeding, dysmenorrhea, and associated anemia nonresponsive to hormonal treatment. An endometrial ablation was planned. Under general inhalation anesthesia, cervical dilatation to 9 mm was performed and suction curettage was accomplished. As the resectoscope was inserted into the uterus but, before the stopcocks were opened to admit the distending liquid medium, end tidal CO_2 was noted to fall precipitously and cardiac arrest ensued. Resuscitation efforts were initiated and a central IV line was inserted. Approximately 15 mL of air was aspirated immediately, presumably from the right atrium via the superior vena cava. The patient died.

DISCUSSION

The prevalence of these tragic accidents is unknown. Incidents involving low degree of transient morbidity without permanent damage probably are unreported. We have collected 10 cases in all, and at least four represent embolism after D&C before or unassociated with hysteroscopy. Diagnostic hysteroscopy usually performed with small bore instruments requiring little or no cervical dilatation probably constitutes an immeasurably small risk. Dilatation to 10 mm to accommodate resectoscopes and operating sheaths is more likely to cause cervico-lower segment lacerations.

The literature on cesarean section-related room air embolus is understandable given the presence of large retroplacental sinusoids as conduits for entry of air into the circulatory system, especially when a deep Trendelenburg position is maintained and the placenta is elevated above the level of the heart, remembering that pressure within the inferior vena cava during systole is actually negative to room air pressure. The same phenomenon or mechanism of entry is probably operative during endoscopic procedures in which uterine venous channels become opened during hysteroscopic procedures, as a consequence of traumatic dilatation of the cervix, from a preceding D&C, or with entry of room air via a uterine open-ended manipulator during laparoscopy.

That insufflation of the peritoneal cavity with CO_2 is a safe procedure when performed with properly designed and functioning equipment was suggested by an early study by Lindemann et al. (9) in large German Shepherd dogs. But work by Root et al. (10) in dogs after an operative death during laparoscopy, suggested that CO_2 could be trapped within the portal system with a delayed embolus. In their patient, a patent foramen ovale was detected at autopsy. When the right side of the heart has its outflow tract occluded with gas bubbles, gas can appear in the left chambers of the heart as the right side pressure increases if there is a patent foramen ovale or a septal defect. However, in the case of Root et al. (10), as with two other cases reported in the literature (11, 12), a D&C was performed first, raising the possibility that the actual culprit was room air entering the venous circulation via the vagina and the uterus as opposed to a carbon dioxide pneumoperitoneum-related mechanism.

Because of its great solubility in blood CO_2 affords a great margin of safety in procedures where it is used to distend the relatively avascular peritoneal cavity and the uterus as well. Corson et al. (13) studied the cardiopulmonary effects of direct insufflation of CO_2 into the femoral vein of the adult ewe. Up to 90 mL/minute was delivered directly into the femoral vein. Provided that the animals were hyperventilated to prevent acidosis from this increased metabolic load, cardiopulmonary parameters remained within reasonable values. Pulmonary artery pressure did increase with time, and cardiac output increased as well. Oxygen saturation was within normal limits and in only one animal was any cardiac abnormality at all seen on the tracing, and this was in the form of premature ventricular contractions. Because these animals weighed between 24 and 32 kg, the conclusion was drawn that for the adult woman there was a large margin of safety for leakage of CO_2 into the vascular system with respect to disruption of metabolic parameters and cardiopulmonary function. So far as embolus was concerned, because CO_2 is so soluble in blood (54 mL/dL), true CO_2 embolus from therapeutic procedures should be very rare. But as shown by Baggish and Daniell (14), this safety margin can be overcome when high flow rates of CO_2 are delivered to the uterus as with a neodymium:yttrium-aluminum-garnet laser fiber where the cooling of the fiber with CO_2 is in the neighborhood of a 1 to 3 L/min flow rate.

Our cases 3 and 5 probably have similar mechanism of action in which the integrity of the endometrium was disturbed and air in the uterine cavity was forced into the circulatory system as a conse-

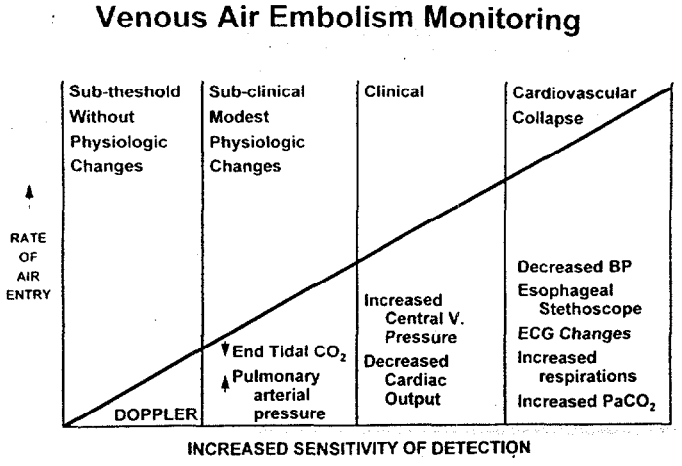

Figure 1 Methods of threshold detection of cardiovascular gas embolism versus increased rate of entry into the circulatory system.

quence of the piston-like action of the hysteroscope as it was inserted. Case 4 demonstrates that room air can enter the uterus in sufficient quantity through an open-ended cannula so as to cause death even without the postulated piston effect, although this is seemingly quite rare. The venous crepitance noted in case 1 can be explained by some of the air being trapped in the external iliac and femoral veins as a consequence of the Trendelenburg position.

Figure 1 demonstrates the sequence of events that occurs during an embolic episode. Although prevention of severe accidents is possible with early diagnosis of intravasation by inserting devices such as pulmonary artery catheters, this maneuver during gynecologic outpatient surgery probably would result in even more complications than it would prevent. Precordial stethoscopes are used frequently, but appearance of the characteristic millwheel murmur constitutes a late sign of impending cardiovascular collapse.

Although a precordial placement of a Doppler device sounds attractive, our experience with this technique has been unsatisfactory with many false-positive readings. This conclusion also has been reached in anesthesia texts devoted to this subject (15). The pulmonary tree is quite capable of dealing with small bubbles, and there is considerable difficulty in keeping the microphone exactly in the proper location.

As the right outflow tract of the heart begins to be occluded, there is precipitous drop of end tidal CO_2 as blood flow to the lungs decreases. All modern anesthesia machines record this measurement in a continuous fashion. Therefore, the earliest practical parameter to follow in order to avoid a calamity is one that can be monitored by an alert anesthesia team. Dropping end tidal CO_2 calls for immediate cessation of any gas delivery system, removal of uterine instruments, and occlusion of the open cervix and vagina as countermeasures. Whether a change in patient position is truly helpful is debatable.

Avoidance of deep Trendelenburg position seems desirable. A steep head-down tilt may be necessary for certain laparoscopic procedures, but it is not a necessity for hysteroscopy. Traumatic cervical dilatation cannot be avoided always, especially after prior cervical cone biopsy and other procedures. In such cases, use of osmotic dilatation preoperatively may be helpful. When it occurs, however, extension of the laceration into the lower segment of the uterus with tearing of the cervical vessels must be considered. Previous roentgenogram documentation of intravasation during infusion of dye also should alert the clinician to a situation in which the patient is operatively at greater risk. Excessive bleeding during hysteroscopy is an obvious sign that exchange of gas and blood by necessity must occur as the vessels are opened. We have been impressed with the size of blood vessels seen in submucous myomas and also with the vascular supply in cases of multiple uterine polyps. Prophylactic use of pitressin to induce contraction of the uterus and the vascular tree in that organ may offer some protection. A recent study showed decreased operative bleeding and reduced intravasation of liquid media during hysteroscopic surgery when pitressin was used prophylactically (16).

If a D&C is to be performed, a decreasingly used procedure given the direct visual accuracy afforded by hysteroscopy, consideration should be given to making it the last, rather than the first, procedure performed. When uterine manipulators are used with a channel for delivery of fluids for tubal perfusion, the simple expedient of capping the exposed opening should be followed. All these suggestions and admonitions (Table 1) follow logical, albeit unproven, reasoning and there is no documentation that these preventive measures actually will decrease an admittedly rare complication. Yet, the prevention of mortality in what generally is considered to be a minor operation through exercising increased

Table 1 Methods of Minimizing Room Air Embolism

- Careful monitoring of end tidal CO_2
- Nontraumatic cervical dilatation
  - Preoperative osmotic dilators in selected cases (nonparous patients, prior cervical surgery)
- Avoidance of steep Trendelenburg position
- Prophylactic dilute pitressin injection in cervix
- Lessen exposure of dilated cervix to room air
  - Remove weighted vaginal speculum
  - Place the last dilator used in cervix
  - Consider occluding the vagina with a sponge
- Fill hysteroscopic system with fluid before insertion into uterus

vigilance and by instituting easily performed safety measures is prudent. It is hoped that this report, if it has no other effect, will serve to alert the gynecologic surgeon to the risk and possible prevention of room air embolus during endoscopy.

REFERENCES

1. Bray P, Myers RAM, Cowley RA. Orogenital sex as a cause of nonfatal air embolism in pregnancy. Obstet Gynecol 1983;61:653–7.
2. Buxton CL. Death from air embolus. Report of a case following tubal insufflation. Am J Obstet Gynecol 1957;74:430–3.
3. Munsick RA. Air embolism and maternal death from therapeutic abortion. Obstet Gynecol 1972;39:688–90.
4. Karandy EJ, Dick HJ, Dwyer RP, McKinnon J. Fatal air embolism. A report of two cases, including a case of paradoxical air embolism. Am J Obstet Gynecol 1959;78:96–9.
5. Davies DE, Digwood KJ, Hilton JN. Air embolism during caesarean section. Med J Aust 1980;1:644–6.
6. Younker D, Rodriguez V, Kavanagh J. Massive air embolism during cesarean section. Anesthesiology 1986;65:77–9.
7. Lowenwirt IP, Chi DS, Handwerker SM. Obstet Gynecol Surv 1994;49:72–6.
8. Naulty JS, Beisel LB, Datta S, Ostheimer GW. Air embolism during radical hysterectomy. Anesthesiology 1982;57:420–2.
9. Lindemann HJ, Mohr J, Gallinat A, Buros M. Der Enflub von CO_2-Gas wahrend der Hysteroskopie. Geburtshilfe Frauenheilkd 1976;36:153–62.
10. Root B, Levy MN, Pollack S, Lubert M, Pathak K. Gas embolism death after laparoscopy delayed by "trapping" in portal circulation. Anesth Analg 1978;7:232–7.
11. Nichols SL, Tompkins BM, Henderson PA. Probable carbon dioxide embolism during laparoscopy; case report. Wis Med J 1981;80:27–9.
12. Yacoub OF, Cardona I Jr, Coveler LA, et al. Carbon dioxide embolism during laparoscopy. Anesthesiology 1982;57: 533–5.
13. Corson SL, Hoffman JJ, Jackowski J, Chapman GA. Cardiopulmonary effects of direct venous CO_2 insufflation in ewes. A model for CO_2 hysteroscopy. J Reprod Med 1988;33: 440–4.
14. Baggish MS, Daniell JF. Death caused by air embolism associated with neodymium:yttrium-aluminum-garnet-laser surgery and artificial sapphire tips. Am J Obstet Gynecol 1989;161:877–8.
15. Michenfelder JD. Air embolism. In: Orkin FK, Cooperman LH, editors. Complications in anesthesiology. Philadelphia: Lippincott Company 1983:268–73.
16. Corson SL, Brooks PG, Serden SP, Batzer FR, Gocial B. Effects of vasopressin administration during hysteroscopic surgery. J Reprod Med 1994;39:419–23.

because of the potential for this complication they regard the use of saline, rather than air, should be mandatory when locating the epidural space in patients who are to undergo ESWL.

A recent report has further implicated the use of air to locate the epidural space with the development of neurological complications.[2] A 52-year-old woman was described in whom a lumbar epidural was inserted to treat chronic back pain. She subsequently developed paraplegia and an immediate CAT scan demonstrated significant displacement of the cauda equina by air within the epidural space. In this case, it was proposed that nerve root compression was the cause of the neurological sequelae, and that resorption of the air resulted in the gradual resolution of her symptoms and signs. This scenario took place in the abscence of ESWL, and could provide an alternative mechanism for the findings in the report of Deam and Scott.

Although rare, a number of complications have been described when employing the loss-of-resistance-to-air technique, including incomplete analgesia,[3] subcutaneous emphysema[4] and venous air emboli.[5] We suggest that in the case reported by Deam and Scott, neurological damage may have resulted from nerve compression by extradural air rather than from ESWL *per se*. For this season, during the provision of analgesia for ESWL, the use of saline rather than air when locating the epidural space would appear to further minimise the risk of neurological complications.

A. CONE
S. STOTT
Royal Perth Hospital, Perth, W.A.

References

1. Abbott MA, Samuel JR, Webb DR. Anaesthesia for extracorporeal shock wave lithotripsy. Anaesthesia 1985; 40:1065-72.
2. Nay PG, Milaszkiewicz R, Jothilingam S. Extradual air as a course of paraplegia following lumbar analgesia. Anaesthesia 1993; 48:402-4.
3. Dalens B, Barin JE, Huberer JP. Epidural bubbles as a cause of incomplete analgesia during epidural anaesthesia. Anesth Analg 1987; 66:679-83.
4. Rosenberg B, Tischler S, Glick A. Abdominal subcutaneous emphysema: an unusual complication of lumbar epidural block. Can J Anaesth 1988; 35:325.
5. Jackson KE, Rauck RL. Suspected venous air embolism during epidural anaesthesia. Anesthesiology 1991; 74:190-1.

Cardiac Arrest During Endometrial Ablation

I read with interest the account of Andrew Michael's experience of air embolism during endometrial ablation[1] and would like to report another suspected case.

A 36-year-old previously well woman presented to the Day Surgery Unit for endometrial ablation. She had had one uncomplicated general anaesthetic 10 years ago. No premedication was given. Anaesthesia was induced with propofol 150 mg, fentanyl 100 mcg and droperidol 1 mg, and intubation of the trachea was facilitated with vecuronium 6 mg. The lungs were ventilated with nitrous oxide, oxygen (FiO_2 0.3) and isoflurane 0.7% min. with a total fresh gas flow of 2 l/min. Monitoring consisted of ECG, noninvasive blood pressure (BP), pulse oximetry and capnography ($EtCO_2$). Her BP was stable at 120/80, heart rate 50-60 bpm, O_2 saturation 98% and $EtCO_2$ 32 mm Hg. The patient was placed in the lithotomy position with slight head-up tilt. Endometrial ablation was performed with a loop resectoscope, 1.5% glycine and an Ellik evacuator used as in transurethral resection of prostate surgery.

About 15 minutes into the procedure the SpO_2 suddenly fell to 92% and $EtCO_2$ to 18 mm Hg. The BP at this stafe was 90/60 and pulse was 60 bpm. Auscultation of the heart and chest revealed a mill-wheel murmur and normal breath sounds. Despite immediate manual ventilation with 100% the O_2 saturation and $ETCO_2$ continued to decrease. The procedure was abandoned and the patient placed in the left lateral position. A 14 gauge cannula was inserted peripherally and 1 litre of Hartmann's solution infused. A triple-lumen catheter was inserted into the right internal jugular vein but no air was aspirated.
At this stage the SpO_2 was 68% $EtCO_2$ was 8 mm Hg pulse was 30 bpm and the BP was unrecordable. Asystole followed and external cardiac massage was immediately commenced while atropine 1200 mcg and adrenaline 1 mg were given intravenously. After about 30 seconds the pulse had returned to 140 bpm and BP was restored to 140/80. The peak inspiratory airway pressure was 40 cm H_2O and auscultation of chest revealed mild bronchospasm. Two heart sounds were audible with no other added sounds.

The patient was transferred to the Intensive Care Unit for monitoring and extubated one hour post cardiac arrest with no subsequent sequelae.

As in Dr Michael's case, air embolism has been reported previously in association with loop diathermy endometrial ablation, possibly from air in the glycine bag or tubing. One episode occurred in a patient breathing spontaneously and it was suggested that an anaesthetic technique employing positive pressure ventilation may be preferred.[2] Several episodes of fatal air embolism have also been recorded, two of these during endometrial ablation using neodymium:

yttrium-aluminium-garnet laser.[3] This was attributed to installation of gas used for cooling the sapphire tip attached to the laser. Two other episodes have been reported during transurethral resection of prostate where the air may have entered through the Ellik evacuator, but could have also entered through a faulty connection or empty irrigant container.[4,5]

In this case I believe air was introduced via the Ellik evacuator as the incident occurred after 15 minutes and the first 3 litre bag of glycine still contained 2 litres (deficit 200 ml). Other possibilities include an acute vasovagal episode, allergic reaction or excessive absorption of glycine, all of which can present in a similar fashion. The presence of a mill-wheel murmur makes air embolism the most likely diagnosis. Anaesthetists should be aware that this procedure carries a risk of significant air embolism.

D. EUGSTER
Royal Hobart Hospital,
Hobart, Tas.

References

1. Michael A. Endometrial ablation and air embolism (letter). Anaes Intens Care 1993; 21:475.
2. Wood SM, Roberts FL. Air embolism during trancervical resection of endometrium (letter). Br Med J 1990; 300:945.
3. Baggish MS. Death caused by air embolus associated with neodymium:yttrium-aluminium-garnet laser surgery and artificial sapphire tips. Am J Obstet Gynecol 1989; 161:877-878.
4. Hofsess DW. Fatal air embolism during transurethral resection. Urol 1984; 131:355.
5. Vacanti CA, Lodhia KL. Fatal massive air embolism during transurethral resection of the prostate. Anesthesiology 1991; 74:186-187.

Selectatec gas leak

I wish to report an incident which involved a leak at the connection between a Cyprane Selectatec manifold, mounted on a newly acquired Ulco Minor anaesthetic machine and a Penlon PPV Sigma isoflurane vapouriser.

Two children were scheduled to be anaesthetised for CT scan. The anaesthetic machine was checked with a Fluotec 3 halothane vapouriser in place. The same anaesthetic technique was used for both patients. The first procedure, which lasted about 15 minutes, was uneventful. The second patient weighed 15 kg and, in addition to the CT scan, was to have auditory evoked potentials recorded as part of assessment for suitability for insertion of cochlear implant. An inhalational induction using halothane and nitrous oxide in oxygen was performed and a laryngeal mask airway inserted. Monitoring included capnography and nitrous oxide and oxygen analysis using side-stream gas sampling (Datex Oscar Oxy™). The halothane Fluotec 3 was then removed and replaced with the isoflurane vapouriser, which locked easily onto the Selectatec. This Penlon vapouriser had been used without problem on other machines in the department but, unknown to the anaesthetist, not with this particular machine.

The CT scan took about 15 minutes, the child and anaesthetic machine were then moved into an adjacent room for the evoked-potential recording. The monitors were reconnected and after time had been allowed for re-equilibration it was noticed that the displayed value for the inspired concentration of oxygen (F_IO_2) was 21%. The rotameters, as set, should have given F_IO_2 33%. The side-stream port was disconnected and the accuracy of the oxygen analyser confirmed using room air. The presence of a large leak was suggested by the failure of the reservoir bag of the T-piece breathing system to inflate when the distal end was occluded. It was assumed that air was being entrained. As the source of the leak was not immediately apparent the nitrous oxide was discontinued and oxygen and isoflurane administered, with an increased fresh gas flow, to ensure an adequate F_IO_2. The maximum F_IO_2 achieved was 57% ($F_IN_2O=0$), with a fresh gas flow of 8 l min^{-1}. The isoflurane concentration set on the vapouriser was 2%. $PE'CO_2$ remained at 48mm Hg, with a respiratory rate of approximately 28.min^{-1} but the child's respiratory pattern became more irregular, with occasional sighs, consistent with a decreasing depth of anaesthesia. After approximately 15 minutes, as the recording was nearing completion, the child woke suddenly and removed the laryngeal mask.

