Tracheoesophageal Fistulas Following Prolonged Artificial Ventilation via Cuffed Tracheostomy Tubes

MICHAEL HEDDEN, F.F.A.R.C.S.,* CLARA JEAN EROZ, M.D.,† PETER SAFAR, M.D.♦

Since 1952, tracheostomy cannulas have been used for long-term ventilatory support.1–3 Under these circumstances, more severe tracheal trauma than hitherto seen may occur.2,4 Such trauma has resulted in tracheoesophageal fistula in a number of reported cases. Several authors have incriminated tracheostomy tube cuffs,2,5 others the tip of the tracheostomy cannula,5 and some believe the evidence does not allow separation of these factors.6,7

In the Intensive Care Unit of Presbyterian–University Hospital, over a two-year period (9/1/65 to 8/31/67), 101 patients received prolonged artificial ventilation via cuffed tracheostomy tubes. Two patients developed tracheoesophageal fistulas and a third, severe tracheal-wall necrosis without fistula. These cases are reported because they suggest variables in patient and treatment which affect the development of tracheal trauma.

CASE REPORTS

Patient 1. Guillain-Barré Syndrome

A 43-year-old housewife was admitted to a community hospital with a four-day history of progressive numbness and paralysis. Previous medical history was unremarkable except for complete situs inversus and resection of a pheochromocytoma 11 years before.

On admission the patient was quadriplegic and anesthetic from the neck down. Respirations were assessed as adequate. Twelve hours later she was cyanotic, semiconscious and hypotensive. Orotracheal intubation was performed with a #36 cuffed, red rubber, orotracheal tube and respirations were assisted, with improvement in consciousness. One dose of vasopressor drug corrected the hypotension. Since stomach contents were aspirated from the tracheobronchial tree after intubation, hydrocortisone and chloromycetin were given. The patient, breathing spontaneously via an endotracheal tube, subsequently was transferred to the Presbyterian–University Hospital (PUH).

On arrival she was cyanotic, with shallow rapid respirations. Controlled ventilation with 100 per cent oxygen was initiated immediately. Following oxygenation, tracheostomy was performed and a #6 silver Jackson tracheostomy tube with a Moeach double-swivel adapter and a #8 Anesthesia Associates slip-on inflatable cuff was inserted.21 A similar tube, changed at intervals, was used until day 24 when an orotracheal tube was again inserted. Controlled ventilation was continued from day 1 until day 21 with a Bennett ventilator set on air dilution. A Puritan heated nebulizer provided humidification. From day 21 until death on day 26, controlled ventilation with 100 per cent oxygen was required to sustain adequate Pao2.

Although the patient became fully conscious after the initial resuscitative efforts, the motor and sensory deficit persisted with no regression to the time of death. CSF examination, electromyographic studies, and autopsy confirmed the diagnosis of polyneuropathy of the Guillain-Barré type. Roentgenogram of the chest on day 1 showed infiltrates in the right lower and left upper lobes. Over the next four days the lungs improved. Sohls-Caref®, 100 mg t.i.d., was continued to day

* Instructor.
† Assistant Professor.
♦ Professor and Chairman.

Received from the Department of Anesthesiology, University of Pittsburgh School of Medicine and Presbyterian–University Hospital, Pittsburgh, Pennsylvania. Supported by U. S. Army contract DA-49-193-2160.
20, thereafter reduced, and discontinued on day
25. Erythromycin was given from admission to
day 6. Persistent cultures of Klebsiella and Aero-
bacter Aerogenes necessitated treatment with Cephi-
losporin. Streptococcus faecalis appeared in the
spum on day 23 and streptomycin was begun.
A nasogastric tube (French size 16, Pharmacalab
atories K-10) was inserted on day 1 and re-
mained in position until death. It was used for
feeding until day 21.

On day 18 small quantities of material similar to
the feeding were removed from the trachea during
suction. This was, at first, thought to represent
spillover past the tracheostomy cuff. Prior to day
16 the cuff had produced an airtight fit with less
than 6 ml of air. However, by day 21, 10 ml of
air were necessary to prevent leakage. At this
stage methylene blue instilled into the pharynx
promptly appeared in the trachea. A tracheo-
esophageal fistula was presumed present and naso-
gastric feedings were discontinued.

