Intensive Care Med (1992) 18:160–169

Intensive Care Medicine

Review article

Bronchoscopy in the intensive care unit

Ph. Jolliet and J.C. Chevrolet

Soins Intensifs de Médecine, Hôpital Cantonal Universitaire, Geneva, Switzerland

Received: 30 August 1991; accepted: 27 February 1992

Abstract. The development of the flexible, fiberoptic bronchoscope has made bronchoscopic examinations possible in ICU patients undergoing mechanical ventilation. Over the years, the number of such procedures has greatly increased, with both diagnostic and therapeutic objectives, such as performing difficult intubation, management of atelectasis and hemoptysis, diagnosis of nosocomial pneumonia in ventilated patients, and early detection of airway lesions in selected situations, such as high-frequency ventilation. The complication rate can be kept low if the endoscopist has a precise knowledge of the many pathophysiological and technical facets particular to bronchoscopy under these difficult conditions. This article reviews some of these aspects, in the light of our personal experience.

Key words: Bronchoscopy – Mechanical ventilation – Hemoptysis – Atelectasis – Bronchoalveolar lavage – High-frequency jet ventilation

The diagnostic and therapeutic usefulness of bronchoscopy is well established [1–3]. Performed for the first time by Killian in 1897 through a rigid tube, it has since then evolved into a practical and much-used technique, thanks to the development of the flexible fiberoptic bronchoscope by Ikeda in the 60 s [1, 4, 5]. Over the years, bronchoscopy has become common practice in intensive care patients [6–9]. However, the bronchoscopist is faced with certain technical problems when these patients are mechanically ventilated. The purpose of this article is to review these problems, and to study the indications for bronchoscopy that are specific to adult critical care patients.

Specific aspects of bronchoscopy in the intensive care setting

1) Type of bronchoscope

Indications for using the rigid bronchoscope have become rare [10]. Indeed, this technique greatly reduces the endoscopist's scope of vision and manoeuverability, while increasing patient discomfort. Furthermore, it cannot be used during mechanical ventilation. Its use is thus restricted to situations in which it has proved more efficient than the flexible bronchoscope, such as masive hemoptysis, foreign body removal, and Nd-YAG laser therapy [3, 10]. In all other indications, the flexible bronchoscope is preferred [4, 5, 10]. Rigid bronchoscopy was also considered initially to be the technique of choice in small children, but flexible bronchoscopy has now gained widespread acceptance in this patient population [11, 12]. To allow bronchoscopy during mechanical ventilation, special adapting units have been designed [7–9]. They usually consist of a unit connecting the endotracheal tube (ETT) with the respirator tubing. The unit is fitted with a side-port, normally sealed with a cap-lock. The latter is opened and the bronchoscope is inserted through the side port.

2) Consequences on respiratory mechanics

In spontaneously breathing patients undergoing fiberoscopy without intubation, tracheal pressure measured by a transducer connected to the air-filled suction-port of the bronchoscope varies between −5 (inspiration) and +3.5 (expiration) cmH_2O [13] (Fig. 1). The same figures are found in the absence of a bronchoscope. Thus, without intubation, bronchoscopy has little or no effect on intratracheal pressure. When the bronchoscope is passed through an ETT, whether it be oro-tracheal or a tracheostomy tube, the intratracheal pressures, recorded with the same technique, now vary between −10 and +9 cmH_2O [13] (Fig. 1). When the same measurements are performed during bronchoscopy while the patient is mechanically ventilated [13], the following changes are observed (Fig. 2):

(i) the ventilator pressure gauge indicates high maximal inspiratory values, reaching a maximum of 80 cmH_2O in the above-mentioned study [13]. This high peak inflation pressure is due to the presence of the