The machine was examined after the procedure. With less background noise a leak was clearly audible at the proximal connection between the Selectatec manifold and the Penlon vapouriser. There was no leak without a vapouriser on the machine nor with the Fluotec 3 on the Selectatec. The machine and vapouriser were removed from service and the supplied contacted. He examined the machine and found that the rear portion of the Penlon vapouriser, which contains the Interlock mechanism, overlapped the back bar. The difference in height between the machine back bar and the Selectatec was lifting the vapouriser off the manifold, preventing an adequate seal between the O ring of the Selectatec manifold and the vapouriser. The Fluotec 3 vapourisers do not have the Interlock system and do not overlap the back bar when located on the manifold. The manufacturer of the anaesthetic machine was informed of the problem and has modified the machine by milling out a length along the top surface of the

Anaesth Intens Care 1998; 26: 112-114

Venous Embolism of Diathermy Evolved Gases Complicating Endometrial Ablation using Glycine Irrigant

P. D. HEBBARD

Department of Anaesthesia, Wangaratta District Hospital, Wangaratta, Victoria

SUMMARY

A case is reported of venous gas embolism in a 44-year-old woman undergoing hysteroscopic endometrial ablation using glycine irrigation without gas insufflation. The postulated source of gases are the vapour and combustion products produced by the diathermy.

Key Words: EMBOLISM: venous, air, hysteroscopy, diathermy

Venous gas embolism under anaesthesia is normally associated with the entry of insufflating gas or air into the bloodstream. In the following report however no gas insufflation was used.

CASE HISTORY

A 44-year-old female of ASA grade 1 presented for hysteroscopic endometrial ablation. Planned spinal anaesthesia was performed. The height of the spinal blockade, however, was inadequate for the procedure and anaesthesia was supplemented with a bolus of propofol followed by placement of a laryngeal mask. Anaesthesia was maintained by isoflurane 1 to 2% in 35% oxygen and 65% nitrous oxide with spontaneous ventilation.

Endometrial ablation was commenced after positioning the patient in the lithotomy position. Initial examination and hysteroscopy showed a markedly anteverted uterus. Warmed glycine 1.5% was used as an irrigant at a pressure of 75 to 80 mmHg via a controlled pressure irrigation pump (Zimmer, Blackburn, Vic.). Surgery proceeded for 30 minutes although the surgical field was impaired more than usual by bubbles derived from the diathermy of tissues. The diathermy electrode used consists of a metal ball rolling on a fixed axis between two supporting wires (Olympus Australia Ltd). The ball was otherwise free in the uterine cavity. There was no gas cooling or insufflation system.

Forty minutes after the commencement of general anaesthesia, the pulse oximeter indicated a sudden decrease in oxygen saturation (SpO_2) from 97% to 92%. The patient's pulse was easily palpable with systolic blood pressure unchanged and normal ventilatory pattern. The end-tidal carbon dioxide ($P_{ET}CO_2$) was noted to be 24 mmHg during spontaneous ventilation. On auscultation of the heart a tinkling sound similar to that of an ornamental fountain was heard widely over the precordium. A diagnosis of venous gas embolism was made, the patient commenced on 100% inspired oxygen and surgery was ceased. The SpO_2 rose promptly to 98% and other observations remained stable. Review of the automatically recorded observations showed a gradual fall in the $P_{ET}CO_2$ over approximately ten minutes before the drop in saturation, this had been accompanied by an increase in pulse rate from 62 to 75 beats per minute and an increase in diastolic blood pressure from 45 to 60 mmHg. The heart sounds remained abnormal for two to three minutes gradually becoming less distinct. As haemostasis was good and surgery completed, no further diathermy was used, and measured glycine loss was 300 ml. Anaesthesia was continued with isoflurane in oxygen for 30 minutes and then the legs lowered from lithotomy and isoflurane discontinued. The patient regained consciousness uneventfully.

DISCUSSION

Venous gas embolism has been reported in many cases in association with direct insufflation of gas into the uterus. A Medline search failed to find any report of clinical venous gas embolism in association with diathermy evolved gases.

The diagnosis in this case was suggested by the unusually low $P_{ET}CO_2$ and confirmed by the distinctively abnormal cardiac sounds. CO_2 measured at the upper airway or distal anaesthetic circuit is influenced by the respiratory pattern. During anaesthesia with

*F.A.N.Z.C.A., Anaesthetist.

Address for Reprints: Dr Peter Hebbard, 134 Templeton Street, Wangaratta, Vic. 3677.

Accepted for publication on September 11, 1997.

spontaneous ventilation the length of expiration may be insufficient to allow the end-tidal measurement to reflect a true mixed alveolar gas. This case demonstrates the effectiveness of airway CO_2 monitoring in spontaneously ventilating patients.

In the absence of altered inspiratory gas a reduction in the $P_{ET}CO_2$ is caused by a change in the balance between minute ventilation and the quantity and distribution of CO_2 presented to the lung in the blood via the pulmonary artery. This balance may be altered by sudden reductions in cardiac output, major changes in regional perfusion of the lung, reduced body production of CO_2 or increased ventilation.

Gas emboli occupy superior parts of the pulmonary arterial tree causing a reduction in perfusion to the upper zones and therefore increased deadspace ventilation. In the absence of a severe reduction in cardiac output this is probably the main mechanism of reduction of $P_{ET}CO_2$ in gas embolism.

Cardiac output may be reduced due to the displacement of blood from the heart and altered cardiac valve function in the presence of gas. Ineffective cardiac pumping is seen in dogs with a bolus of 3 to 8 ml/kg air[1,2]. In the case reported here, a modest reduction in cardiac output was manifest as an increase in diastolic blood pressure and pulse rate, reflecting reflex changes to maintain blood pressure in the face of decreased cardiac output. In the event of cardiac output decreasing suddenly, there is an immediate fall in the total quantity of CO_2 presented to the lung through the pulmonary circulation. This returns to normal once venous CO_2 concentration increases to maintain body CO_2 equilibrium. In the absence of a catastrophic fall in cardiac output the magnitude of this effect is relatively small as only approximately 10% of the available CO_2 in the blood is normally transferred to the alveolus. Any fall in the partial pressure of alveolar CO_2 will cause extra readily available CO_2 to pass into the alveolus limiting any further fall in P_ACO_2. A substantial decrease in cardiac output will also produce an increase in deadspace ventilation with upper zones in the lung receiving negligible perfusion compared with lower zones.

Adornato et al[1] noted two distinct abnormalities in cardiac auscultation during air embolism in dogs. At mean infusion rates of 1.7 ml/kg/min or a bolus of 25 ml (1.2 to 2.5 ml/kg) a tinkling drum-like sound was heard. At mean infusion rates of 1.96 ml/kg/min or a bolus of 200 ml (9.5 to 20 ml/kg) the classic millwheel murmur was heard. The 200 ml bolus was uniformly fatal. The abnormal heart sounds heard in this case had a distinct tinkling quality and probably correspond to the drum-like sound heard in dogs. Although it is useful to confirm the diagnosis, the late and unpredictable onset of this sound restricts its usefulness.

In this case the only possible source of gas was bubbles evolved from the endometrial diathermy. These obscured the surgical field at times due to inadequate clearance through the endoscopic sheath. The pronounced anteversion of the uterus caused these bubbles to accumulate in the superior part of the uterine cavity under pressure from the infuser controlling the glycine irrigant. This created a strong gradient favouring the entry of gas into the circulation. Gas evolved from a diathermy contains water vapour and products of combustion. The water vapour will rapidly condense leaving CO_2 as the major embolizing gas. Some particulate matter (smoke) may also be present from the combustion. Fine particulate matter is filtered in the pulmonary circulation and removed by phagocytosis.

Most gas emboli reaching the pulmonary circulation will pass through the pulmonary-alveolar membrane and be eliminated. Intravascular bubbles are not inert and cause platelet and leucocyte aggregation, disrupt endothelium and activate the complement and kinin systems[3]. This coating of blood components and damaged endothelium will slow transfer into the alveolar gas and cause residual effects after clearance of bubbles. Air bubbles persist for a mean of 24.5 to 43 minutes after infusions of air at 0.1 to 0.25 ml/kg/min for 15 minutes in dogs[4].

CO_2 is absorbed more rapidly than air as it is 34 times more soluble in blood[5] and therefore the time to clear bubbles will be much shorter. The lethal venous dose of CO_2 is approximately five times that of air in dogs[2] with an air LD50 of 5.1 ml/kg and CO_2 LD50 of 25 ml/kg. In addition, dogs surviving a near fatal CO_2 bolus recovered much more rapidly than those dogs that received a near-fatal bolus of air.

Intravascular gas may distribute along either a flow or a gravity gradient. As the patient was in lithotomy the legs may have provided a large reservoir of venous gas due to retrograde flow of gas into the lower limb veins. The patient was therefore maintained in lithotomy position for 30 minutes while administering 100% oxygen to promote absorption. The legs were slowly lowered while the heart was auscultated, however no further changes were heard and there were no changes in $P_{ET}CO_2$ or SaO_2.

This patient demonstrated several features of gas embolism: $P_{ET}CO_2$ was reduced and a characteristic change in cardiac sounds occurred. These features rapidly resolved with cessation of the inflow of gas. Further support or treatment was unnecessary. The

appearance of changed cardiac sounds however, means that the patient may have been close to the limit of physiological reserve. Caution is advised when using diathermy inside confined spaces in the body, particularly if the bubbles are not quickly cleared through the endoscope sheath.

REFERENCES

1. Adornato DC, Gildenberg PL, Ferrario CM, Smart J, Frost EAM. Pathophysiology of intravenous air embolism in dogs. Anesthesiology 1978; 49:120-127.
2. Graff TD, Arbecast NR, Phillips OC, Harris LC, Frazier TM. Gas embolism: A comparative study of air and carbon dioxide as embolic agents in the systemic venous system. Am J Obst Gyne 1959; 78:259-265.
3. Orebaugh SL. Venous air embolism: clinical and experimental considerations. Critic Care Med 1992; 20:1169-1177.
4. Butler BD, Luehr S, Katz J. Venous gas embolism: Time course of residual pulmonary intravascular bubbles. Undersea Biomed Res 1989; 16:21-29.
5. Kunkler A, King H. Comparison of air, oxygen and carbon dioxide embolization. Ann Surg 1959; 149:95-99.

Complications of Hysteroscopy—Their Cause, Prevention, and Correction

Franklin D. Loffer, M.D.

Abstract

Complications of hysteroscopy occur more frequently in operative than in diagnostic cases. Problems related to uterine distension are common, usually preventable, and potentially extremely serious. Perforation of the uterus may occur during hysteroscopy but do not always cause significant problems. In procedures of high risk for perforation the use of mechanical energy is safer than either laser or electrical energy. Laparoscopy and ultrasonography have some limited use in facilitating operative hysteroscopic procedures. Most complications occur during the hysteroscopic surgical procedure. However some problems may not be apparent until the post operative period.

Complications of hysteroscopy may occur when contraindications are ignored,[1] for example, when proper surgical techniques are not followed or equipment is used in an inappropriate fashion. They also may occur when the procedure has been performed correctly on well-chosen patients.

Complications are not common, and many large series report no serious problems. When community wide experience is reviewed, complication rates exceed those published by experts.[2] Therefore it is difficult to discuss their true frequency, since most go unreported or, if reported, are included in case reports without reference to the number of similar cases done without difficulty.

When complications do occur, most arise during operative rather than diagnostic hysteroscopy. Certain aspects of the procedures are more likely to be associated with difficulties than others.

Anesthesia

A paracervical or intracervical block is commonly used during diagnostic hysteroscopy, although the surgery can be performed without anesthesia. Operative hysteroscopy may be done with local infiltration into the cervix and paracervical area, but patients commonly receive general or regional anesthesia. Hysteroscopy does not add any special risks to these anesthetic techniques except possibly the additive effect of the distending medium.[3]

Failure to Accomplish Hysteroscopy

Inability to introduce the hysteroscope into the uterine cavity and to obtain an adequate view should be considered a complication. Failure to accomplish the procedure will occur in all large series regardless of

From the Department of Gynecologic Endoscopy, Maricopa Medical Center, Phoenix, Arizona.

Address reprint requests to Franklin D. Loffer, M.D., Department of Gynecologic Endoscopy, Maricopa Medical Center, 3410 North 4th Avenue, Phoenix, AZ 85013; fax 602 241 1917.

the expertise of the surgeon. As experience is gained, however, the problem diminishes.[4]

Complications Associated With Distending Media

Uterine distention for panoramic hysteroscopy is necessary for two reasons. First, it must create a cavity by overcoming the resistance of the uterine musculature. Second, it has to be of sufficient pressure to prevent bleeding into the distending medium, which can obscure visualization. Complications are associated primarily with intravasation of distending media. They may occur during both diagnostic and surgical hysteroscopies and result from excessive absorption of or idiosyncratic reactions to the medium. Unfortunately, the perfect medium does not exist.

Carbon dioxide (CO_2) is the usual medium for diagnostic office hysteroscopy. It is clean and safe, but has the disadvantage in operative cases of creating bubbles that may obscure vision. In addition, its low viscosity makes uterine distention during surgical manipulation difficult because of leakage around the gaskets of the instruments.

Thirty-two percent dextran 70 (Hyskon; Kabi Pharmacia, Piscataway, NJ) allows for good visualization since it is not miscible with blood. Because of its high viscosity, administration requires increased pressure, but leakage is seldom a problem. However, complications due to dextran 70 may be difficult to manage.

Low-viscosity fluids are the media of choice for most operative procedures because they are ideally suited for the continuous-flow hysteroscope. The most commonly used solutions are 5% or 10% dextrose and water, Ringer's lactate, normal saline, and the urologic distending media 1.5% glycine, 3% sorbitol, and 2.7% sorbitol with 0.54% mannitol. A 5% mannitol solution has also been suggested as a distending medium, but no reports of its use in hysteroscopy are available. Water should not be used since it causes hemolysis. Problems with intravasation of all these media have been recognized by urologists for over 40 years, but only more recently by gynecologists, and are well summarized elsewhere.[5]

Intrauterine Pressure

Three factors affect intrauterine pressure when creating uterine distention: the pressure of the infusion source forcing the medium through the tubing and hysteroscope into the uterus, the rate at which the medium flows into the uterus to equalize the pressure with the infusion source, and the rate at which the medium escapes from the uterus and must be replaced to maintain intrauterine pressure. Infusion pressure may be created by a hand-held syringe for dextran 70 and low-viscosity fluids; by mechanical insufflators for CO_2, dextran 70, and low-viscosity fluids; and by gravity for low-viscosity fluids. Transmission of this infusion pressure into intrauterine pressure is limited by the rate of flow of the distending medium. The rate at which a medium can be delivered into the uterine cavity is determined by the viscosity of the medium and the diameter of the tubing and hysteroscope channels.[6]

Intrauterine pressure is decreased by the medium's outflow, which is not replaced. The medium may be lost due to leakage around the cervix, the outflow from continuous-flow equipment, passage into the abdomen through the fallopian tubes, and intravasation. Its passage into the vascular system may cause serious complications.

Infusion pressures of approximately 60 to 75 mm Hg are generally more than sufficient for distention and result in intrauterine pressures of approximately 10 to 15 mm Hg less.[7] True intrauterine pressures are not measured by any available insufflating systems. Even if this were possible, the hysteroscopist should use the least infusion pressure necessary to distend the uterus and prevent bleeding into the medium.[8] Higher infusion pressures do not provide better visualization except when the diameter of the tube or instrument is too small or the viscosity of the medium is too high to allow an adequate flow rate at usual pressures. These exceptions apply to low-viscosity fluids used with a 5-mm diagnostic sheath and to dextran 70. For both media, once good visualization is obtained, the infusion pressure should be reduced to the lowest level that maintains visualization.

Intravasation

Intrauterine pressure is the single most important variable the surgeon can control to prevent intravasation. A value greater than the mean arterial pressure significantly increases the risk of intravasation.[9] Other factors also must be considered, for example, hysteroscopic procedures that open vascular channels and the length of the surgery.

Intravasation can occur when high pressures are used even when the endometrial surface is intact. However, procedures such as resection of myomata in the endometrium, lysis of intrauterine adhesions, and

transection of septum increase the chance of intravasation since they may open vascular channels. The number and size of openings are generally beyond the surgeon's control, but the increased risk in these operations must be recognized. Intravasation can also occur if there has been a partial perforation of the uterus, such as when a false passage has been created or when cervical dilatation has caused a tear in the upper cervical or lower uterine segment. Therefore, even diagnostic hysteroscopies and cases of rollerball ablation may carry this risk.

Administration of oxytocin[10] and vasopressin[11] with pretreatment with a gonadotropin-releasing hormone agonist[12] are suggested to be of value in preventing intravasation. Vasopressin can be associated with hypertension, hypotension, and cardiac function abnormalities,[13] however, and its use to reduce the risk of intravasation was severely criticized because of its antidiuretic effect.[14] Hysteroscopists should balance the advantages of this agent against its possible risks.

The length of cases is important, since, all other factors being equal, more intravasation will occur in longer cases than in shorter ones. The surgeon has limited control over this feature except to operate as quickly as is safe and to discontinue the procedure if the amount of intravasation becomes a problem.

Even when intrauterine pressures are controlled, the risk of excessive intravasation exists.[10] Therefore, the most important technique to determine if it is occurring is to monitor inflow and outflow of the distending medium.[8] This is not possible with CO_2. Although there are technical problems with dextran 70 and the viscous fluids, every effort should be made to ensure an accurate measurement.

Complications Associated With Specific Media

Carbon Dioxide

Carbon dioxide is a very safe distending medium since it is rapidly absorbed into the blood and readily released during pulmonary ventilation.[15] No other gas can be used safely for this purpose. Little potential risk exists from the intravasation of CO_2 if a maximum pressure of 100 mm of Hg and a maximum flow rate of 100 ml/minute are not exceeded. When CO_2 hysteroscopy is performed during pregnancy, the maximum pressure and flow rate should be reduced to 50 mm Hg and 60 ml/minute.[16, 17] Higher pressure and flow rate may cause a large volume to be intravasted, which may result in metabolic changes, embolization, and death.[15] The metabolic changes that may occur from excessive CO_2 are an increase in partial pressure of CO_2 and a decrease in partial pressure of oxygen, which result in metabolic acidosis and cardiac irregularity.[18–20] Embolization of CO_2 in small volumes is not dangerous. It occurs in 52% of patients undergoing a hysteroscopy with the gas as distending medium.[21] Intravasation is more frequent at instillation pressures of 60 to 120 mm of Hg (57.7%) than when pressures are less then 60 mm Hg (14.7%).

Despite the fact that thousands of hysteroscopies are performed with CO_2 without difficulty, significant complications have been reported.[3, 15, 22, 23] All of these events occurred with excessive pressure and/or flow rates. Most often, equipment was used that was not designed for hysteroscopy. In one woman the instrument, flow rate, and pressure were appropriate, but the equipment malfunctioned and delivered a higher flow rate and pressure.[3] These potentially catastrophic problems are prevented by using correct instrumentation that is checked periodically for accuracy and proper functioning.