On day 21, 10 ml of air in the tracheostomy
tube cuff would not produce an airtight seal, and a
Moerch volume-cycled ventilator with large
stroke volumes was used to compensate for the
leak. On day 54, an orotrachealuffed #36 neo-
prene tube (Rusch-Forreger) was inserted, im-
poving ventilation for a short time. However, as
ventilation difficulties increased, cardiovascular in-
sufficiency developed and the patient died on day
27 following a period of ventricular tachycardia
and hypotension.

Autopsy. Starting 1 cm below the inferior mar-
gin of the tracheal stoma, involving rings 8 and 9,
the posterior wall of the trachea was completely
eroded over an area of approximately 4 cm² (fig.
1A), with a connection to the lumen of the esoph-
gus. The fistula on the esophageal side measured
2.5 x 0.5 cm (fig. 1B). The fistula site corre-
sponded to the herniating portion of the cuff (fig.
1C). There was gross esophagitis with proximal
ulceration. The right lung weighed 1,250 g and the
left, 880 g. Severe bilateral bronchopneumonia
with areas of consolidation in all lobes and dif-
suse small abscesses filled with yellow-green pus
were seen.

Patient 2. Poliomyelitis

A 31-year-old man was admitted to the ICU
with a 12-hour history of progressive weakness of
the legs and urinary retention preceded by fever
and chills. There was no sensory loss.

In the previous year, the patient had had pneu-
monia three times. Three weeks prior to admiss-
ion he underwent nasal-sinus surgery. During
that hospitalization a bronchogram revealed bron-
chiectasis. A diagnosis of acquired agammaglobu-
linemia was established after paper electrophoresis
showed a gamma-globulin of 0.2 g per cent and
absence of immunoglobulins G, A, and M.

Over the three days following admission the pa-
tient demonstrated a rapidly progressive ascending
paralysis. The spinal fluid showed pleocytosis and
moderately elevated protein. Diagnoses ente-
tained at this time were Guillain-Barré syndrome
and poliomyelitis, although the patient had re-
eceived poliomyelitis vaccines in the past. He was
placed in isolation and vital capacities were deter-
mined hourly to document the progression of pa-
ralysis once it had reached the thoracic level. For
one day, respirations were assisted via an otrora-
cheal #36 neoprene tube (Rusch-Forreger) with a
Benett ventilator employing a heated nebulizer.
Then tracheostomy was performed and a silver
Jackson tracheostomy tube, size 8 with a Moerch
double-swivel adapter and a #6 Anesthesia Asso-
ciates slip-on inflatble cuff, was inserted. A simi-
lar tube, changed at intervals, was used until death.

A plastic nasogastric tube (French size 16, Phar-
macia Laboratories K-10) was inserted. This was
replaced on day 40 by a Steri-loc plastic nasoga-
stric feeding tube, French size 8, and on day 66
by a feeding gastrostomy tube. For the next 40
days ventilation was provided with a volume-
cycled Emerson ventilator with heated humidifi-
ation. Oxygen enrichment was used as necessary
to ensure $P_{A}O_{2}$ between 150 and 200 mm Hg. By
day 4 there was complete peripheral motor pa-
ralysis and significant motor involvement of all
cranial nerves. At this stage he could only slightly
open his eyes and move his tongue.

Until day 40 less than 6 ml of air were neces-
sary in the tracheostomy cuff to provide a seal.
However, between day 40 and day 50, the vol-
ume gradually rose to 13 ml. For the next five
days, no cuff was used and the leak was handled
by increasing tidal volumes. However, on day
52 due to mechanical failure, ventilation ceased
abruptly and the patient had a short period of
hypotension. With the reinstitution of ventilation
and a brief period of external cardiac massage,
the patient's mental status returned to its previous
level without the aid of drugs. The cuff was reinflated
and for a few days small volumes of air were suffi-
cient to provide a seal. However, by day 79 such a
seal could be provided only by 10 ml of air in the
cuff, subsequently 15. A Moerch ventilator was
used from day 62 to day 81. On day 81 another
brief period of hypotension occurred, which was
affected by therapy as before. The patient's men-
tal status returned to its previous level. The Em-
erson Respirator was used from day 81 onwards.

Steroid therapy was administered for six days
from day —1 until day 5. When the first positive
stool culture of Type 2 poliovirus was obtained,
stereos were discontinued. Over the ensuing 48
hours, 400 ml of gamma-globulin were given at the
rate of 60 ml every six hours.