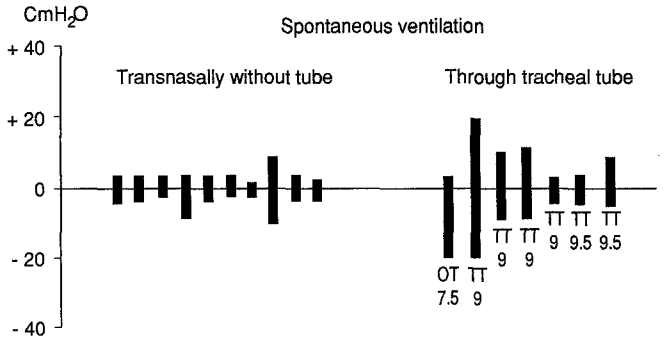

Fig. 1. Intratracheal pressure recordings with (*right*) and without (*left*) an endotracheal tube, during flexible fiberoptic bronchoscopy (external diameter of the bronchoscope 5.7 mm) in 17 patients. Reproduced by permission from [13]. *TT*, tracheostomy tube; *OT*, orotracheal tube; *values below each bar* = tube diameters in mm ID

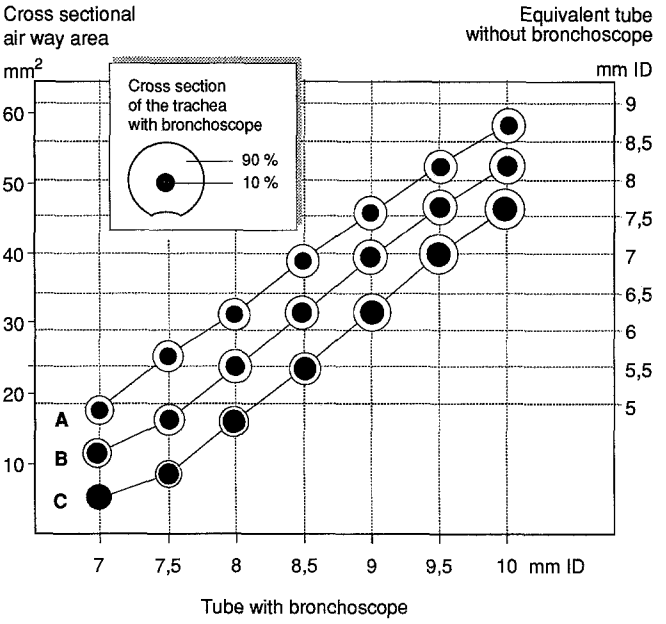

Fig. 3. Diagram of cross-section of different caliber endotracheal tubes (*outer circles*) containing 3 different size bronchoscopes (*filled circles*): *A*) 5 mm external diameter (ED); *B*) 5.7 mm ED; *C*) 6.4 mm. Insert shows cross-section of the trachea and bronchoscope in a non-intubated patient. Reproduced by permission from [13]

fiberoscope in the ETT, and represents ventilator back-pressure rather than true intratracheal pressure;

(ii) true intratracheal pressure, measured through the suction-port of the bronchoscope, is much lower. Nonetheless, it is much higher than during spontaneous breathing, averaging 34 cmH_2O at end-inspiration [13];

(iii) the intratracheal pressure readings remain positive at end-expiration, between 10 and 15 cmH_2O, representing a positive end-expiratory pressure (PEEP) effect. This stems from incomplete lung emptying during the expiratory phase, the presence of the bronchoscope in the ETT adding considerable expiratory resistance. Proof of this was obtained in animal experiments: when suction was applied with the bronchoscope in place, PEEP disappeared [13].

Animal experimentation and clinical studies have shown that these high airway pressures are closely linked to the relative internal diameters of the ETT and the bronchoscope [13–16]. Indeed, in the absence of an ETT, a bronchoscope occupies only 10% of the total cross section of an adult trachea [13]. A 5.7 mm internal diameter bronchoscope occupies 40% of the total cross-section of a 9 mm internal diameter ETT, 51% of that of an 8 mm ETT, and 66% of a 7 mm ETT [13–16] (Fig. 3).