Treatment of excessive CO_2 intravasation consists of immediate cessation of hysteroscopy, ventilation of the patient, and pulmonary and vascular supportive measures. If significant embolization has occurred with accumulation of undissolved CO_2 in the right side of the heart, blood flow may be increased by turning the patient on her left side.

Another complication of CO_2 hysteroscopy is rupture of a blocked fallopian tube. In one report this was related to the excessive pressure.[24] Hypothermia as occurs during laparoscopy[25] would appear to be unlikely, since much smaller volumes of gas are used.

Dextran 70

Complications of dextran 70 intravasation are pulmonary edema, coagulopathies, and allergic reactions including anaphylaxis. Some reports suggest that the pulmonary edema is related to a direct toxic effect on the pulmonary vasculature,[26–29] although no good evidence exists to support this. Authors who believe there is a direct toxic effect on the lungs do not clearly state the amount of dextran 70 that was retained by the patient, and a direct toxic effect has been challenged.[30–32] Many surgeons believe the suggested noncardiogenic pulmonary edema is related to the osmotic properties of dextran 70.[30–36] The agent is a potent plasma expander, and for every 100 millimeters infused, the plasma

volume is expanded by an additional 860 ml.[33] The significance of this can be demonstrated by the fact that infusion of 350 ml of dextran 70 intravascularly in a 60-kg patient will essentially double her plasma volume.[34]

Intravascular coagulopathies[28–30, 32, 36] may be related to an idiosyncratic reaction to dextran 70, but are more likely related to the volume intravasated, since the agent's anticlotting properties are related to the amount used. Anaphylactic reactions have been reported,[37–40] as well as minor allergic reactions manifested by skin changes.[36, 41]

Pulmonary and coagulopathy complications of dextran 70 are prevented by limiting intravasation with careful monitoring of the intake and output to assess the amount that is retained. The manufacturer states that 500 ml is the maximum amount that should be used. It also states that hysteroscopies should be limited to 45 minutes. This time limitation does not seem necessary as long as the amount retained by the patient is known. It is the amount intravasated through the damaged endometrium and myometrium rather than the amount of dextran 70 used that is important.[42] Nevertheless, the recommendations are important, since when the suggested volume was exceeded, there was a 1.1% frequency of pulmonary edema and a 0.5% frequency of disseminated intravascular coagulation.[36]

Another problem with this viscous material is not knowing the intrauterine pressure created, because a very high pressure is necessary to ensure an adequate flow. This may translate into very high uterine pressures. Once distention has been achieved with dextran 70, only minimal additional pressure is necessary to obtain adequate visualization.

Treatment of pulmonary edema resulting from dextran 70 intravasation is oxygen, ventilatory support, and attempted diuresis. Diuresis is difficult, as the agent's half-life is several days.[43] Since coagulopathies very likely are in part dose related, prevention is by limiting the amount intravasated. It is theoretically possible to prevent serious allergic reactions with administration of a hapten inhibitor.[44] Management is supportive until the problem has self-corrected, although epinephrine and possibly hydrocortisone or antihistamines have been suggested.[34]

Low-Viscosity Fluids

Low-viscosity fluids are the most commonly used distending media for operative hysteroscopy because they are ideally suited for the continuous-flow hysteroscope. They must be electrolyte free when used with electrical instruments, or the electrical current will be dissipated away from the electrode, rendering it ineffective. Complications related to these agents are primarily those of excessive intravasation. When excessive amounts are retained, edema with or without pulmonary edema may occur.[45–54] If the infusing medium is dextrose and water, glycine, or sorbitol, hyponatremia or hypo-osmolarity may occur,[47, 48, 55–60] since they are not isotonic electrolyte solutions. The addition of mannitol to sorbitol may help prevent the problem but is not fully protective[61]; 5% mannitol may be preferable.[62]

If electrical equipment is not used, hyponatremia and hypoosmolarity can be prevented by distending with normal saline or Ringer's lactate; however, pulmonary edema is still a potential problem with these media. Fluid overload occurred in 1.1% of patients undergoing the high-risk procedure of resection o submucous myomas.[45] A rate of 0.34% in 1988 dropped to 0.14% in 1991 and returned to 0.20% in 1993 according to surveys of general gynecologists.[63–65]

Minimal changes in serum electrolyte levels may result in inconsequential problems,[66] and significan changes may result in catastrophic problems. It is no the hyponatremia per se that is serious, but the cere bral edema that occurs as the brain absorbs water i an attempt to become isosmotic with the remainder c the interstitial fluid and vascular system. Cerebra edema results in increased intracranial pressure Subsequent herniation of the brain stem may result i death.[67, 68] Young women appear to be at greater ris for this event than postmenopausal women or men, perhaps because their endogenous progesterone inhibi the enzyme sodium-potassium adenosine tripho phatase.[70, 71] Excessive intravasation of glycine ma lead to hyperammonemia encephalopathy, whic causes transient blindness, muscle aches, and mer ory loss.[5]

Some surgeons measure serum electrolytes ro tinely, but most do not consider this to be necessary Excessive intravasation is prevented by using as lit pressure as possible to create adequate distension, a by accurately monitoring intake and output.

When excessive intravasation is recognized d ing the procedure, treatment is to discontinue the ca as soon as safely possible. If the problem is not r ognized until it has occurred, it should be treated an emergency. Patients should be maintained on hi oxygen levels. Electrolytes should be measured a intravenous furosemide administered immediate however, furosemide would not appear to be necess

if the distention fluid was 5% mannitol solution. A Foley catheter will help to monitor diuresis. A brisk diuresis should occur that rapidly elevates the serum sodium level depressed by the intravasation. If this does not happen, administration of hypertonic 3% sodium chloride solution can be considered. No treatment is necessary for hyperammonemia encephalopathy, as the disorder self-corrects in a matter of time.

Inadequate Visualization

Poor visualization is much less of a problem with the continuous-flow instrumentation now available. Even without this equipment, the uterine cavity can be cleaned to a satisfactory point in most cases.[73]

Air and Gas Emboli

Intravascular air embolization can occur whenever uterine veins are open during surgery and the air pressure is greater than venous pressure.[74] Although uncommon, it is a risk of hysteroscopic surgery when the patient is in Trendelenburg position and the uterus is elevated over the heart. Spontaneous ventilation may increase this risk. It may also occur from bubbles in the inflow tubing or gases that form during the procedure.[75]

Early therapy of an air embolism is mandatory. Hyperbaric oxygen can be used to treat patients who survive the initial insult. Hyperbaric consultation can be obtained without charge 24 hours a day through the Divers Alert Network, Duke University, Durham, NC, (919) 684-8111.[76]

Another cause of gaseous embolization that has resulted in death and serious injury is using air, CO_2, and nitrous oxide with coaxial fibers of the neodymium:yttrium-aluminum-garnet (Nd:YAG) laser.[77,78] When gas is used with these fibers in the uterus with or without a sapphire tip, death and serious injury can occur because the high flow rate that is delivered does not vary with the pressure created. Only a liquid should be introduced into the uterus through the coaxial laser fiber.

Traumatic Injuries of the Uterine Cervix and Fundus

Traumatic injuries related to hysteroscopy are more common during operative than diagnostic procedures because more dilatation is necessary for inserting the larger-diameter operative hysteroscopes. These may be nuisance injuries, such as cervical mucosal lacerations from the tenaculum, or they may cause intraoperative or postoperative problems, such as partial uterine perforation. They may also limit the ability to complete the procedure when adequate distention cannot be achieved after a complete uterine perforation.

Cervical tearing by the tenaculum during dilatation is a common event without significant sequelae. It occurs most frequently with a single-tooth tenaculum. A Jacob's tenaculum offers a firmer grip and is less likely to pull free and result in injury to the cervical mucosa. Treatment is suturing if bleeding is brisk.

Laceration of the cervical canal and lower uterine segment may also occur during dilatation. It results primarily when dilatation is required to accommodate larger instruments such as the resectoscope. These lacerations can frequently be seen in the lateral margins of the canal. They very occasionally may cause postoperative bleeding, both immediate and delayed. Correction is generally not necessary, although heavy bleeding could be coagulated with a resectoscope electrode. More commonly they become a source for fluid intravasation. Some authors prefer to avoid lacerations by inserting *Laminaria* the night before surgery.[79]

The cervix or uterus may be perforated during dilatation or insertion of the hysteroscope. This is a potential problem especially in an acutely anteflexed or retroflexed uterus and in women with a stenotic cervix. The incidence is not known but probably comparable to that associated with endometrial curettage, 4 to 13/1000 procedures.[80] This complication is prevented by maintaining appropriate techniques for dilatation. Another preventive measure is to insert the scope under direct vision. This may be difficult with the resectoscope, however, because of its width and sharp end. An obturator may be used, and the visual obturator is especially helpful (Circon ACMI, Stamford, CT).

Generally, perforations even with the large dilators require no treatment. Partial perforation requires no corrective measures. Most full-thickness perforations are in the fundal area, and although no treatment is necessary, adequate uterine distention is usually not possible and the procedure cannot be continued. Laparoscopy can be performed if there is any question as to the location of the perforation or the amount of intraabdominal bleeding.

Uterine Perforation

Uterine perforation rates and laparotomies to manage bowel or urinary tract injuries have risen slightly from 1991 to 1993, even though the complexity of the procedures has changed little.[64,65]

Poor visualization and lack of uterine distension increase the risks of uterine perforation during hysteroscopy. Although complications from perforation are more common with thermal energy sources, they may also occur when mechanical energy is used.[81] Scissors may be used to transect polyps, enucleate small fibroids, and transect uterine septa, and when a perforation occurs, the pelvic viscera usually is not damaged since the immediate outflow of distending medium alerts the surgeon to the event.

The surgical procedure that carries a high chance of uterine perforation even with concomitant laparoscopic control is transection of lateral and fundal uterine adhesions. The complication occurs with a frequency of 2 to 3/100.[82,83] The procedure generally must be discontinued since uterine distention cannot be maintained. Because it is so common, it is my belief that it is inappropriate to use thermal energy to lyse these adhesions. In addition to the risk of injury to pelvic viscera, thermal damage may occur to the surrounding endometrium, which is already compromised.

It is difficult to prevent perforation during lysis of adhesions. Concomitant laparoscopy offers some benefit and should be employed in most cases of lateral and fundal lesions. Ultrasound guidance is also helpful. Unless the perforation is lateral in the lower uterine segment where branches of the uterine artery might be transected, no correction is generally necessary. Unless the case is near completion, it has to be rescheduled in 2 to 3 months after the wound has healed.

The risk of a uterine perforation during septum transection would appear to be approximately 1/100 procedures.[84] It can arise despite concomitant laparoscopy. In addition, the uterus or fallopian tube may be perforated during tubal cannulation. The risk appears to be small, occurring 4 (11%) times during cannulation of 36 tubes, with no complications reported.[85]

Intraoperative and Postoperative Bleeding

Intraoperative bleeding is usually not a problem since the pressure of the distending medium overcomes the pressure of most open vessels. Occasionally a small artery may spurt blood during a fibroid resection, or if myometrium has been entered during a septum transection or lysis of intrauterine adhesions. These can be easily controlled by using 40 W of a modulated (coagulation) current delivered through the loop electrode.

According to surveys of gynecologists, intraoperative and postoperative bleeding and associated complications have increased progressively.[63–65] In a review of the literature of endometrial destruction by ablation or resection, the rates were between 0.2% and 1.0%.[86] The majority of these events occurred during endometrial resection with a resectoscope or when a dragging (touch) Nd:YAG laser technique was used.

Postoperative bleeding is most common during removal of myomas, and reportedly occurred in 2.2% of cases.[45] It should not be a difficulty with rollerball ablation unless there is a partial perforation of the uterus or a laceration has occurred in the lower uterine segment, since no vascular channels are otherwise open. It may occur after transection of the vascular uterine septum if the transection is too anterior, posterior, or high in the uterine fundus, thus opening myometrial vessels. Similarly, bleeding after transection of uterine synechiae indicates that myometrium rather than scar tissue has been cut.

It is difficult to prevent bleeding during removal of sessile fibroids since large vessels are exposed when the fibroid is enucleated from its base. This risk can be diminished if the myometrium is not damaged. This is accomplished by resecting only the portion of the fibroid that is protruding into the uterine cavity. Avoiding bleeding from transection of septum and lysis of adhesions is based on careful techniques in an effort to identify the visual difference between scarring septum and normal myometrium. Lower uterine segment laceration during dilatation can be prevented by inserting *Laminaria*.

At the termination of virtually every operative hysteroscopy there is the initial appearance of brisk bleeding. This generally is bloody distending medium draining from the uterine cavity. In bloodier cases such as fibroid resection, it may continue for a short time until the uterus contracts, very much like a postpartum uterus, and brings the bleeding to an acceptable level. Occasionally it is helpful to inject dilute vasopressin solution 3 υ in 10 ml saline into the cervix to hasten uterine contraction and stop the bleeding. Alternatives are inserting a pack containing vasopressin,[87] a Foley catheter,[88] or a balloon into the uterus.[89]

In over 200 fibroid resections, I have had to use an intrauterine tamponade in only 6. These were all in the first 47 cases and probably reflected anxiety and lack of appreciation that the uterus generally contracts to stop bleeding. None of these women required transfusion. Others reported a 3% transfusion rate and the

need for intrauterine balloons in one-half of all patients.[90] This is not the experience of most authors.

Thermal Injuries Secondary to Laser or Electrical Energy

Thermal injuries to surrounding organs can occur when either electrical or laser energy is used, and may result in significant morbidity and death. The uterus is a thick, muscular organ. It is relatively difficult to apply enough energy to its interior surface to cause full-thickness necrosis with possible damage to the outer surface, but such complications have been reported.[91] Most injuries are the result of physical perforation of the uterus while the energy source is activated.

In expert hands, these injuries are not common. No injuries to intraabdominal organs by thermal energy were reported in a series of 850 endometrial and myoma resections,[92] in a literature review of endometrial ablation,[86] or during the resection of intrauterine lesions even when a perforation had occurred.[45] However, a survey of physicians learning and performing endometrial resection reported that 52% of uterine perforations occurred during the first five cases and 33% occurred during the first one.[93] Injuries to other intraabdominal structures occurred only when the learning physician was not under direct supervision, and affected the bladder, ureters, and major blood vessels. Case reports describe injuries to the bowel.[94–96]

Although uncommon, two injuries to pelvic viscera without perforation were described with the use of the resectoscope[96] and the Nd:YAG laser.[94] In one case a high-power setting of the Nd:YAG laser with slow movement of the laser fiber was the apparent cause, since higher powers have been applied safely.[97] An abnormally thin uterine wall was judged to be the problem in the second case.

It is recognized by all hysteroscopists that the uterine cornual area and possibly the cesarean section scar are the thinnest areas and therefore the most vulnerable to perforation. The thinness of the uterine cornual area created a risk of bowel injury at the time of attempted electrical sterilization and was the main reason the procedure was abandoned.[98] Obturator nerve stimulation with subsequent forceful adduction of the patient's thighs may occur when operating in the cornual region.[99]

Damage to pelvic viscera is most frequent when the activated electrode or laser fiber is advanced away from the operator. Depth perception is difficult during hysteroscopy, and once the fiber or loop is out of sight, the depth to which it is penetrating cannot be determined easily. Perforation of the uterus and injury to pelvic organs are best prevented by not advancing the activated laser fiber or electrode away from the end of the telescope, and not resecting myomas below the level of the uterine cavity. Only a few exceptions to this rule exist. The special circumstances when the electrode or fiber may be advanced away from the end of the hysteroscope are during transection of a septum and when undercutting a piece of partially resected fibroid that is hanging loosely in the uterine cavity. This can be done safely since the electrode or laser fiber is advanced only for very short distances and is always in direct view as it enters the tissue.

It is not necessary to cut below the level of the uterine cavity to remove the intramural portion of the myoma,[100] but it is necessary when removing lateral and fundal intrauterine adhesions. Because of the increased risk of perforation during adhesion removal, thermal energy source presents an increased danger that is not found when mechanical methods are used.

If a perforation occurs and any question exists that thermal energy may have damaged the pelvic viscera, laparoscopy should be undertaken to determine if there is any blanching on the serosa of the uterus. If this is identified, abdominal exploration should usually be the next step, since it is difficult to inspect all of the bowel laparoscopically to look for sites of injury. If an injury is identified, it should be resected with adequate margins, rather than oversewn.

Instrument Breakage

Intrauterine instrument breakage can occur, with separation of scissor tips and resectoscope loops. An effort must be made to try to retrieve the broken piece, and this may be time consuming and difficult. There are probably no risks to leaving the missing piece in the uterine cavity; in fact, the piece may spontaneously exit the uterine cavity.[101] However, retrieval should be attempted to avoid the medicolegal risks of leaving a foreign body inside the patient.

Concomitant Laparoscopy or Ultrasonography and Hysteroscopic Surgery

Laparoscopy or ultrasonography has been recommended to help avoid complications and increase

the accuracy of the surgery. I do not believe that routine laparoscopy is either justified or necessary except when extensive lateral and fundal intrauterine adhesions exist, or when the external configuration of the uterus is not known in the case of septate uterus.

Use of the laparoscope during operative hysteroscopy gives a false sense of security since the uterus is generally not in the laparoscopist's view when the hysteroscopist is operating. This is because the uterus is tipped backward rather than elevated anteriorly out of the pelvis, and therefore is hidden behind bowel. For the laparoscopist to view the uterine surface, the hysteroscopist must anteflex the uterus, which generally makes additional intrauterine surgery at that time difficult or impossible. Laparoscopy does have a role in evaluating the external uterine surface if any questions arise as to whether a perforation with injury to pelvic viscera may have occurred.

Ultrasound is suggested as a better method than laparoscopy to assist the hysteroscopist.[102–105] It has value in determining the external configuration of the uterus in cases of septum, determining the extent of an intramural fibroid, identifying normal endometrium in patients with Asherman syndrome, and locating a hematometra after ablation.[106] In addition it can contribute to the safety of the procedure by demonstrating the closeness of the operating instruments to the uterine surface.

Infection

Infection is seldom a problem in diagnostic hysteroscopy and is not reported in many large series. In 4000 cases, most of which were diagnostic hysteroscopies, 8 (0.2%) infections were reported.[15] All occurred in the first 1000 cases and all were mild except one that occurred after laparotomy. The equipment was not sterilized between these cases, but only washed and rinsed with 70% alcohol.

Operative hysteroscopy increases the risk of infection not only by opening tissue planes, which allows the introduction of pathogens, but because the extended length of these procedures requires the hysteroscope to be removed and replaced into the uterine cavity through the vagina on numerous occasions. Infections may arise as endometritis, parametritis, or pyometra.[107] A review of endometrial ablation showed a risk of 0.3%,[86] and a general survey of operative hysteroscopy identified a rate of 1.6%.[93] The latter figure may have been elevated by the inexperience of many of the operators. A 2.0% infection rate was found in reviewing the published reports on fibroid resection.[45] Infections were found in 0.5% of 850 operative hysteroscopies even though the women received postoperative prophylactic antibiotics.[92] In performing over 274 endometrial ablation cases, 156 fibroid resections, and 44 myoma resections and ablations, I have not yet had a patient who developed infection.

The first step in preventing infections is proper cleaning, disinfecting, and sterilization of equipment. Routines similar to those used in laparoscopy are satisfactory.[108] Prophylactic administration of antibiotics, although practiced by some, is not universally employed by hysteroscopists and does not necessarily provide protection against infection.[92] It would appear, however, that patients with a history of pelvic inflammatory disease should receive prophylactic antibiotics.