Various organisms were grown from the sputum
during the patient's hospital course. However,
with constant respiratory care and suitable anti-
biotics, it was not until day 50 that infiltrations
appeared on the chest x-ray. From this point on,
Fig. 1. Patient 1. A (above), tracheoesophageal fistula seen from trachea. B (above right), tracheoesophageal fistula seen from esophagus. C (right), cannula repositioned in autopsy specimen to simulate in vivo relationships. Tracheal erosion is seen at the site of eccentrically inflating cuff.

Chest x-rays showed steadily progressing infiltration, with atelectasis in both lower lobes.

There was no evidence of communication between the trachea and the esophagus until, suddenly, on day 95 severe gastric distention developed. The gastrostomy tube was unclamped and placed under water, where it was seen to bubble with each inspiration. A tracheoesophageal fistula was presumed to have occurred. There was increasing difficulty in maintaining adequate oxygenation, and on day 99 the patient became hypotensive and died.

Autopsy. Starting 2.5 cm below the inferior margin of the tracheal stoma and involving rings 3 and 4, the posterior wall of the trachea was completely eroded over an area of 5 cm² (Fig. 2A), with a small connection to the lumen of the esophagus. The fistula on the esophageal side measured 1 x 0.5 cm (Fig. 2B). A postmortem roentgenographic study revealed that the tip of the cannula impinged against the anterior tracheal wall and had no relationship to the fistula, which was lo-
Fig. 2. Patient 2. A (above left), tracheoesophageal fistula seen from trachea. B (above), tracheoesophageal fistula seen from esophagus. C (left), lateral chest roentgenogram taken at autopsy. Tubes filled with barium inserted through esophagus (tube A) and via tracheostomy through fistula into esophagus (tube B). Tracheostomy cannula of the same size and type as that used prior to death repositioned and cuff inflated with 10 ml of air. Tip of cannula impinging against anterior tracheal wall. Fistula at site of cuff herniation.

cated 2.5 cm above the tip of the cannula (fig. 2C). The right lung weighed 1,200 g and the left, 1,000 g. Both showed extensive broncho-pneumonia with multiple pulmonary abscesses.

Patient 3. AHG Antibody Bleeding Diathesis

A 50-year-old housewife was admitted to a community hospital with gastrointestinal symptoms consistent with a duodenal ulcer. While in the hospital, she fell out of bed, bruising the right shoulder and the right side of her face. Hematoma formation in the extremities and gastrointestinal bleeding had necessitated hospitalization on three occasions in the previous ten months. On the first admission, bleeding was found to be due to a circulating anti-AHG antibody which pro-
duced an AHF less than 10 per cent of normal. Since that admission the patient had been receiving large doses of prednisone (30-60 mg/day) and had received a course of 6-mercaptopurine. Mild diabetes had developed, for which she was taking Diabenese (250 mg/b.i.d.). At each admission to the hospital, large transfusions of fresh blood and cryoprecipitate were necessary to control bleeding.

Because of the history of hemorrhage following minor trauma the patient was transferred to the PUH. On admission, she had a massive hematoma of the side of the face, the floor of the mouth, and the base of the tongue. The airway, at this time, seemed adequate, and with oxygen via face mask the skin was pink. In spite of four units of fresh whole blood and 20 units of cryoprecipitate in the first 24 hours, the hematoma enlarged. On day 1 severe respiratory distress developed. Owing to the large size of the hematoma, an endotracheal tube could not be inserted. During emergency tracheostomy the patient developed hypotension, which rapidly responded to external cardiac compression and infusion of blood. The tracheostomy cannula was a #6 silver Jackson cannula with a Moirich double-swivel adapter and an Anesthesia Associates #6 slip-on cuff.

Over the next ten days, respirations were assisted with a Bennett ventilator. Humidification was provided with a Puritan heated nebulizer. Further large transfusions were necessary, but gradually the hematoma subsided and the patient's general condition improved. By day 12 the tracheostomy cannula was removed and she was transferred from the intensive care unit.

She progressed satisfactorily until day 25. Excoriations of her buttocks began to bleed intermittently and profusely, temporarily subsiding following transfusions. Each time she was turned face downward to relieve pressure on her buttocks, she developed respiratory embarrassment. In retrospect, this was thought to be due to tracheal collapse following extensive erosion of the tracheal rings and non-healing of the tracheotomy site. On day 31, shortly after being placed prone, the patient became hypotensive and, in spite of further transfusions, became comatose and died.