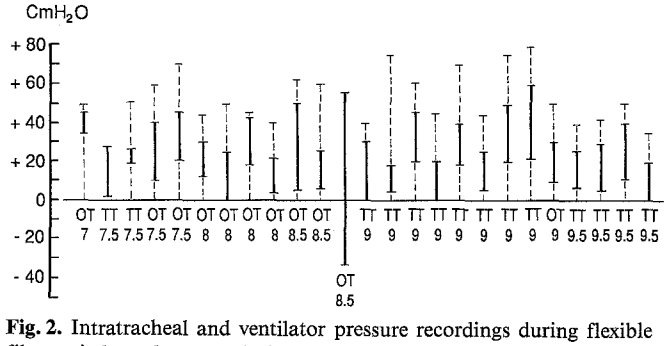

Fig. 2. Intratracheal and ventilator pressure recordings during flexible fiberoptic bronchoscopy during volume-controlled mechanical ventilation. Center solid line represents spontaneous respiratory efforts by an incompletely sedated patient. Reproduced by permission from [13]. *TT*, tracheostomy tube; *OT*, orotracheal tube; *values below each bar* = tube diameters in mm ID; *dotted bars*, ventilator pressure; *solid bars*, intratracheal pressure

This considerably increases resistance to flux, up to 11 times in an 8 mm ETT [14]. High inflation pressures result from this, as does a PEEP effect. A PEEP of up to 35 cmH_2O has been recorded with a 7 mm ETT [13]. Usually, however, PEEP remains below 20 cmH_2O in an 8 mm ETT [13]. Thus, an 8 mm internal diameter is considered the minimum allowing bronchoscopy with a reduced risk of barotrauma [13, 14].

Other effects of fiberoptic bronchoscopy on lung volumes and respiratory mechanics have been documented: in intubated patients, insertion of the bronchoscope induces a 30% increase in the functional residual capacity (FRC), as well as a 40% decrease in the one-second forced expiratory volume (FEV_1) [14]. These modifications return to baseline values after removal of the bronchoscope. However, there persists a decrease in mid-expiratory flow rate (FEF 25%–75%), considered to stem from small airway obstruction [14].

3) Gas exchange and hemodynamic alterations

The presence of a bronchoscope in the airways induces a slight increase, averaging 1.1 kPa [13] in the $PaCO_2$ and a moderate decrease, averaging between 1.1 [14] and 2,5 kPa [13, 17, 18] in the PaO_2 [13, 14, 18]. This probably derives from the smaller tidal volume delivered while the bronchoscope is in place [9]. However, when suctioning is applied, $PaCO_2$ rises by about 30%, while PaO_2 decreases by about 40% [13] (Fig. 4). The hypothesized mechanism of these alterations is a reduction in expired tidal volume by suction, thus decreasing the volume participating in gas exchange. Furthermore, as we have seen in the preceding section, suction reduces end-expiratory volume and PEEP. This in turn facilitates alveolar clo-

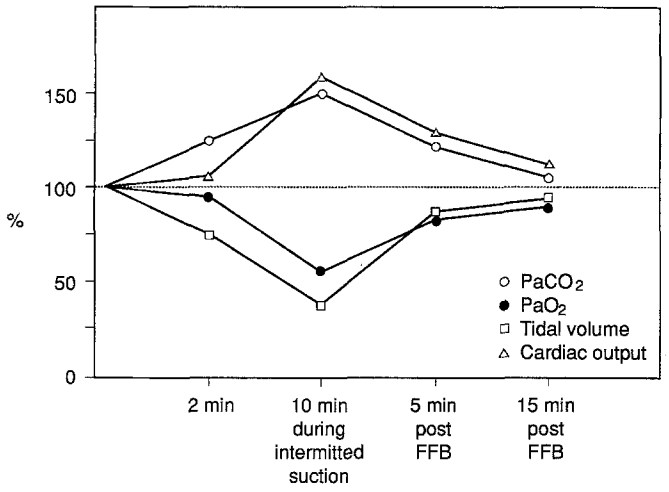

Fig. 4. Changes in 4 variables during flexible fiberoptic bronchoscopy (FFB) in 6 patients during mechanical ventilation. Pre-bronchoscopy values set at 100%. Reproduced by permission from [13]