This recommendation is based on a series of 700 patients, 200 of whom did not receive the agents.[109] Four (2%) of the 200 had a history of pelvic inflammatory disease, and three of them developed severe postoperative infections, including tubo-ovarian abscess. In the second group of 500 women, 10 (2%) had a history of pelvic inflammatory disease. All received doxycycline 200 mg twice/day starting at the time of *Laminaria* insertion. None developed a subsequent infection.

Although no infection or other problems have been demonstrated clinically, contamination with particulate matter in insufflated gas can occur, and the use of filters has been recommended.[110]

Infection after hysteroscopy should be managed in the standard fashion.

Nerve Injuries

Nerve injuries are a small but persistent problem in hysteroscopy, occurring 0.01% to 0.04% of patients.[63–65] They also occur with other gynecologic procedures, usually the result of stretching of the sciatic or peroneal nerve.[111] Recovery generally is complete although it may be prolonged and associated with pain.

Development of Hematometra

Hematometra after endometrial resection or ablation is not reported frequently. One report found that it occurred in 1.8% of patients.[92] It caused cyclic pain with or without uterine bleeding between 2 and 12 months after the procedure. Fourteen of the 16 women

were managed by cervical dilatation. Two required transection of uterine synechiae to approach the hematometra in the uterine fundus. Another report identified a hematometria from cervical stenosis in 3 of 61 patients.[112]

A variation of hematometra was described in women undergoing rollerball ablation who were or had been previously sterilized.[113] These patients experienced intermittent vaginal bleeding that was associated with severe cramping pain. All had marked endometrial scarring at hysteroscopy, and unilateral or bilateral swollen, blood-filled fallopian tubes that appeared similar to early ectopic pregnancy. Symptoms in most of these patients were alleviated by laparoscopic removal of the remaining proximal tube or tubes.

A review of my 247 cases of endometrial ablation demonstrated this postablation tubal sterilization syndrome in 4 women and a suspicion that it existed in 3 others. Of interest, only one of the seven women had Nd:YAG laser ablation and six had resectoscope rollerball ablation. In reviewing these findings it is my opinion that better cornual destruction was achieved with the Nd:YAG laser, since in-depth destruction occurred at approximately the same time surface changes were seen. Because of concern for the thinness of the cornual area, destruction apparently was inadequate most frequently in cases in which a rollerball was used. This would have been caused not only by inadequate application of the rollerball at the cornua, but also by the fact that the rollerball would have been touching tissue in its anterior, posterior, and forward positions, thus effectively diminishing current density by two-thirds. I am now more conscientious about thoroughly destroying the cornual areas with the rollerball.

Creation of Iatrogenic Adenomyosis

The risk of creating iatrogenic adenomyosis may be more common with the resection technique than with the rollerball or Nd:YAG laser.[114, 115] One reason may be that resection does not uniformly remove all of the deeper-lying normal endometrium that is usually destroyed by the Nd:YAG and rollerball. The orientation of the resected strips does not always allow the pathologist to determine if the full thickness of the endometrium has been resected. In such case, the remaining endometrium may be covered in scarring and appear as iatrogenic adenomyosis. Another theory for the adenomyosis is the transport of viable endometrial debris into the myometrium by the vessels opened at resection.[115]

Adenomyosis may precede the ablation, and this is a matter of controversy. Whereas some pathologists are comfortable making a diagnosis of adenomyosis from hysteroscopic resection chips, and a clinical correlation appears to exist,[116] others are unwilling to do so since a maximum depth of only 5 mm is obtained by resection.

Endometrial Cancer

Two concerns have been raised with regard to carcinoma of the endometrium and hysteroscopic procedures. The first is spreading of endometrial cancer cells during hysteroscopy. It is known that hysteroscopy even using CO_2 can spread endometrial cells into the peritoneal cavity.[117] It has been suggested this may increase the FIGO staging from a I to III.[118] The viability of these cells and whether or not their presence would have any long-term effect can be debated. It would, however, commit the patient to more aggressive therapy.

I reviewed 34 patients in whom endometrial carcinoma was identified at diagnostic hysteroscopy. Carbon dioxide only was used in 26 women. Four of them were switched from CO_2 to a fluid medium because of poor visualization, and four had only fluid medium used. Positive cytology was found in two women. One in whom glycine was used already had ovarian spread. The other had moderately differentiated tumor extending two-thirds of the way through the endometrial surface; CO_2 was the distending medium. Whether the positive cytology in these two patients related to the hysteroscopy cannot be determined, but it would appear that the chance of this is of low magnitude.

The other potential complication raise by detractors of endometrial ablation is the risk of obscuring the early symptoms of bleeding from an endometrial carcinoma. Five patients were reported in whom carcinoma of the endometrium developed after endometrial ablation.[119–123] An early diagnosis was made in all four. However, in my opinion, none of these women was a good candidate for endometrial ablation.

Four women were menopausal when the ablation was done. One had hyperplasia without atypia that had responded to progestin, but she was not given the agents after the ablation.[119] Endometrial cancer

developed 5 years postoperatively. The second woman had adenomatous hyperplasia with focal architectural atypia but no cytologic atypia.[120] She remained asymptomatic, and 1 year after ablation a predominantly grade I focal grade 2 carcinoma with greater than 50% myometrial invasion was found at a hysterectomy for urinary incontinence. The third patient was a morbidly obese diabetic who was 4 years postmenopausal at the time of ablation. Benign adenomatous hyperplasia had been found at dilatation and curettage (D&C) 2 years before, but a D&C 5 months before ablation revealed only an atrophic endometrium.[122] She, too, did not receive progestins after ablation. The fourth patient had focally atypical endometrium at the time of the ablation. Fourteen months later she was diagnosed with metastatic carcinoma of the endometrium.[123] The one woman who was not menopausal was a 39-year-old chronic anovulator who had an endometrial ablation rather than cyclic progestins for dysfunctional bleeding.[121] She did not receive postablation progestins.

Offering endometrial ablation to postmenopausal and anovulatory women should always be looked upon skeptically. The postmenopausal women described above manifested considerable endogenous estrogen effect, and two even suggested some endometrial atypia. The fifth patient probably could have been managed without surgery by cyclic progestins. All of these women were less than ideal candidates for the procedure, but once it was done, they should have received cyclic progestin therapy.

Endometrial cancer associated with endometrial hyperplasia[124] was found at the time of endometrial resection. No malignancy remained in the hysterectomy specimen. A complete preoperative work-up should be able to avoid this potential problem in all but the most unusual case.

Intrauterine Adhesion Formation after Correctly Done Hysteroscopy

Complications of hysteroscopic surgery when taken in its broadest sense can occur when the procedure has been performed correctly and safely. Postoperative intrauterine adhesions are undesirable in women who seek to maintain their fertility. This is not a major problem after fibroid resection, having been reported in 6% of patients undergoing follow-up hysteroscopy.[125] The removal of numerous fibroids or fibroids on opposing walls probably increases this risk. Although it would be theoretically possible to introduce a balloon or intrauterine device (IUD) into the uterine cavity after fibroid resection, the problem is infrequent enough that it seems more prudent to perform a repeat hysteroscopy in the office after the first spontaneous menses. If an adhesion is found, it can be easily lysed under direct vision. Consideration can also be given to administering oral estrogen to help reepithelize the raw surfaces. However, this was not found to be necessary after transection of uterine septum where normal endometrium will cover the surface in 2 months.[126]

Recurrence of intrauterine synechiae is considerably more difficult to manage. The extent of adhesion reformation depends on their location and the amount of uterine surface involved. When extensive adhesions are lysed, little normal endometrium remains to be reepithelized and the risk of recurrence is high.[127] It is difficult to keep separate the walls of the lateral and fundal adhesions to allow reepithelialization. An effort can be made to separate the walls by inserting a noncopper, nonprogressive IUD for the short term, or an intrauterine balloon or Foley catheter. Most authors believe estrogen should be administered to these patients to facilitate endometrial regrowth. When the adhesions are limited to the upper cervical canal and lower uterine segment, IUDs are not necessary, and office dilatation for several weeks will prevent reformation.

The formation of scarring after a septum transection is seldom a problem, and an intrauterine stent is not necessary. These cut surface are avascular and do not heal together. Reepithelization is rapid.[126]

Difficulties With Pregnancies

No problems are expected after the uncomplicated removal of a fibroid or transecton of a septum and fertility should be enhanced.[45, 128] However, pregnancy after intrauterine surgery may be unexpected or be associated with difficulties.

Abnormal implantation resulting in a placenta accreta or a poor pregnancy outcome may occur in women who have had lysis of uterine synechiae.[129, 130] Abnormal implantation is caused by lack of normal endometrium on which the placenta can form a normal attachment. This is almost impossible to prevent. Correction consists of obstetric management similar to that when the disorder occurs spontaneously.

Unplanned pregnancies may occur after endometrial ablation in patients who were not sterilized previously. Their frequency is reported to be 0.2%

1.6%,[92,131] but the true figure is probably much higher, since many women undergo hysteroscopic sterilization or use other forms of contraception. If a pregnancy occurs after ablation, an ectopic gestation should be ruled out. The woman who chooses to continue the pregnancy should be made aware of the fact that since the vascular supply to the uterine surface has been damaged by the endometrial surgery, a placental insufficiency-like syndrome could develop,[131] as well as other serious placental problems.[129,132]

Uterine rupture has been reported during pregnancy after a uterine perforation at hysteroscopy for septum transection for submucous myoma resection and during lysis of adhesions.[129,133-137] One of my patients experienced such an event at 28 weeks' gestation. It was in the left fundal area, at the site of a perforation that occurred during lysis of severe intrauterine adhesions.

Even though the tubal ostia are easily approached hysteroscopically, pregnancies commonly occur after most hysteroscopic sterilizations.[138] Only formed-in-place silicone plugs (Ovabloc System; Aphation, Rotterdam, The Netherlands) have been effective in preventing pregnancies.[139] Other ostial tubal plugs have not justified clinical trials. The primary reason that an electrical hysteroscopic sterilization techniques were abandoned was the high frequency of perforation and bowel injuries. However, a second reason makes any type of tubal sterilization that attempts to close the tube at the ostium unreliable, even if the depth of the destruction could be controlled; that is, the 1% ectopic pregnancy rate, with three-fourths of them being interstitial ectopic pregnancies.[138]

Death

Fortunately, deaths related to hysteroscopy are infrequent, and many large series do not report any. The true rate is not known, since deaths may go unreported and the denominators are not generally given in published case reports. A rate of 1.7/10,000 cases has been reported.[64]

Summary

If the contraindications to hysteroscopy are observed, complications should be kept at a minimum. The risk of fluid overload, especially in operative cases, is the single most common and critical complication. Accurate recording of fluid intake and outflow are absolutely essential. This and all other complications are most likely to arise in the hands of inexperienced and unsupervised surgeon.[2,93]

References

1. Loffer FD: Contraindications and complications. In Obstetrics and Gynecology Clinics of North America: Hysteroscopy. Edited by AM Siegler. Philadelphia, WB Saunders, 1995, pp 445–472
2. Smith DC, Donohue LR, Waszak SJ: A hospital review of advanced gynecologic endoscopic procedures. Am J Obstet Gynecol 170:1635–1642, 1994
3. Loffer FD: Complications from uterine distention. In Complications of Laparoscopy and Hysteroscopy. Edited by RS Corfman, MP Diamond, AH DeCherney. Boston, Blackwell, 1993, pp 177–186
4. Taylor PJ, Hamou JE: Hysteroscopy. J Reprod Med 28:359–389, 1983
5. Shirk GJ, Kaigh J: The use of low-viscosity fluids for hysteroscopy. J Am Assoc Gynecol Laparosc 2:11–21, 1994
6. Vulgaropoulos SP, Haley LC, Hulka JF: Intrauterine pressure and fluid absorption during continuous flow hysteroscopy. Am J Obstet Gynecol 167:386–391, 1992
7. Shirk GJ, Gimpelson RJ: Control of intrauterine fluid pressure during operative hysteroscopy. J Am Assoc Gynecol Laparosc 1:229–233, 1994
8. Loffer FD: The need to monitor intrauterine pressure—Myth or necessity? J Am Assoc Gynecol Laparosc 2:1–2, 1994
9. Garry R, Hasham F, Kokri M, et al: The effects of pressure on fluid absorption during endometrial ablation. J Gynecol Surg 8:10, 1992
10. Garry R, Mooney P, Hasham F, et al: A uterine distention system to prevent fluid absorption during Nd:YAG laser endometrial ablation. Gynaecol Endosc 1:23–27, 1992
11. Corson SL, Brooks PF, Serden SP, et al: Effects of vasopressin administration during hysteroscopic surgery. J Reprod Med 39:419–423, 1994
12. Vercellini P, Ragni G, Colombo A, et al: Gonadotropin releasing hormone agonist treatment before hysteroscopic metroplasty. Gynaecol Endosc 2:153–157, 1993
13. Nezhat F, Admon D, Nezhat CH, et al: Life-threatening hypotension after vasopressin injection during operative laparoscopy, followed by uneventful repeat laparoscopy. J Am Assoc Gynecol Laparosc 2:83–86, 1994

14. Arieff AI, Ayus JC: Hyponatremic encephalopathy after endometrial ablation. JAMA 271:345, 1994

15. Salat-Baroux J, Hamou JE, Maillard G, et al: Complications from microhysteroscopy. In Hysteroscopy: Principles in Practice. Edited by AM Siegler, HJ Lindemann. Philadelphia, JB Lippincott, 1984, pp 112–117

16. Van der Pas H, Vancaille T: Manual of Hysteroscopy. Gynecologic Endoscopy, vol. 2. Amsterdam, Elsevier, 1990, pp 177–187

17. Loffer FD: Hysteroscopy in differentiating ectopic pregnancy from other non-viable pregnancies when serum hCG titers remain below the threshold of ultrasound diagnosis. J Gynecol Surg 8:165–166, 1992

18. Lindemann HJ, Mohr J: CO_2 hysteroscopy: Diagnosis and treatment. Am J Obstet Gynecol 124:129–133, 1976

19. Gallinat A: Carbon dioxide hysteroscopy: Principles and physiology. In Hysteroscopy: Principles and Practice. Edited by AM Siegler, HJ Lindemann. Philadelphia, JB Lippincott, 1984, pp 45–48

20. Corson SL, Hoffman JJ, Jackowski J, et al: Cardiopulmonary effects of direct venous CO_2 insufflation in ewes. A model for CO_2 hysteroscopy. J Reprod Med 33:440–444, 1988

21. Rythen-Alder E, Brundin J, Notini-Gudmarsson A, et al: Detection of carbon dioxide embolism during hysteroscopy. Gynaecol Endosc 1:207–210, 1992

22. Mahmoud F, Fraser IS: CO_2 hysteroscopy and embolism. Gynaecol Endosc 3:91–95, 1994

23. Brink DM, DeJong P, Fawcus S, et al: Carbon dioxide embolism following diagnostic hysteroscopy. Br J Obstet Gynaecol 101:717–718, 1994

24. Siegler AM, Valle RF, Lindemann HJ, et al: Therapeutic Hysteroscopy: Indications and Techniques. St. Louis, CV Mosby, 1990, pp 53–61

25. Ott DE: Laparoscopic hypothermia. J Laparoendosc Surg 1:127–131, 1991

26. Leake JF, Murphy AA, Zacur HA: Noncardiogenic pulmonary edema: A complication of operative hysteroscopy. Fertil Steril 48:497–499, 1987

27. Zbella EA, Moise J, Carson SA: Noncardiogenic pulmonary edema secondary to intrauterine instillation of 32% dextran 70. Fertil Steril 43:479–480, 1985

28. Mangar D, Gerson JI, Constantine RM, et al: Pulmonary edema and coagulopathy due to Hyskon (32% dextran 70) administration. Anesth Analg 68:686–687, 1989

29. Jedeikin R, Olsfanger D, Kessler I: Disseminate intravascular coagulopathy and adult respiratory dis tress syndrome: Life threatening complications of hys teroscopy. Am J Obstet Gynecol 162:44–45, 1990

30. Schinagl EF: Hyskon (32% dextran 70). hysteroscop surgery and pulmonary edema. Anesth Ana 70:223–224, 1990

31. Loffer FD: Explanation of mechanism of Hyskon sol tion reaction needed. Am J Obstet Gynecol 163:20 1990

32. Ljungstrom K: Safety of 32% dextran 70 for hyst oscopy. Am J Obstet Gynecol 163:2029, 1990

33. Lukacsko P: Noncardogenic pulmonary edema s ondary to intrauterine instillation of 32% dextran Fertil Steril 54:560–561, 1985

34. Witz CA, Schenken RS, Silverberg KM, et Complications associated with the absorption of h teroscopic fluid media. Fertil Steril 60:745–756, 1

35. Golan A, Siedner M, Bahar M, et al: High-output ventricular failure after dextran use in an operative teroscopy. Fertil Steril 54:939–941, 1990

36. Ruiz JM, Neuwirth RS: The incidence of complica associated with the use of Hyskon during hysterosc Experience in 1783 consecutive patients. J Gyn Surg 8:219, 1992

37. Anonymous. Users reminded about adverse reac to Dextran. FDA Drug Bull 13:23–24, 1983

38. Taylor PJ, Cumming DC: Hysteroscopy in 100 pa Fertil Steril 31:301–308, 1979

39. Trimbos-Kemper TCM, Veering BT: Anaphy shock from intracavitary 32% dextran 70 durin teroscopy. Fertil Steril 51:1053–1054, 1989

40. Ahmed N, Falcone T, Tulandi T, et al: Anaph reaction because of intrauterine 32% dextran 70 lation. Fertil Steril 55:1014–1016, 1991

41. Loffer FD: Hysteroscopic sterilization with the formed-in-place silicone plugs. Am J Obstet G 149:261–270, 1984

42. Baggish MS, Davauluri C, Rodriguez F, et al: V uptake of Hyskon (dextran 70) during operative a nostic hysteroscopy. J Gynecol Surg 8:211–21

43. Data JL, Nies AS: Dextran 40. Ann Inte 81:500–504, 1974

44. Heinonen PK: For heavy users of dextran wi read Acta Chiurgica Scandinavica. Fertil Steril 1990

45. Loffer FD: Removing intrauterine lesions: Myomectomy and polypectomy. In The Gynecologic Resectoscope. Edited by EJ Bieber, FD Loffer. Cambridge, Blackwell, 1994, pp 168–194

46. Garry R, Erian J, Grochmal S: A multi-centre collaborative study into the treatment of menorrhagia by Nd-YAG laser ablation of the endometrium. Br J Obstet Gynaecol 98:357–362, 1991

47. Goldrath MH, Fuller TA, Segal S: Laser photovaporization of endometrium for the treatment of menorrhagia. Am J Obstet Gynecol 140:14–19, 1981

48. Goldrath MH: Intrauterine laser surgery. In Laser Surgery in Gynecology and Obstetrics. Edited by WR Keye. Boston, GK Hall, 1984, pp 93–110

49. Lomano JM: Photocoagulation of the endometrium with the Nd:YAG laser for the treatment of menorrhagia. J Reprod Med 31:148–150, 1986

50. Lomano JM, Feste JR, Loffer FD, et al: Ablation of the endometrium with the neodymium:YAG laser: A multi-center study. Colposc Gynecol Laser Surg 2:203–207, 1986