Autopsy. No healing of the tracheal stoma site which involved tracheal rings 3 and 4 had occurred. Starting 2 cm below the inferior border of the tracheotomy, the posterior wall of the trachea was completely eroded over an area of 6 cm². The esophagus lay in this crater, isolated but intact (fig. 3). Again, postmortem simulation placed the herniated portion of the cuff over the erosion. The left lung weighed 430 g and the right, 530 g. There was bronchopneumonia in both lungs.

DISCUSSION

The cases of tracheoesophageal fistula described here are listed in table 1, with 12 others found in the literature.1-10,12

The interval between tracheotomy and the appearance of the tracheoesophageal fistula can be seen to vary from 8 to 94 days. We chose not to include tracheoesophageal fistulas presenting within 48 hours of the tracheotomy because there is good reason to believe that these result from intraoperative trauma.1,12,14 In all cases where details are recorded, tracheoesophageal fistula occurred in patients in whom cuffed tracheostomy tubes were used along with varying periods of positive-pressure ventilation.

The precipitating cause of the lesions has been ascribed to the cuff, the tip of the tracheotomy tube, or both, although the evidence to distinguish between these factors has been minimal so far. In our cases there was strong evidence to incriminate the tracheostomy tube cuff and little to suggest that the tip of the cannula played a major role (figs. 1 and 2). In all three cases, the areas of tracheal erosion corresponded closely to the areas contacted by the bulge of the unevenly-inflating tracheostomy tube cuff.

FIG. 3. Tracheal-wall necrosis with intact esophagus in base of crater.
<table>
<thead>
<tr>
<th>Series</th>
<th>Patient</th>
<th>Diagnosis</th>
<th>Age (Years)</th>
<th>Sex</th>
<th>Time from Tracheotomy to Tracheoesophageal Fistula (days)</th>
<th>Duration IPPV via Tracheotomy to Tracheoesophageal Fistula (days)</th>
<th>Etiology of Tracheoesophageal Fistula</th>
<th>Repair</th>
<th>Outcome</th>
<th>Time from Tracheoesophageal Fistula to Death (days)</th>
<th>Cause of Death</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flege, 1967*</td>
<td>1</td>
<td>Flail chest</td>
<td>49</td>
<td>M</td>
<td>24</td>
<td>24</td>
<td>Pressure necrosis from cuff</td>
<td>No</td>
<td>Died</td>
<td>2</td>
<td>Progressive respiratory failure</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>Traumatic rupture of thoracic aorta</td>
<td>37</td>
<td>M</td>
<td>8</td>
<td>8</td>
<td>Pressure necrosis from cuff</td>
<td>Yes</td>
<td>Died</td>
<td>6</td>
<td>Progressive <em>Pseudomonas</em> infection</td>
</tr>
<tr>
<td>Bargo and Clawson, 1965*</td>
<td>6</td>
<td>Syringomyelia</td>
<td>21</td>
<td>F</td>
<td>22</td>
<td>22</td>
<td>Trophic lesion</td>
<td>No</td>
<td>Died</td>
<td>0</td>
<td>Asphyxia following aspiration</td>
</tr>
<tr>
<td>LaBrignand et Roy, 1969*</td>
<td>9</td>
<td>Tetanus</td>
<td>64</td>
<td>M</td>
<td>23</td>
<td>23</td>
<td>Pressure of cuff or cannula</td>
<td>Yes</td>
<td>Survived</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>Cullin-Barre syndrome</td>
<td>59</td>
<td>M</td>
<td>30</td>
<td>0</td>
<td>Pressure of cuff or cannula</td>
<td>Yes</td>
<td>Died</td>
<td>28</td>
<td>Asphyxia following aspiration</td>
</tr>
<tr>
<td></td>
<td>11</td>
<td>Myasthenia Gravis</td>
<td>27</td>
<td>F</td>
<td>60</td>
<td>12-60</td>
<td>Pressure of cuff or cannula</td>
<td>Yes</td>
<td>Survived</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td>12</td>
<td>Flail chest</td>
<td>32</td>
<td>M</td>
<td>26</td>
<td>?</td>
<td>Pressure of cuff or cannula</td>
<td>No</td>
<td>Died</td>
<td>75</td>
<td>Asphyxia following aspiration</td>
</tr>
<tr>
<td>Hedden and Safar</td>
<td>13</td>
<td>Cullin-Barre syndrome</td>
<td>43</td>
<td>F</td>
<td>10</td>
<td>16</td>
<td>Pressure of cuff</td>
<td>No</td>
<td>Died</td>
<td>10</td>
<td>Progressive respiratory failure</td>
</tr>
<tr>
<td></td>
<td>14</td>
<td>Poliomyelitis</td>
<td>31</td>
<td>M</td>
<td>94</td>
<td>94</td>
<td>Pressure of cuff</td>
<td>No</td>
<td>Died</td>
<td>3</td>
<td>Progressive respiratory failure</td>
</tr>
</tbody>
</table>
In a previous series, 24 poliomyelitis patients who required prolonged artificial ventilation for as long as four months (one patient for seven years) developed no evidence of serious tracheal injury. At that time, a long, large-residual-volume, low-pressure, large contact area, evenly-inflating, single-walled, Sanders-Foregger cuff and a "minimal leak technique" were used. Because of difficulties in sealing the cuff to the cannula, and because of occasional extensions of the cuff into the pretracheal space, a short, double-walled Anesthesia Associates cuff was substituted. The extent and incidence of superficial tracheal necroses seen at autopsies seem to have increased since this change of cuffs. This impression, and the three serious complications described in this paper, stimulated studies of tracheostomy tube cuffs in vivo and in vitro. The Anesthesia Associates short cuff used in the patients reported here was found to have a small residual volume, high intracuff pressure and small contact area; to inflate unevenly and produce a high lateral tracheal-wall pressure (147 mm Hg). When the cuff was inflated 1 ml beyond the "no leak" volume, tracheal-wall pressure increased by 130 mm Hg. In contrast, the previously-used Sanders cuff inflated evenly and produced low tracheal-wall pressure (5–21 mm Hg). Recently, we have replaced the Anesthesia Associates cuff by an experimental Foregger fluted cuff which inflates evenly. Nevertheless, one case of tracheal necrosis has been found in the approximately 200 patients in whom this fluted cuff has been used. Manufacturers were asked to make tubes with condom-like large-volume built-in cuffs.