sure and venous admixture [13, 14]. These modifications slowly subside following completion of bronchoscopy. The delay before normalization of gas exchange varies from about 15 min for normal lungs to several hours in severe parenchymal disease [13]. These findings have led to the continuous monitoring of arterial oxygenation during and up to 1–2 h after bronchoscopy [17, 19]. It is beyond the scope of this article to compare the various techniques available. Suffice it to say that the method of choice at the present time seems to be pulse oximetry, which allows on-line monitoring of the O_2 saturation of hemoglobin by spectrophotometry [20]. It offers more flexibility in its use than transcutaneous PO_2 ($PtcO_2$) [17, 19], which requires heating the skin, and offers less reliability in low cardiac output states [20]. In unstable or hypercapnic patients, measurement of the end-tidal expired PCO_2 ($PetCO_2$) at the endotracheal tube opening allows a breath-by breath analysis of changes in the ventilatory status [20].

Few studies have specifically examined the hemodynamic consequences of bronchoscopy in mechanically ventilated patients. In the study by Lindholm [13], cardiac output was measured and showed an increase reaching 50% during the procedure, returning to normal in 15 min after its termination. Heart rate and blood pressure were not reported. A recent study [21] of the cardiopulmonary risk of fiberoptic bronchoscopy in 107 ventilated patients reported no deaths or cardiac arrest during or within two hours of the procedure. There was a 5% incidence of major arrhythmias and a 13% incidence of hypoxemia with a $PaO_2 \leq 60$ mmHg on FiO_2 of 0.8. The latter occurred mostly in patients with ARDS or those that were insufficiently sedated.

These findings tend to confirm the overall safety of fiberoptic bronchoscopy in mechanically ventilated patients. However, in a subset of severely ill patiens with limited gas-exchange and hemodynamic reserve close monitoring of key parameters is mandatory.

Thus, guidelines can be drawn in order to minimize the risk of fiberoscopy during mechanical ventilation, which are summarized in Table 1.

Table 1. Bronchoscopy in mechanically ventilated patients

1) Use an endotracheal tube with an internal diameter of at least 8 mm, if a standard fiberoscope size (5.7 mm diameter) is utilized.
2) Discontinue PEEP or reduce by 50%, or monitor bronchoscope tip pressure.
3) Increase FiO_2 to 1.0 starting 15 min before procedure and tidal volume by about 30% during bronchoscopy.
4) Check arterial blood gases before and after procedure. Postpone examination if $SaO_2 < 90\%$ on FiO_2 1.0.
5) Monitor SaO_2 by continuous pulse oximetry.
6) Monitor tidal volume.
7) Monitor $PetCO_2$ in selected unstable and/or hypercapnic patients.
8) Monitor pulse and blood pressure.

4) High-risk patients

a) Coronary-artery disease patients. No study has specifically addressed the issue of evaluating the risk of fiberoptic bronchoscopy in coronary patients. However, from different studies dating back to the 70 s, it can be deduced that, in stable coronary patients, there is no increase in the incidence of serious arrhythmias or in mortality resulting from the procedure [22–24]. In a series of 48,000 procedures, there were 10 reported deaths [24]. Ischemic heart disease was documented in 6 of these patients. Cardiac arrest or intractable ventricular arrhythmia during or immediately after the procedure was the cause of death. An 11% incidence of major arrhythmias during bronchoscopy has been recorded in ambulatory patients [25]. All were self-limited and had no hemodynamic consequences. The presence of known coronary disease was not associated with a higher risk of arrhythmia. However, hypoxemia at the end of bronchoscopy ($PaO_2 < 60$ mmHg) did entail an added risk of arrhythmia. The issue of patients in the immediate postinfarction is not resolved. Nonetheless, it seems reasonable to differ if possible the procedure in patients who have just had a myocardial infarction, as well as to monitor with extreme care SaO_2 during fiberoscopy in any patient with known coronary disease. Sedation should also be adequate, to prevent tachycardia and oxygen desaturation.