51. Goldfarb HA: A review of 35 endometrial ablations using the Nd:YAG laser for recurrent menometrorrhagia. Obstet Gynecol 76:833–835, 1990

52. Feinberg BI, Gimpelson RJ, Godier DE: Pulmonary edema after photocoagulation of the endometrium with the Nd:YAG laser—A case report. J Reprod Med 34:431–434, 1989

53. Morrison LMM, Davis J, Sumner D: Absorption of irrigating fluid during laser photocoagulation of the endometrium in the treatment of menorrhagia. Br J Obstet Gynaecol 96:346–352, 1989

54. Davis JA: Hysteroscopic endometrial ablation with the neodymium-YAG laser. Br J Obstet Gynaecol 96:928–932, 1989

55. Carson SA, Hubert GD, Schriock ED, et al: Hyperglycemia and hyponatremia during operative hysteroscopy with 5% dextrose in water distention. Fertil Steril 51:341–343, 1989

56. Van Boven MJ, Singelyn F, Donnez J, et al: Dilutional hyponatremia associated with intrauterine endoscopic laser surgery. Anesthesiology 71:449–450, 1989

57. Van Boven M, Singelyn F, Gribmont B: Complications and precautions in operative hysteroscopic surgery. In Laser Operative Laparoscopy and Hysteroscopy. Edited by J Donnez. Leuven, The Netherlands, Nauwelaerts Printing, 1989, pp 209–306

58. Magos AL, Baumann R, Turnbull AC: Safety of transcervical endometrial resection. Lancet 336:44, 1990

59. Baumann R, Magos AL, Kay JDS, et al: Absorption of glycine irrigating solution during transcervical resection of the endometrium. BMJ 304–305, 1990

60. Boto TCA, Fowler CG, Cockroft S, et al: Absorption of irrigating fluid during transcervical resection of the endometrium. BMJ 300:748, 1990

61. Kim AH, Keltz MD, Arici A, et al: Dilutional hyponatremia during hysteroscopic myomectomy with sorbitol-manitol distention medium. J Am Assoc Gynecol Laparosc 2:237–243, 1995

62. Allgen LG, Norlen H, Kolmert, et al: Absorption and elimination of mannitol solution when used as an isotonic irrigating agent in connection with transurethral resection of the prostate. Scand J Urol Nephrol 21:177–184, 1987

63. Peterson HB, Hulka JF, Phillips JM: American Association of Gynecologic Laparoscopists' 1988 membership survey of operative hysteroscopy. J Reprod Med 35:590–591, 1990

64. Hulka JA, Peterson HB, Phillips JM, et al: Operative hysteroscopy: American Association of Gynecologic Laparoscopists' 1991 membership survey. J Reprod Med 38:572–573, 1993

65. Hulka JA, Peterson HA, Phillips JM, et al: Operative hysteroscopy: American Association of Gynecologic Laparoscopists' 1993 membership survey. J Am Assoc Gynecol Laparosc 2:131–132, 1995

66. Istai O, Skajaa J, Schjoensby AP, et al: Changes in serum electrolytes of the transcervical resection of endometrium and submucous fibroids with the use of glycine 1.5% for uterine irrigation. Obstet Gynecol 80:218–222, 1992

67. Baggish MS, Brill AI, Rosenswieg B, et al: Fatal acute glycine and sorbitol toxicity during operative hysteroscopy. J Gynecol Surg 9:137–143, 1993

68. Arieff AI, Ayus JC: Endometrial ablation complicated by fatal hyponatremic encephalopathy. JAMA 270:1230–1232, 1993

69. Ayus JC, Wheeler JM, Arieff AI: Postoperative hyponatremic encephalopathy in menstruant women. Ann Intern Med 117:891–897, 1992

70. Arieff AI: Hyponatremia, convulsions, respiratory arrest and permanent brain damage after elective surgery in healthy women. N Engl J Med 314:1529–1535, 1986

71. Berl T: Treating hyponatremia: What is all the controversy about? Ann Intern Med 113:417–419, 1990

72. Byers GF, Pinion S, Parkin DE, et al: Fluid absorption during transcervical resection of the endometrium. Gynaecol Endosc 2:21–23, 1993

73. Loffer FD: Hysteroscopy. In Infertility Surgery. Edited by JJ Stangel. East Norwalk, CT, Appleton & Lange, 1990, pp 72–85

74. Wood SM, Roberts FL: Air embolism during transcervical resection of endometrium. BMJ 300:945, 1990

75. Perry PM, Baughman VL: A complication of hysteroscopy: Air embolism. Anesthesiology 73:546–547, 1990

76. Tur-Kaspa I: Hyperbaric oxygen therapy for air embolism complicating operative hysteroscopy. Am J Obstet Gynecol 163:680–681, 1990

77. Baggish MS, Daniell JF: Catastrophic injury secondary to the use of coaxial gas-cooled fibers and artificial sapphire tips for intrauterine surgery: A report of five cases. Laser Surg Med 9:581–584, 1989

78. Challener RC, Kaufman B: Fatal venous air embolism following sequential unsheathed (bare) and sheathed quartz fiber Nd:YAG laser endometrial ablation. Anesthesiology 73:548–551, 1990

79. Ostrzenski A: Resectoscopic cervical trauma minimized by inserting Laminaria digitata preoperatively. Int J Fertil 39:111–113, 1994

80. Grimes DA: Diagnostic dilation and curettage. A reappraisal. Am J Obstet Gynecol 142:1–6, 1982

81. Gentile GP, Siegler AM: Inadvertent intestinal biopsy during laparoscopy and hysteroscopy: A report of two cases. Fertil Steril 36:402–404, 1981

82. Valle RF, Sciarra JJ: Intrauterine adhesions: Hysteroscopic diagnosis, classification, treatment and reproductive outcome. Am J Obstet Gynecol 158:1459–1470, 1988

83. March CM, Israel R, March AD: Hysteroscopic management of uterine adhesions. Am J Obstet Gynecol 130:653–657, 1978

84. Hassiakos DK, Zourlas PA: Transcervical division of uterine septa. Obstet Gynecol Surv 45:165–173, 1990

85. Flood JT, Grow DR: Transcervical tubal cannulation: A review. Obstet Gynecol Surv 48:768–776, 1993

86. Loffer FD: Endometrial ablation—Where do we stand? Gynaecol Endosc 1:175–179, 1992

87. Townsend DE: Vasopressin pack for treatment of bleeding after myoma resection. Am J Obstet Gynecol 165:1405–1407, 1991

88. Goldrath MH: Uterine tamponade for the control of acute uterine bleeding. Am J Obstet Gynecol 147:869–872, 1983

89. Neuwirth RS: A new technique for and additonal experience with hysteroscopic resection of submucous fibroids. Am J Obstet Gynecol 131:91–94, 1978

90. Molsberry RL, Neuwirth RS: Complications of hysteroscopic myomectomy. In Complications of Laparoscopy and Hysteroscopy. Edited by RS Corfman, MP Diamond, AH DeCherney. Boston, Blackwell, 1993, pp 197–202

91. Voss E, Tiernan C: Full-thickness thermal necrosis invisible at endometrial ablation—A common occurrence? Gynaecol Endosc 2:239–240, 1993

92. Hill D, Maher P, Wood C, et al: Complications of operative hysteroscopy. Gynaecol Endosc 1:185–189, 1992

93. Macdonald R, Phipps J, Singer A: Endometrial ablation: A safe procedure. Gynaecol Endosc 1:7–9, 1992

94. Perry CP, Daniell JF, Gimpelson RJ: Bowel injury from Nd:YAG laser endometrial ablation. J Gynecol Surg 6:199–203, 1990

95. Sullivan B, Kenney P, Seibel M: Hysteroscopic resection of fibroids with thermal injury to the sigmoid. Obstet Gynecol 80:546–547, 1992

96. Kivnick S, Kanter MH: Bowel injury from rollerball ablation of the endometrium. Obstet Gynecol 79:833–834, 1992

97. Indman PD: High power Nd:YAG laser ablation of the endometrium. J Reprod Med 36:501–504, 1991

98. Darabi K, Roy K, Richart RM: Collaborative studies on hysteroscopic sterilization procedures: Final report. In Risks, Benefits and Controversies in Fertility Control. Edited by JJ Sciarra, GI Zatauchni, JJ Speidel. Hagerstown, MD, Harper & Row, 1978, pp 81–101

99. Pittroff R, Darwish DH, Shabib G: Near-fatal uterine perforation during transcervical endometrial resection. Lancet 338:197–198, 1991

100. Loffer FD: Removal of large symptomatic intrauterine growths by the hysteroscopic resectoscope. Obstet Gynecol 76:836–840, 1990

101. Letteric GS: Loop fracture during intrauterine surgery with a gynecologic resectoscope. Am J Obstet Gynecol 170:1839–1840, 1994

102. Shalev E, Zuckerman H: Operative hysteroscopy under real-time ultrasonography. Am J Obstet Gynecol 155:1360–1361, 1986

103. Lin BL, Iwata Y, Miyamoto N, et al: Three-contrasts method: An ultrasound technique for monitoring transcervical operations. Am J Obstet Gynecol 156:469–472, 1987

104. Dabirashrafi H, Mohamad K, Maghadomi-Tabrizi N: Three-contrasts method hysteroscopy: The use of real-time ultra-sonography for monitoring intrauterine operations. Fertil Steril 57:450–452, 1992

105. Letterie GS, Kramer DJ: Intraoperative ultrasound guidance for intrauterine endoscopic surgery. Fertil Steril 62:654–656, 1994

106. Fraser IS, Song JY, Jansen RPS, et al: Hysteroscopic lysis of intra-uterine adhesions under ultrasound guidance. Gynaecol Endosc 4:35–40, 1995

107. Amin-Hanjani S, Good JM: Pyometra after endometrial resection and ablation. Obstet Gynecol 85:893–894, 1995

108. Loffer FD: Disinfection vs. sterilization of gynecologic laparoscopy equipment—The experience of the Phoenix Surgicenter. J Reprod Med 25:263–266, 1980

109. McCausland VM, Fields GA, McCausland AM, et al: Tubal ovarian abscesses after operative hysteroscopy. J Reprod Med 38:198–200, 1993

110. Ott DE: Contamination via gynecologic endoscopy insulation. J Gynecol Surg 5:205–208, 1989

111. Loffer FD, Pent D, Goodkin R: Sciatic nerve injury in a patient undergoing laparoscopy. J Reprod Med 21:371–372, 1978

112. Jacobs SA, Blumenthani NJ: Endometrial resection follow up: Late onset of pain and the effect of depot medroxyprogesterone acetate. Br J Obstet Gynaecol 101:605–609, 1994

113. Townsend DE, McCausland A, Fields G, et al: Postablation tubal sterilization syndrome. Obstet Gynecol 82:422–424, 1993

114. Coltart TM, Smith RNJ: Letter. Lancet 338:312, 1991

115. McLucas B, Perrella R: Does endometrial resection cause adenomyosis? Gynaecol Endosc 4:123–127, 1995

116. McCausland AM: Hysteroscopic myometrial biopsy: Its use in diagnosis, adenomyosis and its clinical application. Am J Obstet Gynecol 166:1619–1628, 1992

117. Ranta H, Aine R, Oskanen H, et al: Dissemination of endometrial cells during carbon dioxide hysteroscopy and chromotubation among infertile patients. Fertil Steril 53:751–753, 1990

118. Romano S, Shimoni Y, Muralee D, et al: Retrograde seeding of endometrial carcinoma during hysteroscopy. Gynecol Oncol 44:116–118, 1992

119. Cooperman AB, DeCherney AH, Olive DL: A case of endometrial cancer following endometrial ablation for dysfunctional uterine bleeding. Obstet Gynecol 82:640–642, 1993

120. Margolis MT, Thoen LD, Boike GM, et al: Asymptomatic endometrial carcinoma after endometrial ablation. Int J Gynaecol Obstet. in press.

121. Ramey JW, Koonings PP, Given FT, et al: The process of carcinogenesis for endometrial adenocarcinoma could be short: Development of a malignancy after endometrial ablation. Am J Obstet Gynecol 170:1370–1371, 1994

122. Baggish MS, Ringgenberg E, Sze EHM: Adenocarcinoma of the corpus uterine following endometrial ablation. J Gynecol Surg 11:91–94, 1995

123. Horowitz IR, Copas PR, Aaronoff M, et al: Endometrial andenocarcinoma following endometrial ablation for postmenopausal bleeding. Gynecol Oncol 56, 460–463, 1995

124. Dwyer NA, Stirrat GM: Early endometrial carcinoma: An incidental finding after endometrial resection. Case report. Br J Obstet Gynaecol 98:733–734, 1991

125. Wamsteker K, Emanuel MH, deKruif JH: Transcervical hysteroscopic resection of submucous fibroids for abnormal uterine bleeding: Results regarding the degree of intramural extension. Obstet Gynecol 82:736–740, 1993

126. Candiani GB, Carinelli SG, Vercellini P, et al: Repair of the uterine cavity after hysteroscopic septal incision. Fertil Steril 54:991–994, 1990

127. Schlaff WD, Hurst BS: Preoperative sonographic measurement of endometrial pattern predicts outcome of surgical repair in patients with severe Asherman's syndrome. Fertil Steril 63:410–413, 1995

128. March CM, Israel R: Gestational outcome following hysteroscopic lysis of adhesions. Fertil Steril 36:455–459, 1981

129. Friedman A, DeFazio J, DeCherney A: Severe obstetric complications after aggressive treatment of Asherman syndrome. Obstet Gynecol 67:864–867, 1986

130. Valle RF, Sciarra JJ: Intrauterine adhesions: Hysteroscopic diagnosis, classification, treatment and reproductive outcome. Am J Obstet Gynecol 158:1459–1470, 1988

131. Whitlaw NL, Garry R, Sutton CJG: Pregnancy following endometrial ablation: 2 case reports. Gynaecol Endosc 1:129–132, 1992

132. Hill DJ, Maher PJ: Pregnancy following endometrial ablation. Gynaecol Endosc 1:47–49, 1992

133. Deaton JL, Maier D, Andreoli J: Spontaneous uterine rupture during pregnancy after treatment of Asherman's syndrome. Am J Obstet Gynecol 160:1053–1054, 1989

134. Creinin M, Chen M: Uterine defect in a twin pregnancy with a history of hysteroscopic fundal perforation. Obstet Gynecol 79:879–880, 1992

135. Howe RS: Third-trimester uterine rupture following hysteroscopic uterine perforation. Obstet Gynecol 81:827–829, 1993

136. Lobaugh ML, Bammel BM, Duke D, et al: Uterine rupture during pregnancy in a patient with a history of hysteroscopic uterine perforation. Am J Obstet Gynecol 83:838–840, 1994

137. Yaron Y, Shenhav M, Jaffa AJ, et al: Uterine rupture at 33 weeks gestation subsequent to hysteroscopic uterine metroplasty. Am J Obstet Gynecol 170:786–787, 1994

138. Loffer FD: Hysteroscopic tubal occlusion. In Endoscopic Surgery for Gynaecologists. Edited by C Sutton, M Diamond. London, WB Saunders, 1993, pp 345–354

139. Loffer FD, Loffer PS: Hysteroscopic tubal occlusion with the use of formed-in-place silicone devices: A long-term follow-up. Gynaecol Endosc 1:203–205, 1992

given four hours later. Thirteen hours after the ingestion the theophylline level was 31 μg/ml (170.5 μmol/l) and had fallen further to 23.2 μg/ml (128.8 μmol/l) at 14.5 hours. No further dose of antiemetic was required and she was discharged from hospital after psychiatric review at 36 hours.

Although this girl may have reached her peak plasma theophylline level at the time of administration of ondansetron, the serotonin antagonist promptly "switched off" her vomiting, allowing successful gut decontamination with activated charcoal. Despite the expense of this drug, our Pharmaceutical Advisory Committee has, on the basis of this case, approved the use of ondansetron for refractory vomiting following theophylline overdose. We believe that use of this drug warrants consideration under such circumstances.

DAVID DALY
JAMES N. TAYLOR
Emergency Department,
Royal Melbourne Hospital, Melbourne, Vic.

References
1. Tobias JD. Ondansetron: Indications and applications in the paediatric intensive care unit. Anaesth Intens Care 1992; 20:504-528.
2. Brown SGA, Prentice DA. Ondansetron in the treatment of theophylline overdoses. Med J Aust 1992; 156, April 6:512.

Endometrial ablation and air embolism

Dear Sir,

A 40-year-old woman weighing 80 kg presented for endometrial ablation. She was generally in good health and had had previous uncomplicated anaesthetics. Preoperative haemoglobin and electrolytes were normal.

She received temazepam 20 mg as premedication and was induced with fentanyl 100 μg, followed by a sleep dose of thiopentone 350 mg. Intubation was facilitated with vecuronium 7 mg. After intubation, anaesthesia was maintained with nitrous oxide, oxygen (FiO_2 0.3) and isoflurane 0.8%. Monitoring consisted of ECG, noninvasive blood pressure, pulse oximetry and capnography.

During the course of the anaesthetic, her blood pressure was 120-130 mmHg systolic, pulse oximetry 98%, and endtidal CO_2 32 mmHg.

Endometrial ablation was performed with the use of "roller ball" electrosurgery and 1.5% glycine under pressure of 90-100 mmHg. She was placed in the lithotomy position with slight head-down tilt. Approximately 40 minutes after commencement of the operation, the capnograph showed an endtidal carbon dioxide level of 20 mmHg and pulse oximetry reading of 90%. However, a radial pulse was easily felt, and blood pressure was 120 mmHg. There was no change in ventilation parameters. Auscultation of the chest showed equal air entry, normal heart sounds, with no added sounds. Surgery was immediately ceased and a presumptive diagnosis of air embolus was made. Within approximately two minutes pulse oximetry and endtidal carbon dioxide readings returned to normal. It was calculated that she had absorbed 600 ml of glycine solution. The acute changes in oximetry and $ETCO_2$ were verified on the trend mode of the Datex monitor. Inspection of the glycine bags and tubing showed no evidence of air. As the patient's clinical condition was stable and surgery was nearly finished, surgery was allowed to be completed. This took about five minutes.

Muscle relaxation was reversed with neostigmine and atropine. She developed laryngeal spasm on extubation, requiring re-intubation, but recovery was uneventful and she was discharged from hospital the following day. Postoperative electrolytes and haemoglobin were normal.

Endometrial ablation with the use of "roller ball" electrosurgery is a new surgical procedure in which air embolus is a rare but possible complication. It has been reported with the use of "loop diathermy" endometrial ablation. Anaesthetists should be alert to the possibility of air embolus in this procedure. Air could come from the glycine bag or tubing, but also from the "bubbles" produced by the diathermy in the glycine. The surgeon's comment during this case was that the bubbles generated by the diathermy were "very small". However, they are under pressure of 90-100 mmHg and would expand when in the venous system with a much lower pressure. It may also be argued that an acute absorption of glycine may have caused the changes in pulse oximetry and $EtCO_2$.

ANDREW MICHAEL
Lyell McEwin Health Service,
Elizabeth Vale, S.A.

References
1. Gillespie A. Endometrial ablation, a conservative alternative to hysterectomy for menorrhagia? Med J Aust 1991, 154, June 17: 791-792.
2. Wood SM, Roberts FL. Air embolism during transcervical resection of endometrium (letter), Br Med J 1990; 300:945.

Use of a size 2 LMA to relieve life-threatening hypoxia in an adult with quinsy

Dear Sir,

One of the major advantages of the LMA is the rapid relief of hypoxia following failure to intubate where alternative airway techniques prove inadequate. We would like to report the unusual use of a size 2

4/01

Review Articles

Primary Care

GAS EMBOLISM

CLAUS M. MUTH, M.D., AND ERIK S. SHANK, M.D.