The unevenness of inflation of the short cuff reported here became even more exaggerated when larger volumes of air were inserted, which led to herniation. Such herniation may be directed posteriorly by the shape of the tracheostomy cannula, the pliability of the posterior wall of the trachea, and the weakness of the cuff at the junction with the inflation tube, if placed posteriorly on the cannula. These directional influences probably explain why the tracheal damage occurred predominantly on the posterior and posteralateral walls of the trachea.

The preponderance of tracheal trauma from cuffs in patients on ventilators is explained by the fact that even under ideal circumstances the pressure exerted by the cuff against the trachea must equal the inflation pressure of the ventilator to prevent air leak, and high lateral pressures are necessitated by high inflation pressures. Indeed, high inflation pressures were necessitated by progressively decreasing lung compliance in Patient 1 following day 20 and in Patient 2 following day 70.

A technique providing for leak obviates the use of a cuff and may be a way around these difficulties. However, other major disadvantages are inherent in this: 1) over- or under-inflation of the lungs can occur; 2) pressure-cycled respirators may not cycle; 3) respiration may not trigger assisters; 4) there is the constant hazard of aspiration. Minimal leak during peak positive-pressure inflation by inflating the cuff only partially is a satisfactory compromise when there is no aspiration risk.

The three patients described were all nursed in an intensive care unit where an aseptic,atraumatic, tracheostomy care routine is enforced. This routine includes periodic brief cuff deflation accompanied by IPPV to exsufflate secretions and reinflation of the cuff only to the point of abolishing audible leak. We, therefore, may speculate on additional factors in the development of tracheal destruction:

Level of tracheostomy. The cricoid cartilage must be clearly identified and the second and third tracheal rings selected for a window or an inverted V-shaped flap. Several tracheostomies thought to be ideally situated were found to be much lower at autopsy (e.g., Patient 1). Low tracheostomy causes the cuff to have an abrasive effect on the trachea as the tube follows respiratory movements, may cause erosion of large vessels, and makes reintubation beyond the fistula difficult.

Steroid therapy. There may be a relationship between the steroid therapy administered to all three patients and the course of the development of destructive lesions.

Nasogastric tube. Patient 1, who had a large nasogastric tube in place throughout hospitalization, developed a large fistula. Patient 2, in whom the large tube was replaced by a
smaller feeding tube and later by a feeding gastrostomy, had only a small fistula. In Patient 3, who had no nasogastric tube, the esophagus remained intact.

Infection. In spite of aseptic tracheostomy care, a variety of organisms grew from the lungs of all three patients. The degree to which infection added to the destruction of the tracheal wall is difficult to determine. However, the combination of IPPV, lung infection, decreased compliance, and increased airway pressure seems hazardous.