b) Asthmastic patients. Fiberoptic bronchoscopy in asthmatic patients is usually indicated for removing obstructive mucous plugs and secretions from the airways [26]. However, severe laryngospasm and bronchospasm have been described as a result of this procedure [27]. Another study showed no decline in FEV_1 after fiberoscopy and bronchoalveolar lavage (BAL) in mild asthmatics. A recent study comparing asthmatic and non-asthmatic patients undergoing bronchalveolar lavage and endobronchial biopsies documented arterial oxygen desaturation and a significantly greater decrease in post-procedure FEV_1 in the asthmatic group [28]. The latter correlated with the measured degree of bronchial reactivity 5 days prior to bronchoscopy [28]. There were no serious complications asides from mild, reversible, bronchospasm. It is to be noted that the patients in these studies were not under conditions of mechanical ventilation. Thus, it

seems that indications for bronchoscopy, BAL, and endobronchial biopsies should be weighed carefully. If possible, corticosteroids should be administered for a few days prior to the procedure, especially in unstable patients.

c) Other potential high-risk patients. In the study by Suratt [24], all 10 patients who died presented with either ischemic heart disease, as we have already seen, chronic lung disease, or cancer. It is however obvious that belonging to either of the two latter diagnostic categories is insufficient evidence per se of being at higher risk to undergo bronchoscopy. Rather, the degree of severity of these illnesses conditions the risk factor.

Finally, respiratory failure requiring intubation and mechanical ventilation after bronchoscopy with bronchoalveolar lavage has been observed in immunocompromised patients [29, 30].

Indications for bronchoscopy in the intensive care unit (ICU)

1) Intubation

Bronchoscopy allows direct visualization of the laryngeal opening and the vocal cords. It is therefore possible, once the fiberoscope is in the trachea, to slide an ETT over it, thus enabling intubation when the laryngeal structures cannot be visualized with the laryngoscope [10, 31, 32]. When the bronchoscope is of a small diameter, however, it is possible for it to be bended by the ETT just prior to the laryngeal opening, with resultant esophageal intubation [33]. This can be avoided by continuously viewing the trachea while the ETT is inserted, and by visualizing the tip of the ETT through the bronchoscope before advancing both as a unit until reaching proper ETT position above the carina [32, 34]. Some manufacturers have also developed stiffer bronchoscopes designed to avoid bending by the ETT and esophageal intubation [32]. The claimed merits of such a fiberoscope have not, to our knowledge been confirmed by rigorous studies. Bronchoscopy, nonetheless, is seldom used for intubation in the ICU: a recent series showed that intubation represented 0.5% of all indications [31].

We have studied this problem in our institution (general hospital, 1,600 beds): during the year 1987, about 17,000 intubations were performed in the different operating rooms, ICU's, emergency rooms and wards. Only 12 of these were carried out with the help of a bronchoscope, a mere 0.07% of the total. A detailed analysis of these cases shows 8 elective and 4 emergency situations (Table 2). As can be seen from the short time needed, intubation with the bronchoscope was fairly easy in the elective cases. The only failure to intubate resulted from the inadvertent administration of a muscle-relaxant, which stopped the inspiratory movements that were guiding the endoscopist. Quite different was the situation in the emergency cases: the failure rate was 25%, vs the 14% in the elective cases, and the time necessary to succeed in the intubation was much longer. This stems from the fact that emergency intubations with the fiberoscope were performed after many failed attempts with the laryngoscope, the latter having caused local bleeding and edema. Some conclusions can be drawn from this small series:

(i) The bronchoscope is a very helpful tool for intubation, but is needed in only a small number of cases;

(ii) if a difficult intubation is anticipated, it is preferable to use the bronchoscope from the onset, rather than use it after many unsuccessful attempts with a laryngoscope;

(iii) bronchoscopy must be performed by an experienced endoscopist.