GAS embolism, the entry of gas into vascular structures, is a largely iatrogenic clinical problem that can result in serious morbidity and even death.[1] Since gas embolism can result from procedures performed in almost all clinical specialties (Table 1), it is important for all clinicians to be aware of this problem. In most cases, gas embolism is air embolism, although the medical use of other gases, such as carbon dioxide, nitrous oxide, and nitrogen, can also result in the condition. There are two broad categories of gas embolism, venous and arterial, which are distinguished by the mechanism of gas entry and the site where the emboli ultimately lodge.

VENOUS GAS EMBOLISM

Venous gas embolism occurs when gas enters the systemic venous system.[2] The gas is transported to the lungs through the pulmonary arteries, causing interference with gas exchange, cardiac arrhythmias, pulmonary hypertension, right ventricular strain, and eventually cardiac failure. Physical preconditions for the entry of gas into the venous system are the incising of noncollapsed veins and the presence of subatmospheric pressure in these vessels. Noncollapsing veins include the epiploic veins, the emissary veins, and the dural venous sinuses. Air may enter these veins during neurosurgical operations, especially those performed with the patient in the sitting position.[3] The veins of the throat, and in some cases the veins in the coagulated operative field,[4] may also be entryways for air. Air may also enter veins through central venous and hemodialysis catheters[5,6] and may enter the veins of the myometrium during pregnancy and after delivery.[7,8]

From the Druckkammerzentrum Homburg, University Hospital Homburg, University of the Saarland, Homburg/Saar, Germany (C.M.M.); and the Department of Anesthesiology and Critical Care, Massachusetts General Hospital and the Department of Anesthesiology, Harvard Medical School — both in Boston (E.S.S.). Address reprint requests to Dr. Muth at the Druckkammerzentrum Homburg, Universitätskliniker des Saarlandes, 66424 Homburg/Saar, Germany, or at cmmuth@aol.com.

Pathophysiology

The most frequent form of venous gas embolism is the insidious venous aeroembolism, in which a series of gas bubbles resembling a string of pearls enters the venous system. Rapid entry or large volumes of gas put a strain on the right ventricle because of the migration of the emboli to the pulmonary circulation. The pulmonary arterial pressure increases, and the increased resistance to right ventricular outflow causes diminished pulmonary venous return. Because of the diminished pulmonary venous return, there is decreased left ventricular preload, resulting in diminished cardiac output and, ultimately, systemic cardiovascular collapse.[9] Tachyarrhythmias often develop, but bradycardias are possible as well. When large quantities of gas (over 50 ml) are injected abruptly, acute cor pulmonale, asystole, or both are likely to occur.[2] The alteration in the resistance of the lung vessels and the mismatch between ventilation and perfusion cause intrapulmonary right-to-left shunting and increased alveolar dead space, leading to arterial hypoxia and hypercapnia.

Diagnosis

To diagnose venous gas embolism, the physician should assess the clinical findings. The so-called millwheel murmur, a splashing auscultatory sound due to the presence of gas in the cardiac chambers and great vessels, is often present and can be auscultated by a precordial or esophageal stethoscope. A decrease in the end-tidal carbon dioxide levels, as determined by capnometry, suggests a change in the relation between ventilation and perfusion due to the obstruction of the pulmonary arteries.[10] Doppler ultrasonography is a sensitive and practical means of detecting intracardiac air, and it is often used during neurosurgical procedures,[3,11] procedures with the patient in the sitting position, and other procedures that entail a high risk of gas embolism. An even more sensitive and definitive method for detecting intracardiac gas is transesophageal echocardiography, although it requires training in performance and interpretation (Fig. 1).[2,3,12]

Treatment

When venous gas embolism is suspected, further entry of gas must be prevented. In certain cases, therapy with catecholamines is required, and, if necessary, aggressive cardiopulmonary resuscitation is performed. Adequate oxygenation is often possible only with an increase in the oxygen concentration of the inspired gas (up to 100 percent oxygen). Supplemental oxygen also reduces the size of the gas embolus

TABLE 1. MEDICAL SPECIALTIES WITH DOCUMENTED CASES OF GAS EMBOLISM.

SPECIALTY	MECHANISM OF GAS EMBOLISM
All medical specialties	Inadvertent entry of air through peripheral intravenous circuit
All surgical specialties	Intraoperative use of hydrogen peroxide, causing formation of arterial and venous oxygen emboli
Anesthesiology	Entry of air through disconnected intravascular catheter, inadvertent infusion of air through intravascular catheter
Cardiac surgery	Entry of air into extracorporeal-bypass pump circuit, incomplete removal of air from heart after cardioplegic arrest, carbon dioxide–assisted harvesting of peripheral veins
Cardiology	Entry of air through intravascular catheter during angiographic study or procedure
Critical care and pulmonology	Entry of air through disconnected intravascular catheter, pulmonary barotrauma, rupture of intraaortic balloon, entry of air into extracorporeal-membrane-oxygenator circuit
Diving and hyperbaric medicine	Pulmonary barotrauma, paradoxical embolism after decompression injury, entry of gas through disconnected intravascular catheter
Endoscopic and laparoscopic surgery	Entry of gas into veins or arteries during insufflation of body cavities
Gastroenterology	Entry of gas into veins during upper or lower endoscopy or endoscopic retrograde pancreatography
Neonatology and pediatrics	Pulmonary barotrauma in treatment of premature infants
Nephrology	Inadvertent entry of air through hemodialysis catheter and circuit on hemodialysis machine
Neurosurgery	Entry of air through incised veins and calvarial bone, especially during craniotomy with the patient in a sitting position
Obstetrics and gynecology	Cesarean section, gas insufflation into veins during endoscopic surgery, intravaginal and intrauterine gas insufflation during pregnancy
Otolaryngology	Nd:YAG laser surgery on the larynx, trachea, or bronchi*
Orthopedics	Gas insufflation into veins during arthroscopy, total hip arthroplasty, or spine surgery with the patient in a prone position
Radiology	Injection of air or gas as a contrast agent, inadvertent injection of air during angiography
Thoracic surgery	Entry of air into pulmonary vasculature during lung biopsy or video-assisted thoracoscopy, chest trauma (penetrating or blunt), lung transplantation
Urology	Entry of air during transurethral prostatectomy or radical prostatectomy
Vascular surgery	Entry of air during carotid endarterectomy

*Nd:YAG denotes neodymium:yttrium–aluminum–garnet.

by increasing the gradient for the egress of nitrogen from the bubble.[13] Rapid resuscitation with volume expansion is recommended to elevate venous pressure, thus preventing the continued entry of gas into the venous circulation.

Some authors recommend attempting to evacuate air from the right ventricle with the use of a central venous catheter (a multiorifice catheter may be more effective than one with a single lumen) or a pulmonary arterial catheter.[2,14,15] It may be possible to aspirate about 50 percent of the entrained air from an appropriately placed right atrial catheter,[2,14] but depending on the placement of the catheter and the position of the patient, a smaller effect is more likely.[2,15] Hyperbaric oxygen therapy is not a first-line treatment but may be a useful adjunct in severe cases. It should certainly be considered if there is evidence of neurologic changes. In this case, it should be assumed that a paradoxical embolism is present.

Paradoxical Embolism

A paradoxical embolism occurs when air or gas that has entered the venous circulation manages to enter the systemic arterial circulation and causes symptoms of end-artery obstruction. There are a number of mechanisms by which this can occur. One is the passage of gas across a patent foramen ovale into the systemic circulation. A patent foramen ovale, which is detectable in about 30 percent of the general population, makes possible right-to-left shunting of gas bubbles.[16] If there is a patent foramen ovale and if the pressure in the right atrium exceeds the pressure in the left atrium, right-to-left flow through the foramen ovale may occur.[17] Elevated pulmonary arterial pressure due to venous gas embolism may result in elevated right atrial pressure, making it possible for a bubble to be transported through a patent foramen. Furthermore, the decrease in left atrial pressure caused by controlled ventilation and the use of positive end-expiratory pressure may create a pressure gradient across the patent foramen ovale, favoring the passage of gas into the systemic circulation.[2,3]

In other situations, venous gas may enter the arterial circulation by overwhelming the mechanisms that normally prevent arterial gas embolism. Studies in animals suggest that either a large bolus of gas (20 ml or more) or small continuous amounts (11 ml per minute) introduced into the venous system may

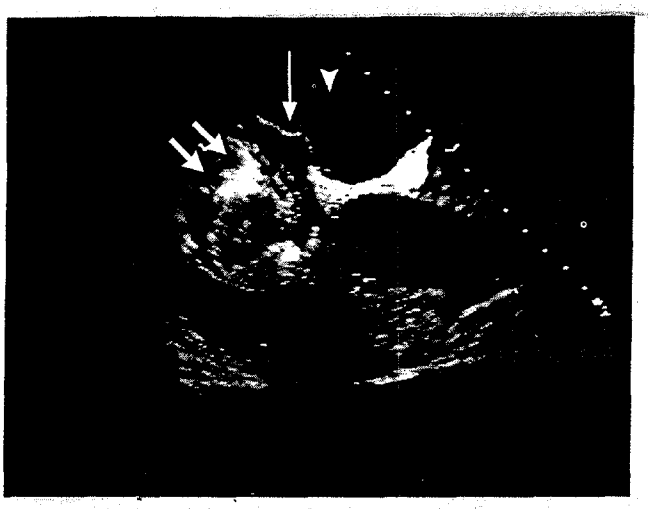

Figure 1. Transesophageal Echocardiogram from a Patient Evaluated for the Presence of a Patent Foramen Ovale.

Saline was agitated and injected rapidly into a central venous catheter. The bubbles appear as echodense areas in the right atrium (double arrows). If this patient had a patent foramen ovale, bubbles would be seen crossing the interatrial septum (thin arrow) and entering the left atrium (arrowhead). (Echocardiogram provided courtesy of S. Streckenbach.)

generate intraarterial bubbles.[18-20] There have been reports of fatal cerebral arterial gas embolism caused by a large venous gas embolus, although no intracardiac defects or shunt mechanisms could be demonstrated.[21] Various anesthetic agents diminish the ability of the pulmonary circulation to filter out gas emboli.[22] Studies in animals have shown that volatile anesthetics, specifically, may raise the threshold for a spillover of venous bubbles into systemic arteries. This finding may have relevance to surgical procedures that carry a substantial risk of venous gas embolism.

The treatment of paradoxical embolism is identical to that of primary arterial gas embolism (discussed below). Every venous gas embolism has the potential to evolve into an arterial gas embolism.

ARTERIAL GAS EMBOLISM

Arterial gas embolism is caused by the entry of gas into the pulmonary veins or directly into the arteries of the systemic circulation. Gas may enter the arteries as a result of overexpansion of the lung by decompression barotrauma or as a result of paradoxical embolism. Any cardiac surgical operation that uses extracorporeal bypass may also cause arterial gas embolism.[23] Even if only small amounts of gas enter the arterial system, the flow of gas bubbles into functional end arteries occludes these vessels. Although obstruction is possible in any artery, obstruction of either the coronary arteries or the nutritive arteries of the brain (cerebral arterial gas embolism) is especially serious and may be fatal because of the vulnerability of the heart and brain to short periods of hypoxia.[24]

Pathophysiology

The entry of gas into the aorta causes the distribution of gas bubbles into nearly all organs. Small emboli in the vessels of the skeletal muscles or viscera are well tolerated, but embolization to the cerebral or coronary circulation may result in severe morbidity or death.

Embolization into the coronary arteries induces electrocardiographic changes typical of ischemia and infarction; dysrhythmias, myocardial suppression, cardiac failure, and cardiac arrest are all possible, depending on the amount of gas embolized.[25] Circulatory responses may also be seen with embolization to the cerebral vessels.[26]

Cerebral arterial gas embolization typically involves the migration of gas to small arteries (average diameter, 30 to 60 μm).[25] The emboli cause pathologic changes by two mechanisms: a reduction in perfusion distal to the obstruction and an inflammatory response to the bubble (Fig. 2).[24]

Symptoms

The symptoms of cerebral arterial gas embolism develop suddenly. The clinical presentation, however, is determined by the absolute quantity of gas and the areas of the brain that are affected. Thus, there may be minor motor weakness and headache or moderate confusion; conversely, complete disorientation, hemiparesis, convulsions, loss of consciousness, and coma may occur.[27] Asymmetry of the pupils, hemianopia, and impairment of the respiratory and circulatory centers (manifested as bradypnea, Cheyne–Stokes breathing, cardiac arrhythmias, and circulatory failure)[25,26] are other well-known complications. In patients who have undergone surgical procedures that carry a risk of gas embolism, a delayed recovery from general anesthesia or a transitional stage of impaired consciousness may be a clue to the occurrence of cerebral arterial gas embolism. The diagnosis is not easy to establish in such patients, because complications of anesthesia, such as the central anticholinergic syndrome or the presence of residual anesthetic or muscle relaxant, can mimic mild cerebral arterial gas embolism.

Diagnosis

The most important diagnostic criterion is the patient's history, because the clinical suspicion of embolism is based on the initial neurologic symptoms and the direct temporal relation between these symptoms and the performance of an invasive procedure. The procedures that carry the greatest risk of venous or arterial gas embolism are craniotomy performed with the patient in the sitting position, cesarean section, hip replacement, and cardiac surgery with cardiopulmonary bypass. All these procedures have in common an incised vascular bed and a hydrostatic gradient favoring the intravascular entry of gas.

Cerebral arterial gas embolism can sometimes be

protection after cerebral ischemia, the use of barbiturates for ischemic brain lesions has certain advantages. They reduce cerebral oxygen consumption, intracranial pressure, the production of free radicals, and the release of endogenous catecholamines.[34,35] High doses of barbiturates depress respiration; ventilatory support must therefore be available when a patient is given barbiturates.

Hyperbaric-Oxygen Therapy

With hyperbaric-oxygen therapy, the patient breathes 100 percent oxygen at a pressure above that of the atmosphere at sea level. This therapy decreases the size of the gas bubble both by raising the ambient pressure (Fig. 3) and by causing systemic hyperoxia. An arterial partial pressure of oxygen greater than 2000 mm Hg is frequently achieved. The hyperoxia produces enormous diffusion gradients for oxygen into the bubble and for nitrogen out of the bubble.[31,32] The hyperoxia also allows much larger quantities of oxygen to be dissolved in the plasma and increases the extent of oxygen diffusion in tissues.[27] The improvements in the oxygen-carrying capacity of plasma and in the delivery of oxygen to tissues offset the embolic insult to the microvasculature.

Other benefits of hyperbaric oxygen have been proposed. It may help prevent cerebral edema by reducing the permeability of blood vessels while supporting the integrity of the blood–brain barrier.[36,37] Furthermore, experiments have suggested that hyperbaric oxygen diminishes the adherence of leukocytes to damaged endothelium.[38]

These benefits suggest that all patients with clinical symptoms of arterial gas embolism should receive recompression treatment with hyperbaric oxygen. Although immediate recompression produces the best response,[23] delayed treatment in a hyperbaric chamber may still be indicated to ameliorate the patient's condition.[39] Hyperbaric oxygen is the first-line treatment of choice for arterial gas embolism.[23,25,31,40] Thus, as soon as cardiopulmonary stabilization has been achieved, the patient should be transferred to a hyperbaric chamber.

Infusion Therapy

There is some evidence that gas embolism may cause hemoconcentration, which increases blood viscosity and impairs the already compromised microcirculation.[29] Therefore, normovolemia should be achieved to optimize the microcirculation. Hypovolemia is always tolerated less well than relative anemia. It is therefore acceptable to decrease the hematocrit, within certain limits. In animals, moderate hemodilution to a hematocrit of 30 percent reduces neurologic damage.[41] Colloid solutions are preferable to crystalloid solutions for hemodilution, because the latter may promote cerebral edema. Hypertonic solutions (e.g., 7.5 percent sodium chloride solution) may become the first choice for volume replacement, but their use is still controversial and therefore cannot yet be recommended.

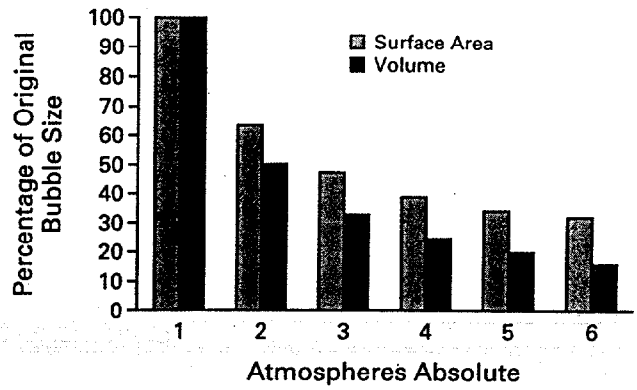

Figure 3. Relation between the Size of the Bubble and Pressure. The surface area and volume of the gas bubble are inversely proportional to pressure at a constant temperature (Boyle's law). Thus, as the patient is exposed to increasing ambient pressure, the gas bubbles shrink.

The goal of infusion therapy is normovolemia. Placement of a central venous catheter is strongly recommended to assess central venous pressure, which should be maintained at approximately 12 mm Hg. As a further method of ensuring adequate volume status, the urinary output should be monitored with a Foley catheter and maintained above 1 to 2 ml per kilogram of body weight per hour.

Anticoagulant Therapy

There is evidence that heparin may be beneficial in the treatment of gas embolism.[42] Studies have shown that the clinical course of arterial gas embolism is less severe if the patient has been treated with heparin before the embolic event occurs. An argument against heparin therapy is the risk it entails of hemorrhage into the infarcted tissue. At present, the use of heparin for the short-term treatment of cerebral arterial gas embolism is not generally recommended.

Corticosteroid Therapy

The use of corticosteroids in patients with arterial gas embolism remains controversial. Some authors recommend treatment with corticosteroids to combat the brain edema[43] that results from gas embolization in the cerebral arteries. Cerebral gas embolism initially induces the rapid development of cytotoxic brain edema, with diminished extracellular space and enlarged intracellular areas. This form of edema does not usually respond to corticosteroids.[44] Some authors report that corticosteroids aggravate ischemic injury after occlusion of the vessels.[45,46] Thus, since corticosteroids appear to offer no benefit in patients with cytotoxic edema and since these drugs may aggravate neuronal ischemic injury, we do not recommend them.

TABLE 2. TREATMENT OF GAS EMBOLISM.

TYPE OF TREATMENT	VENOUS GAS EMBOLISM	ARTERIAL GAS EMBOLISM
Prevention of further entry of gas	Measures to increase venous pressure (e.g., Valsalva maneuver or intravenous administration of fluids) Identification and shutting down of entryway for gas	Identification and shutting down of entryway for gas
Primary therapy	Supportive	Hyperbaric oxygen therapy as soon as patient's condition is stable enough for transfer to hyperbaric chamber
Supportive therapy	Oxygen, intravascular volume expansion, catecholamines	Oxygen, intravascular volume expansion, catecholamines
Positioning of patient	Supine, flat	Supine, flat
Evacuation of embolized gas	Aspiration with multiluminal central venous catheter (with patient in left lateral decubitus position)	Hyperbaric oxygen
Adjunctive therapy	Hyperbaric oxygen	Lidocaine, antiepileptic agents, physical therapy

Lidocaine Therapy

Although the results of clinical studies of lidocaine for the treatment of arterial gas embolism are not yet available, studies in animals suggest that lidocaine may be beneficial.[47-51] In animals given prophylactic doses of lidocaine, the depressant effects of gas embolism on somatosensory evoked potentials and the elevations in intracranial pressure caused by gas embolism were both reduced. In a clinical trial, lidocaine provided cerebral protection during cardiac surgery.[51] Therefore, a strong argument can be made for administering lidocaine in a bolus dose of 1.5 mg per kilogram and maintaining a therapeutic concentration, with continuous intravenous administration in patients with severe arterial gas embolism. However, an overdose of lidocaine may cause central nervous system depression, cerebral convulsions, and bradyarrhythmias.