Assessment of the degree of tracheal damage is difficult with tracheoscopic visualization. Measurements of cuff diameter by radiographs indicate tracheal dilatation, but provide little more information than that obtained by simply recording the quantity of air needed to expand the cuff to prevent air leakage.

The diagnosis of tracheoesophageal fistula was not difficult in the cases described. In Patient 1 it was proved by instilling methylene blue into the esophagus and observing its appearance in the trachea. In Patient 2 it was recognized by air leak into the gastrointestinal tract coincident with inflation. It was not possible to show the fistulas with contrast media and fluoroscopy or to visualize them by tracheoscopy. They are more easily seen at esophagoscopy.11

Once the fistula has developed, air leakage into the gastrointestinal tract may be prevented by using a larger tracheostomy tube with the cuff inflated below the fistula. This was impossible in Patient 1 because of the low level of the fistula. Operative intervention has been tried in a number of cases, and was successful in two patients cited in table 1, who had improved with spontaneous respirations.

SUMMARY AND CONCLUSIONS

Two patients developed tracheoesophageal fistulas, and one, tracheal-wall necrosis without fistula, during prolonged IPPV via cuffed tracheostomy tubes. The lesions were recognized 16, 95 and 12 days after tracheostomy. All three patients died and their laryngotraheas were examined at autopsy. The locations of the lesions corresponded with the herniated portions of the tracheostomy tube cuffs. These were of the narrow, small-residual-volume, high-intracuff-pressure, small-contact-area type which produces high lateral tracheal-wall pressures and inflates unevenly.16 Low tracheostomy, long-term steroid therapy, presence of a nasogastric tube and respiratory infection may have been potentiating factors. To prevent such tracheal destruction, the following are suggested: 1) use of long, large-residual-volume, low-pressure, large-contact-area, evenly-inflating cuffs which provide isolation of the respiratory from the gastrointestinal system in all phases of respiration at the lowest possible lateral tracheal-wall pressures; 2) meticulous cuff technique, with air introduced into the cuff never exceeding the volume necessary to produce a seal and recording of cuff inflation volumes.

REFERENCES

14. Meade, J. W.: Tracheotomy—its complications...
CLINICAL WORKSHOP

289

Volume 31
Number 3


A Simple Technique for Prolonged Arterial Cannulation

ROBERTO LLAMAS, M.D.,* SIRK K. GUPTA, M.D.,† GEORGE L. BAUM, M.D.‡

The care of critically-ill patients has made arterial blood-gas analysis a routine procedure. Frequently, the physician must resort to repeated arterial punctures 1 or to the difficult technique of arterial cannulation 2,3 when multiple sampling is necessary. This report describes a simple technique for prolonged percutaneous arterial cannulation. The method allows repeated blood sampling as well as pressure monitoring.

The cannula § used (fig. 1A) consists of an inner 19-gauge 3¾-inch needle and an outer 17-gauge 2¼-inch catheter. Flow through the catheter is controlled with the built-in three-way stopcock. After preparation of the skin, the wrist is hyperextended over a towel. One per cent xylocaine is infiltrated into the skin and around the radial artery from the crease of the wrist to about 1–1.5 cm. above. A scalpel nick is made in the skin just above the wrist. After the deadspace of the needle is filled with heparin, the cannula is introduced slowly through the nick at a 30 degree angle in the direction of the arterial pulsation (fig. 1B). As soon as blood issues through the stopcock, the cannula is advanced slightly to ensure its placement within the lumen. The needle is withdrawn, at which point a pulsatile stream of blood should issue through the stopcock. The catheter is then slowly advanced up to the hilt. After the deadspace of the cannula is filled with heparinized saline solution, the stopcock is closed (fig. 1C).

The present authors have used this cannula in over 130 patients in a Medical Intensive Care Unit. There have been no instances of loss of pulse or of ischemic changes distal to the cannula. The cannula has been maintained in situ for approximately three days per patient and for periods as long as ten days without untoward effects.

REFERENCES


* Assistant Professor of Medicine, University of Miami School of Medicine; Chief, Medical Intensive Care Unit, Veterans Administration Hospital.
† Fellow in Pulmonary Disease, Veterans Administration Hospital.
‡ Associate Professor of Medicine, University of Miami School of Medicine; Chief, Pulmonary Disease Section, Veterans Administration Hospital.