CONCLUSIONS

The entry of gas into the venous or arterial system is a risk in virtually all areas of clinical care. Venous emboli may lead to cardiovascular collapse or to paradoxical arterial emboli. Arterial emboli may occlude end arteries throughout the body and may cause serious morbidity or death if they occlude cardiac or cerebral vessels.

Regardless of the mechanism responsible for the embolism, rapid and aggressive treatment is essential to preserve life and function (Table 2). For venous gas embolism, the mainstays of treatment are the prevention of further entry of gas, volume expansion, the administration of 100 percent oxygen, often with ventilatory support; positive inotropic support; and cardiopulmonary resuscitation, if necessary. For arterial gas embolism, hyperbaric oxygen is the treatment of choice, as soon as cardiopulmonary stabilization has been achieved.

REFERENCES

1. Murphy BP, Harford FJ, Cramer FS. Cerebral air embolism resulting from invasive medical procedures. Ann Surg 1985;201:242-5.
2. Palmon SC, Moore LE, Lundberg J, Toung T. Venous air embolism: a review. J Clin Anesth 1997;9:251-7.
3. Porter JM, Pidgeon C, Cunningham AJ. The sitting position in neurosurgery: a critical appraisal. Br J Anaesth 1999;82:117-28.
4. Donlon JV Jr. Anesthesia and eye, ear, nose, and throat surgery. In: Miller RD, ed. Anesthesia. 3rd ed. Vol. 2. New York: Churchill Livingstone, 1990:2001-23.
5. Halliday P, Anderson DN, Davidson AI, Page JG. Management of cerebral air embolism secondary to a disconnected central venous catheter. Br J Surg 1994;81:71.
6. Yu AS, Levy E. Paradoxical cerebral air embolism from a hemodialysis catheter. Am J Kidney Dis 1997;29:453-5.
7. Hill BF, Jones JS. Venous air embolism following orogenital sex during pregnancy. Am J Emerg Med 1993;11:155-7.
8. Weissman A, Kol S, Peretz BA. Gas embolism in obstetrics and gynecology: a review. J Reprod Med 1996;41:103-11.
9. Durant TM, Long J, Oppenheimer MJ. Pulmonary (venous) air embolism. Am Heart J 1947;33:269-81.
10. Shapiro HM, Drummond JC. Neurosurgical anesthesia and intracranial hypertension. In: Miller RD, ed. Anesthesia. 3rd ed. Vol. 2. New York: Churchill Livingstone, 1990:1737-89.
11. Gildenberg PL, O'Brien RP, Britt WJ, Frost EA. The efficacy of Doppler monitoring for the detection of venous air embolism. J Neurosurg 1981;54:75-8.
12. Mammoto T, Hayashi Y, Ohnishi Y, Kuro M. Incidence of venous and paradoxical air embolism in neurosurgical patients in the sitting position: detection by transesophageal echocardiography. Acta Anaesthesiol Scand 1998;42:643-7.
13. Van Liew HD, Conkin J, Burkard ME. The oxygen window and decompression bubbles: estimates and significance. Aviat Space Environ Med 1993;64:859-65.
14. De Angelis J. A simple and rapid method for evacuation of embolized air. Anesthesiology 1975;43:110-1.
15. Albin MS. Air embolism. In: Albin MS, ed. Textbook of neuroanesthesia with neurosurgical and neuroscience perspectives. New York: McGraw-Hill, 1997:1009-25.
16. Lynch JJ, Schuchard GH, Gross CM, Wann LS. Prevalence of right-to-left atrial shunting in a healthy population: detection by Valsalva maneuver contrast echocardiography. Am J Cardiol 1984;53:1478-80.
17. Gronert GA, Messick JM Jr, Cucchiara RF, Michenfelder JD. Paradoxical air embolism from a patent foramen ovale. Anesthesiology 1979;50:548-9.
18. Butler BD, Hills BA. The lung as a filter for microbubbles. J Appl Physiol 1979;47:537-43.
19. Vik A, Brubakk AO, Hennessy TR, Jenssen BM, Ekker M, Slørdahl SA. Venous air embolism in swine: transport of gas bubbles through the pulmonary circulation. J Appl Physiol 1990;69:237-44.
20. Spencer MP, Oyama Y. Pulmonary capacity for dissipation of venous gas emboli. Aerosp Med 1971;42:822-7.
21. Tommasino C, Rizzardi R, Beretta L, Venturino M, Piccoli S. Cerebral ischemia after venous air embolism in the absence of intracardiac defects. J Neurosurg Anesthesiol 1996;8:30-4.
22. Katz J, Leiman BC, Butler BD. Effects of inhalation anaesthetics on filtration of venous gas emboli by the pulmonary vasculature. Br J Anaesth 1988;61:200-5.
23. Ziser A, Adir Y, Lavon H, Shupak A. Hyperbaric oxygen therapy for

massive arterial air embolism during cardiac operations. J Thorac Cardiovasc Surg 1999;117:818-21.
24. Moon RE. Gas embolism. In: Oriani G, Marroni A, Wattel F, eds. Handbook on hyperbaric medicine. Milan, Italy: Springer, 1996:229-48.
25. Dutka AJ. A review of the pathophysiology and potential application of experimental therapies for cerebral ischemia to the treatment of cerebral arterial gas embolism. Undersea Biomed Res 1985;12:403-21.
26. Evans DE, Kobrine AI, Weathersby PK, Bradley ME. Cardiovascular effects of cerebral air embolism. Stroke 1981;12:338-44.
27. Tovar EA, Del Campo C, Borsari A, Webb RP, Dell JR, Weinstein PB. Postoperative management of cerebral air embolism: gas physiology for surgeons. Ann Thorac Surg 1995;60:1138-42.
28. Voorhies RM, Fraser RAR. Cerebral air embolism occurring at angiography and diagnosed by computerized tomography. J Neurosurg 1984; 60:177-8.
29. Smith RM, Van Hoesen KB, Neuman TS. Arterial gas embolism and hemoconcentration. J Emerg Med 1994;12:147-53.
30. Annane D, Troché G, Delisle F, et al. Effects of mechanical ventilation with normobaric oxygen therapy on the rate of air removal from cerebral arteries. Crit Care Med 1994;22:851-7.
31. Workshop Panel. Final summary of recommendations: diving accident workshop 1990. In: Bennett PB, Moon RE, eds. Diving accident management. Bethesda, Md.: Undersea and Hyperbaric Medical Society, 1990:366-9.
32. Moon RE, Dear GL, Stolp BW. Treatment of decompression illness and iatrogenic gas embolism. Respir Care Clin North Am 1999;5:93-135.
33. Bleck TP. Management approaches to prolonged seizures and status epilepticus. Epilepsia 1999;40:Suppl 1:S59-S66.
34. Patel PM, Drummond JC, Cole DJ, Kelly PJ, Watson M. Isoflurane and pentobarbital reduce the frequency of transient ischemic depolarizations during focal ischemia in rats. Anesth Analg 1998;86:773-80.
35. Hoffman WE, Charbel FT, Edelman G, Ausman JI. Thiopental and desflurane treatment for brain protection. Neurosurgery 1998;43:1050-3.
36. Miller JD, Ledingham IM, Jennett WB. Effects of hyperbaric oxygen on intracranial pressure and cerebral blood flow in experimental cerebral oedema. J Neurol Neurosurg Psychiatry 1970;33:745-55.
37. Mink RB, Dutka AJ. Hyperbaric oxygen after global cerebral ischemia in rabbits reduces brain vascular permeability and blood flow. Stroke 1995; 26:2307-12.
38. Thom SR, Mendiguren I, Hardy K, et al. Inhibition of human neutrophil beta2-integrin-dependent adherence by hyperbaric O2. Am J Physiol 1997;272:C770-C777.
39. Dexter F, Hindman BJ. Recommendations for hyperbaric oxygen therapy of cerebral air embolism based on a mathematical model of bubble absorption. Anesth Analg 1997;84:1203-7.
40. Peirce EC II. Specific therapy for arterial air embolism. Ann Thorac Surg 1980;29:300-3.
41. Reasoner DK, Ryu KH, Hindman BJ, Cutkomp J, Smith T. Marked hemodilution increases neurologic injury after focal cerebral ischemia in rabbits. Anesth Analg 1996;82:61-7.
42. Ryu KH, Hindman BJ, Reasoner DK, Dexter F. Heparin reduces neurological impairment after cerebral arterial air embolism in the rabbit. Stroke 1996;27:303-10.
43. Kizer KW. Corticosteroids in treatment of serious decompression sickness. Ann Emerg Med 1981;10:485-8.
44. Ganshirt H. Significance of hemorheology in diagnosis and therapy of ischemic cerebrovascular diseases: possibilities of therapy: antiedema procedures. Eur Neurol 1983;22:Suppl 1:78-82.
45. Sapolsky RM, Pulsinelli WA. Glucocorticoids potentiate ischemic injury to neurons: therapeutic implications. Science 1985;229:1397-400.
46. Dutka AJ, Mink RB, Pearson RR, Hallenbeck JM. Effects of treatment with dexamethasone on recovery from experimental cerebral arterial gas embolism. Undersea Biomed Res 1992;19:131-41.
47. Evans DE, Kobrine AI, LeGrys DC, Bradley ME. Protective effect of lidocaine in acute cerebral ischemia induced by air embolism. J Neurosurg 1984;60:257-63.
48. Evans DE, Catron PW, McDermott JJ, Thomas LB, Kobrine AI, Flynn ET. Effect of lidocaine after experimental cerebral ischemia induced by air embolism. J Neurosurg 1989;70:97-102.
49. Dutka AJ, Mink R, McDermott JJ, Clark JB, Hallenbeck JM. Effect of lidocaine on somatosensory evoked response and cerebral blood flow after canine cerebral air embolism. Stroke 1992;23:1515-20.
50. McDermott JJ, Dutka AJ, Evans DE, Flynn ET. Treatment of experimental cerebral air embolism with lidocaine and hyperbaric oxygen. Undersea Biomed Res 1990;17:525-34.
51. Mitchell SJ, Pellett O, Gorman DF. Cerebral protection by lidocaine during cardiac operations. Ann Thorac Surg 1999;67:1117-24.

FERTILITY AND STERILITY

Vol. 58, No. 4, Octobe
Printed on acid-free paper in

Massive air embolism—a possible cause of death after operati hysteroscopy using a 32% dextran-70* pump

Zohar Nachum, M.D.
Shahar Kol, M.D.†
Yochai Adir, M.D.
Yehuda Melamed, M.D.

Israeli Naval Hyperbaric Institute, Haifa, Israel

Although hysteroscopy is considered a safe procedure, potentially fatal complications such as cardiac CO_2 embolism and anaphylactic reaction have been reported (1, 2). We report a fatal outcome after operative hysteroscopy using a 32% dextran-70 pump.

CASE REPORT

A 27-year-old woman was admitted for operative hysteroscopy because of intrauterine adhesions diagnosed by hysterosalpingogram. Her past medical history was unremarkable. The procedure was performed under epidural block. Thirty-two percent dextran-70 in dextrose (Hyskon; Pharmacia, Uppsala, Sweden) was administered through a pump (Quinones pump; Storz, Tuttlingen, Germany). Approximately 75 minutes after the beginning of the operation, a Hyskon bottle was replaced, apparently while the pump was still functioning. The patient became restless and coughed. Severe bradycardia appeared (50 beats/min), followed by electromechanical dissociation lasting 30 seconds, cyanosis, and respiratory arrest. A generalized rash was also noted and disappeared a few minutes later. The procedure was terminated. During the vigorous resuscitative efforts, crepitant femoral pulses were palpated, and when an arterial line was introduce blood with foam was obtained. Initial arterial bloc gases were PCO_2, 77 mm Hg; PO_2, 16 mm Hg; pH 7.18. Massive air embolism was suspected by th attending team, and the patient was transferred t our institute for hyperbaric oxygen (HBO) therapy

On arrival at our institute, 3 hours after the event the patient was in deep coma and artificially ven tilated via an endotracheal tube. She was unrespon sive to painful stimuli, with dilated pupils unreactiv to light, and no tendon reflexes could be elicited Systolic blood pressure was 80 mm Hg with intravenous dopamine drip. Chest x ray revealed interstitial congestion compatible with pulmonary edema and ruled out pneumothorax. Arterial blood gases before initiation of HBO were (FIO_2, 1.0); PO_2, 40; PCO_2, 35; pH, 7.34; bicarbonate, 19; base excess, −6. Hyperbaric oxygen treatment was given in a multiplace hyperbaric chamber while the patient was ventilated with a gas mixture according to the following treatment protocol: [1] 30 minutes at 6 atmospheres of absolute (ATA), 50% O_2, 50% N_2. [2] Three cycles of 20 minutes at 2.8 ATA, 100% O_2. [3] 30 minutes slow decompression to 1.9 ATA, 100% O_2. [4] Two cycles of 60 minutes at 1.9 ATA, 100% O_2. During the treatment, a neurological examination revealed that the patient was in coma but had regained spontaneous breathing. Right hand movements were noted, both spontaneously and as a response to pain. The pupils were dilated and unreactive to light. General hypotonia was noted more on the left side. Biceps reflexes were weak bilaterally,

Received September 18, 1991; revised and accepted June 11, 1992.

* Hyskon, Pharmacia, Uppsala, Sweden.

† Reprint requests: Shahar Kol, M.D., Israeli Naval Hyperbaric Institute, Post Office Box 8040, 31 080 Haifa, Israel.

and the Achilles reflex was absent on the right side. Arterial blood gases were PO_2, 361; PCO_2, 42; pH, 7.23; bicarbonate, 18; base excess, −9. Throughout the 5-hour treatment the patient was hemodynamically unstable, and the pulmonary edema did not resolve. The patient was transferred to an intensive care facility, where she died 2 days later.

The main findings at autopsy were cerebral edema, hemorrhagic infarction of the left occipital lobe, epicardial and pleural effusions, pulmonary edema, and ascites. The heart was examined under immersion, and a few gas bubbles escaped from the left ventricle. Microscopic examination of the endometrium revealed inflammatory infiltrates, necrosis, and hemorrhage. The tubes and ovaries were normal. The pathologist's diagnosis was anaphylactic shock.

DISCUSSION

The pump used during the procedure can generate a maximal pressure of 300 mm Hg, which is sufficient to push air or dextran into the endometrial vascular bed during operative hysteroscopy. The same mechanism of introducing gas under pressure into a body cavity resulting in gas embolism has been reported in laparoscopy and in orogenital sex during pregnancy (3). The resultant massive venous air embolism would explain the acute respiratory failure, the electromechanical dissociation, and the prolonged pulmonary edema, as large volumes of air are trapped in the right ventricle and in the pulmonary vasculature.

Massive venous air embolism can lead to arterial embolism in one of two ways. First, the increase in right atrial pressure may open a potentially patent foramen ovale, which has been reported in 20% to 35% of the general population. Unfortunately, the pathologist did not rule out this possibility in our patient. Second, large volumes of air in the pulmonary vascular bed may be transferred to the left atrium by means of physiological pulmonary shunts, or by overcoming a critical volume.

The crepitant pulses, the frothy blood obtained through the arterial line, the sudden neurological and cardiovascular collapse at the time the pump was functioning without Hyskon, and the initial favorable reaction to HBO all support the diagnosis of massive arterial air embolism. Generalized rash may also accompany air embolism.

Ahmed et al. (4) describe three cases of severe anaphylactic reaction associated with the use of 32% dextran 70 during hysteroscopy. In all three cases, the anaphylactic reaction occurred approximately 10 minutes after exposure to dextran. The authors emphasize that sensitization may occur even without previous exposure to dextran. According to the pathologist's report in our case, the fact that shock occurred 75 minutes after exposure to dextran does not rule out the possibility that the cause of death was anaphylaxis.

The use of HBO for air embolism is well documented. The treatment is based on mechanical compression of the air bubbles to reduce their size (volume and pressure are inversely related). A reduction in the bubbles' volume relieves the vascular obstruction and restores perfusion. Concomitantly, HBO increases the gas gradient between the bubbles (mainly nitrogen) and the surrounding tissue (mainly oxygen) and hastens their resorption. Hyperoxygenation may oxygenate hypoxic neural tissue and by means of vasoconstriction reduce brain edema, which may result from gas embolism.

Hysteroscopy is generally a safe and reliable diagnostic and therapeutic procedure. However, the gynecologist must be aware of the possible serious complications. Cardiac CO_2 embolism and anaphylactic complications have both been described (1, 2). The practicing gynecologist must realize that the female genital tract is a potential portal of entry of gas into the venous plexus that drains the uterus; gas embolism has been reported after orogenital sex (3), criminal and therapeutic abortions (5), hysteroscopy (1), and cesarean section (6). The pregnant woman is at a higher risk of this complication. However, the possibility must be borne in mind when treating the nonpregnant patient too.

SUMMARY

Although considered a safe procedure, operative hysteroscopy has been reported to result in serious and even fatal complications. A fatal outcome is described after operative hysteroscopy. The attending team made a diagnosis of massive air embolism. However, HBO therapy, which is the specific treatment for air embolism, yielded only transient improvement. The pathologist's diagnosis on autopsy was anaphylaxis. These two complications must be borne in mind during the

procedure, and a contingency plan developed for dealing with them should they arise.

Key Words: Hysteroscopy, complications, dextran-70, air embolism, anaphylaxis, hyperbaric oxygenation.

REFERENCES

1. Brundin J, Thomasson K. Cardiac gas embolism during carbon dioxide hysteroscopy: risk and management. Eur J Obstet Gynecol Reprod Biol 1989;33:241–5.
2. Trimbos-Kemper TCM, Veering BT. Anaphylactic shock from intracavitary 32% dextran-70 during hysteroscopy. Fertil Steril 1989;51:1053–4.
3. Bray P, Myers RAM, Cowley RA. Orogenital sex as a cause of nonfatal air embolism in pregnancy. Obstet Gynecol 1983;61:653–7.
4. Ahmed N, Falcone T, Tulandi T, Houle G. Anaphylactic reaction because of intrauterine 32% dextran-70 instillation. Fertil Steril 1991;55:1014–6.
5. Munsick RA. Air embolism and maternal death from therapeutic abortion. Obstet Gynecol 1972;39:688–90.
6. Malinow AM, Naulty JS, Hunt CO, Datta S, Ostheimer GW. Precordial ultrasonic monitoring during cesarean delivery. Anesthesiology 1987;66:816–9.

CASE REPORTS

Anesthesiology
73:546–547, 1990

A Complication of Hysteroscopy: Air Embolism

PATRICIA M. PERRY, M.D.,* VERNA L. BAUGHMAN, M.D.†

Hysteroscopy is an established gynecologic procedure that has been used for the past 20 yr as a diagnostic technique. It is also used therapeutically to remove foreign bodies and intrauterine devices and more recently to treat Asherman's syndrome, excise uterine septa and submucosal myomas, and for endometrial ablation for intractable uterine bleeding. These newer applications use the neodymium:yttrium-aluminum-garnet (Nd:YAG) laser or the urologic resectoscope. We present two cases of air embolism during operative hysteroscopy using the Nd:YAG laser and the resectoscope.

CASE REPORTS

Case 1. Patient 1 was a healthy 35-yr-old woman. ASA Physical Status 1, weighing 65 kg, scheduled for outpatient laser hysteroscopy for menorrhagia. General anesthesia was induced with thiopental, and succinylcholine was used to facilitate tracheal intubation. Anesthesia was maintained with isoflurane, nitrous oxide, and oxygen, and vecuronium was administered for relaxation. Intraoperative monitors included blood pressure cuff, ECG, pulse oximeter, temperature probe, and capnograph. The cervix was dilated, and a dual-channel hysteroscope with attached video camera was inserted into the endometrial cavity. The uterus was distended using lactated Ringer's solution in 5-l bags connected to clear y-type TUR tubing with gravity drainage. A 600-μm bare laser fiber was inserted through the accessory portal. The endometrium was ablated using a combination of "dragging touch" and "nontouch" techniques at powers of 50–65 watts. Intermittently the uterine cavity was drained and redistended. Approximately 30 min into the case "excessive bubbling" was noted within the fluid distending the uterine cavity. Some bubbles had been noted in the tubing of the irrigant solution as bags were changed. The end-tidal CO_2 decreased from 34 to 22 mmHg. The gynecologist discontinued laser ablation and uterine irrigation. One hundred percent oxygen was administered. The patient remained hemodynamically stable; thus, patient position was not changed. The end-tidal CO_2 returned to normal within 3–4 min. A precordial doppler was added as an additional monitor and surgery resumed. Again, the end-tidal CO_2 decreased from 35 to 21 mmHg with a change in the doppler tones characteristic of air embolism. No bubbles had been noted in the tubing. The surgical procedure was discontinued and the lungs were ventilated with 100% oxygen. Again, there were no hemodynamic changes and the end-tidal CO_2 returned to normal within minutes without additional treatment. Neuromuscular blockade was reversed, the trachea was extubated, and the patient was taken to the recovery room awake and subsequently discharged without neurologic or cardiac sequelae.

Case 2. Patient 2 was a 39-yr-old woman, ASA Physical Status 1, weighing 65 kg, scheduled for operative hysteroscopy using a resectoscope for symptomatic uterine fibroids. Following preanesthetic medication with midazolam, glycopyrrolate, *d*-tubocurarine, and droperidol, general anesthesia was induced with thiopental and tracheal intubation facilitated with succinylcholine. Anesthesia was maintained with isoflurane, oxygen, air, and fentanyl, and vecuronium was added for relaxation. Monitors used were a blood pressure cuff, ECG, pulse oximeter, capnograph, precordial doppler, temperature probe, nerve stimulator, and stethoscope. Following cervical dilation a resectoscope was inserted into the uterine cavity. Uterine distention was achieved with 1.5% glycine solution attached to tubing suspended above the patient. Glycine solution was released from the uterus intermittently *via* a port of the resectoscope controlled by the surgeon. A wire loop was placed in the endometrial cavity, and cutting and coagulation modes were used for resection and hemostasis.

After 30 min of surgery, while the uterus was being irrigated using a urologic bulb syringe, the doppler sounds suddenly increased, pulse oximeter readings decreased from 99% to 90%, end-tidal CO_2 decreased from 31 to 17 mmHg, and the blood pressure decreased from 120/80 to 90/60 mmHg. Hysteroscopy was suspended and the lungs were ventilated with 100% oxygen. The end-tidal CO_2 had spontaneously increased to 25 mmHg when an arterial blood gas was drawn and showed a $pH = 7.30$, $Pa_{CO_2} = 43$, $Pa_{O_2} = 267$, and hemoglobin saturation = 98%. The patient's pulse oximeter readings, end-tidal CO_2, and blood pressure returned to baseline within 5 min without additional treatment and hysteroscopy was resumed. A central venous catheter was inserted following an increase in peak ventilatory pressures from 24 to 40 cm water and concern about both intrauterine fluid absorption and further air emboli. No air was aspirated nor were other signs of air embolism noted. The initial central venous pressure was 25 mmHg and plasma sodium was 121 mEq/l, indicating a dilutional hyponatremia and hypervolemia. Treatment was initiated with 10 mg iv furosemide, resulting in a diuresis of approximately 2 l over the next 2 h. The operative hysteroscopy portion of the surgery lasted for 90 min and was followed by a laparotomy for myomectomy, with total surgical time of 150 min. At completion of surgery neuromuscular blockade was reversed and the patient's trachea was extubated without incident in the operating room. The patient was discharged home 2 days later without sequelae.

DISCUSSION

Complications of hysteroscopy result primarily from the media used for distending the uterus. The distending medium for hysteroscopy done as an office procedure is CO_2, although small infusions of dextran 70 are sometimes used to irrigate out blood, mucus, smoke, and de-

* Associate Attending Physician; Assistant Professor of Anesthesiology.

† Attending Physician; Assistant Professor of Anesthesiology.

Received from the Department of Anesthesiology, Michael Reese Hospital and Medical Center, and the University of Illinois College of Medicine, Chicago, Illinois. Accepted for publication February 6, 1990.

Address reprint requests to Dr. Perry: Department of Anesthesiology, Michael Reese Hospital and Medical Center, Lake Shore Drive at 31st Street, Chicago, Illinois 60616.

Key words: Air embolism. Surgery: hysteroscopy; laser.

bris.‡* Siegler and Valle[1] warned that intrauterine gas pressures should not exceed 200 mmHg and that CO_2 flow should not exceed 100 ml/min at standard temperature and pressure to avoid air embolus. During operative hysteroscopy a liquid distending medium is used. Uterine distention is achieved by gravity flow or manual pumping of the liquid into the uterus with outflow controlled by an outflow catheter, leakage around the laser or resectoscope, or removal of the instrument from the cervix.[1-3]

With either the Nd:YAG laser or resectoscope, endometrial blood vessels are disrupted and bleeding occurs. A high distending pressure tamponades blood vessels and reduces blood loss.[1,2,4] However, fluid absorption occurs *via* the raw surface of severed venous channels.[5-8]

The possibility of gas embolization from gas under pressure and a denuded endometrium was reported by Loffer[2] and Baggish and Baltoyannis.[3] They noted that the use of a coaxial quartz fiber with sapphire tip on the Nd:YAG laser required a gas or liquid coolant transmitted *via* a sheath to the fiber tip and that introduction of a gas into the distended uterus could pose a risk of embolism. Baggish and Daniell[9] recently reported two deaths from gaseous emboli and cardiovascular collapse in Nd:YAG laser operative hysteroscopies using such sheathed coaxial fibers and sapphire tips cooled with nitrogen and air. Prior to introduction of sapphire tips in 1985, however, bare fibers were used that required no cooling mechanism, and these remain in use.

We report two more cases of air emboli. For laser hysteroscopy our gynecologists use the bare fiber requiring no coolant instead of the coaxial sapphire tip. One possible explanation for our cases is air in the irrigation solution entering open venous sinuses under pressure. Another possible explanation is the venous absorption of some of the bubbles from vaporization of tissue. Gas bubbles, endometrial fragments, blood, and debris occur with laser vaporization and resectoscope coagulation of endometrial tissue and are evacuated from the uterus by the irrigant.[2-4] These bubbles may contain particulates, CO_2, carbon monoxide, and other products of combustion.§ Although they have been observed to wash out of the uterus, it merits investigation whether these bubbles under pressure can pass into the disrupted vasculature of the distended uterus without immediately dissolving in the blood.

Our precautions and recommendations for operative hysteroscopy therefore now include continuous precordial doppler monitoring in addition to end-tidal CO_2 monitoring and pulse oximetry. We do not recommend routine insertion of central venous catheters, but in the event of a suspected embolus not immediately resolving on halting surgery, insertion of such a catheter should be considered along with repositioning the patient in left lateral decubitus position. We also avoid nitrous oxide to prevent enlarging an occult air embolus.[10] Special attention is paid to complete evacuation of air from the irrigation fluid and tubing. Finally, prospective studies using intraoperative transesophageal echocardiography or precordial doppler ultrasound in cases of operative hysteroscopy would be helpful to clarify the risk of gas embolization during this procedure.

‡ Neuwirth RS: Some new applications for hysteroscopy. Contemp OB/Gyn Special Issue 11–28, 1987

§ Mitchell MM: Anesthesia for laser surgery: General considerations. Refresher Courses in Anesthesiology 14:159–169, 1986

References

1. Siegler AM, Valle RF: Therapeutic hysteroscopic procedures. Fertil Steril 50:685–701, 1988
2. Loffer FD: Laser ablation of the endometrium. Obstet Gynecol Clin North Am 15:77–89, 1988
3. Baggish MS, Baltoyannis P: New techniques for laser ablation of the endometrium in high-risk patients. Am J Obstet Gynecol 159:287–292, 1988
4. Goldrath MH, Fuller TA, Segal S: Laser photovaporization of endometrium for the treatment of menorrhagia. Am J Obstet Gynecol 140:14–19, 1981
5. Leake JF, Murphy AA, Zacur HA: Noncardiogenic pulmonary edema: A complication of operative hysteroscopy. Fertil Steril 48:497–499, 1987
6. Pellicer A, Diamond MP: Distending media for hysteroscopy. Obstet Gynecol Clin North Am 15:23–28, 1988
7. Zbella EA, Moise J, Carson SA: Noncardiogenic pulmonary edema secondary to intrauterine instillation of 32% dextran-70. Fertil Steril 43:479–480, 1985
8. Lavy G, Diamond MP, Shapiro B, DeCherney AH: A new device to facilitate intrauterine instillation of dextran 70 for hysteroscopy. Obstet Gynecol 70:955–957, 1987
9. Baggish MS, Daniell JF: Death caused by air embolism associated with neodymium: Yttrium-aluminum-garnet laser surgery and artificial sapphire tips. Am J Obstet Gynecol 161:877–878, 1989
10. Munson EJ, Merrick HC: Effect of nitrous oxide on venous air embolism. ANESTHESIOLOGY 27:783–787, 1966

a twofold increase of the risk of testicular cancer at the 0·05 level of significance.[1] It is doubtful that one large cohort study could, in fact, produce definitive results to confirm or negate the putative association.

The sample size needed to achieve the desired study power in a case-control study is determined by the prevalence of vasectomy rather than by the incidence of testicular cancer. In a number of countries, including the United Kingdom, about 10% of couples of reproductive age rely on vasectomy for contraception.[2] With this prevalence, a case-control study will need 221 pairs to detect a twofold increased risk for $\alpha = 0{\cdot}10$ (two sided) and $\beta = 0{\cdot}20$. Only 78 pairs are needed to detect a threefold difference in relative risks for same study power.[3] The prevalence of vasectomy should be higher in big cities where more hospitals are located, and thus fewer testicular cancer patients will be needed. If, in practice, the needed number of cases cannot be reached in a reasonable period of time then multicentre study, retrospective case finding, and increasing the number of controls relative to cases offer solutions. Little suspicion of such an association is entertained by the general population and the exposure variable (vasectomy) is an event not easily forgotten by the study subjects. Both these aspects will help avoid the bias due to selective recall and memory decay, and they will add validity to this much less costly and more easily manageable approach, which produces results more quickly. I believe that the case-control approach is the method of choice.

I-CHENG CHI

Family Health International,
Research Triangle Park,
North Carolina 27709,
United States

1 Cale ARJ, Farouk M, Prescott RJ, Wallace IEJ. Does vasectomy accelerate testicular tumour? Importance of testicular examinations before and after vasectomy. *Br Med J* 1990;300:370. (10 February.)
2 Shottenfeld D, Warshauer M, Sherlock S, *et al*. The epidemiology of testicular cancer in young adults. *Am J Epidemiol* 1980;112:232-46.
3 Moss AR, Osmond D, Bacchetti P, *et al*. Hormonal risk factors in testicular cancer: a case control study. *Am J Epidemiol* 1986;124:39-52.
4 Strader CH, Weiss NS, Daling JR. Vasectomy and the incidence of testicular cancer. *Am J Epidemiol* 1988;128:56-63.
5 Liskin L, Pile JM, Quillin WF. Vasectomy safe and simple. *Popul Rep* [D] 1983;No 4:85.
6 Schlesselman JJ. Sample size requirements in cohort and case-control studies of diseases. *Am J Epidemiol* 1974;99:381-4.

Air embolism during transcervical resection of endometrium

SIR,—Dr Ralf Baumann and colleagues in their report point out the risk during transcervical resection of the endometrium from systemic absorption of irrigating fluid from the denuded myometrium and through transtubal loss.[1] We have attempted to reduce transtubal absorption by combining laparoscopic tubal occlusion (using Silastic rings) with transcervical endometrial resection. In nine cases by this combined technique the mean fluid deficit was 259 ml (range 0-900 ml) compared with 643 ml (100-2030 ml) reported by Dr Baumann and colleagues. This small number of cases is insufficient to prove the value of tubal occlusion, but the study gave us the opportunity of observing a hitherto undescribed hazard of endometrial resection.

A 46 year old woman suffering from uncontrolled menorrhagia was found at hysteroscopy to have a small (diameter of 1 cm) adenomatous endometrial polyp. After two months' treatment with danazol she underwent hysteroscopic resection of the polyp followed by total endometrial resection by a modification of the diathermy loop technique.[2] General anaesthesia was induced with propofol and maintained with nitrous oxide, oxygen, and isoflurane breathed spontaneously through a laryngeal mask. The electrocardiogram, blood pressure, and pulse oximetry were monitored continuously. Dextran 40 (10%) was used as the irrigating fluid: 4900 ml was used, of which 4300 ml was recovered.

On completion of the resection the pelvic organs were inspected by laparoscopy, with the patient in a head down position. The uterus was intact but there was congestion of the broad ligaments, from which air bubbles could be seen entering small calibre veins in the lateral pelvic wall. Over the next four minutes the patient's oxygen saturation fell from 97% to 84% and her pulse rate rose from 72 to 110 beats/min, consistent with air embolism. Her blood pressure was unchanged. The head down position was reversed and positive pressure ventilation started with 100% oxygen. The oxygen saturation and pulse returned to normal within 10 minutes and the patient made a full recovery.

Air emboli can occur during surgery whenever a vein is open and the air pressure exceeds venous pressure. This can be the result of a rise in air pressure, such as occurs during insertion of a cemented femoral prosthesis,[3] or a fall in venous pressure caused by raising the operative site above the level of the heart, which is a particular risk when the patient is breathing spontaneously. Air emboli have been reported to occur during neurosurgery[4,5] and less commonly during caesarean section[6] and transurethral resection of the prostate.[7]

We present these data to warn others embarking on the newly developed procedure of transcervical resection of the endometrium that an anaesthetic technique employing positive pressure ventilation may be preferred. Our experience also suggests that patients should not be nursed in a head down position after operation.

S M WOOD
F L ROBERTS

Royal Devon and Exeter Hospital,
Exeter EX1 2ED

1 Baumann R, Magos AL, Kay JDS, Turnbull AC. Absorption of glycine irrigating solution during transcervical resection of endometrium. *Br Med J* 1990;300:304-5. (3 February.)
2 Magos AL, Baumann R, Turnbull AC. Transcervical resection of endometrium in women with menorrhagia. *Br Med J* 1989;298:1209-12.
3 Michel R. Air embolism in hip surgery. *Anaesthesia* 1980;35:858-62.
4 Michenfelder JD, Miller RH, Gronert GA. Evaluation of an ultrasonic device (Doppler) for the diagnosis of venous embolism. *Anesthesiology* 1972;36:164-7.
5 Clayton DG, Evans P, Williams C, Thurlow AC. Paradoxical air embolism during neurosurgery. *Anaesthesia* 1985;40:981-9.
6 Younker D, Rodriguez V, Kavanagh J. Massive air embolism during caesarean section. *Anesthesiology* 1986;65:77-9.
7 Hofsess DW. Fatal air embolism during transurethral resection (prostate). *J Urol* 1984;131:355.

Portal vein thrombosis in myeloproliferative disease

SIR,—The extensive thrombosis of the portal venous system that may accompany the myeloproliferative diseases was clearly described in the recent Hammersmith Hospital staff round report.[1] Haematological disorders, however, represent only one of several possible aetiologies of portal vein occlusion, which include congenital malformations, trauma, sepsis, tumours, and invasive procedures such as catheterisation of the umbilical vein. Our recent analysis of portal venous occlusion in 27 patients suggested a relation between the aetiology and the anatomical distribution of the occlusion. Each patient was investigated with digital subtraction angiography, and the anatomical features were interpreted by one observer (JK), who was without knowledge of the clinical history of the case. The angiographic features were classified into four main groups:

(1) Seven patients had occlusion of the main portal vein and bifurcation. Five of these had no history of important illness and one patient had undergone an operation for duodenal atresia. Cavernous transformation of the portal vein was seen in patients in this group, which might have represented a congenital anomaly of the portal vein.

(2) Five patients had occlusion of the main portal vein and distal intrahepatic branches. Four of these had a history of severe illness and two had undergone neonatal catheterisation of the umbilical vein.

(3) Six patients had total occlusion of the portal venous system excluding the splenic vein. Five of this group had been treated for abdominal problems and two had undergone catheterisation of the umbilical vein.

(4) Nine patients had total occlusion of the portal venous system. Six of these patients had haematological abnormalities including polycythaemia, prekallikrein deficiency, and myeloid dysplasia. Three had associated hepatic vein occlusion (the Budd-Chiari syndrome). No haematological anomaly was found in three patients, who had histories of pancreatitis, surgical trauma to the superior mesenteric artery, and catheterisation of the umbilical vein.

The preliminary analysis of patients with portal venous occlusion suggests that congenital anomalies may spare the splenic and superior mesenteric veins, which may be used for surgical decompression if indicated. At the other end of the range total occlusion of the splanchnic veins may indicate an underlying haematological disorder. Catheterisation of the umbilical vein and intra-abdominal sepsis are associated with occlusions of variable extent that usually extend beyond the portal vein itself.

In conclusion, accurate visualisation of the anatomical distribution of venous occlusion may be helpful in determining the aetiology of as well as in managing extrahepatic portal hypertension.

NIGEL HEATON
JOHN KARANI
EDWARD HOWARD

King's College Hospital,
London SE5 9RS

1 Fidler H. Portal vein thrombosis in myeloproliferative disease. *Br Med J* 1990;300:590-2. (3 March.)

Clinical directorates

SIR,—Mr J N Johnson's editorial on clinical directorates raises several important issues in the style of hospital management, both the version that has evolved (and is still evolving) at Guy's Hospital and the predictably different versions that need to be constructed flexibly to fit local circumstances.[1] I will do little more than offer a few personal observations from the vantage of my fifth (and last) year as director of clinical services in medicine at Guy's Hospital. Some of my fellow directors may see things in a different light; Professor Cyril Chantler, who was largely responsible for introducing the system at Guy's, has written about it before.[2]

I agreed to take on the task reluctantly, seeing it partly as a device for administering unpleasant medicine to my colleagues which they might accept less readily from a lay manager. There was indeed much foul tasting medicine to be swallowed in our first few years as we "slimmed down" to try to live within budget. It became clear that we had to do that if only to practise the best medicine possible under the circumstances; it was equally clearly my responsibility to identify where that "best" was not good enough and to argue forcibly against the constraints, sometimes almost crippling, that we