The preferred fiberoscope diameter in an adult is 5.7 mm. A pediatric fiberoscope should not be used, as it is more flexible and thus more prone to bending into the esophagus [32, 33]. Intubation can be carried out through the oral or nasal routes. The oral route is preferable, as the presence of a nasotracheal tube has been shown to increase the incidence of purulent sinusitis [35], and, also as the oro-tracheal route allows insertion of a larger internal-diameter tube. This in turns facilitates tracheal secretion aspirations, bronchoscopy if needed, and decreases the patient's work of breathing [36].

Table 2. Intubations with the help of a fiberoscope, Hôpital Cantonal Universitaire de Genève, over a 12-month period

Patient	Age	Sex	Underlying condition	Time to intubation (min)	Comments
			Elective	Intubations	
1	62	F	RA	5	–
2	60	F	RA	5	–
3	22	F	Juvenile RA	7	–
4	30	M	Short neck	2	–
5	38	M	Short neck	5	–
6	22	M	Dysmorphism	Failure	MR
7	42	F	Previous failure	5	–
8	46	M	Previous failure	0.5	–
			Emergency	Intubations	
9	41	M	Failed intubation	Failure	Epistaxis
10	62	F	Failed intubation	20	UAE, LB
11	54	M	Failed intubation	18	UAE, LB
12	34	F	Failed intubation	20	UAE, LB

Abbreviations: MR, Muscle relaxant administered just prior to procedure; RA, rheumatoid arthritis; UAE, upper airways edema; LB, local bleeding (oropharynx and/or nasal)

2) Atelectasis

Atelectasis is a frequent problem in the ICU. It most often results from retained bronchial secretions, due to increased production and/or decreased cough efficiency (post-operative period, mechanical ventilation, neuromuscular disease) [37–40]. Left untreated, it may impair gas exchange, predispose to infectious complications, or, more rarely evolve into fibrosis [38]. As soon as the fiberoptic bronchoscope was produced, its use to aspirate secretions more efficiently to prevent or treat atelectasis expanded rapidly [39, 41, 42]. The superiority of this approach has not, however, been established through care-

fully controlled trials. Marini et al. studied intubated and non-intubated postoperative patients with acute lobar atelectasis: no difference was observed in the speed of radiologic improvement between the group treated by respiratory therapy alone and the group undergoing bronchoscopy [38]. An interesting finding was that in each group, the presence of an air bronchogram predicted delayed resolution of the atelectasis. This is probably due to the fact that an air bronchogram indicates patency of the large airways, the only ones accessible to either method [38]. Patients with neuromuscular disease seem to benefit from a bronchoscopic approach. It has been shown that these patients frequently develop proximal atelectasis due to mucus-plug forming as a result of ineffective cough mechanisms and other less well-known causes [37, 43]. We have studied 6 patients with the Guillain-Barré syndrome (GBS) requiring mechanical ventilation due to acute respiratory failure. During the period of mechanical ventilation (mean duration ±SD: 30±20 days), 2 patients did not develop atelectasis. The 4 others had repeated episodes of segmental, lobar or pulmonary atelectasis (total 25), 76% of which were located in the lower lobes. There was no air bronchogram on the chest X-ray. Bronchoscopy was performed immediately, successfully removing mucus plugs. A-a DO_2 decreased by a mean (±SD) of 15.9 (±12) kPa. We thus favor rapid bronchoscopy in GBS patients with atelectasis, as the cause is usually located in the central bronchi, and thus accessible to a treatment leading to improved gas exchange.

Finally, a new method of reexpanding atelectatic lung regions, using the flexible bronchoscope, has recently been described [44]: it consists of room air insufflation by means of an Ambu bag into the working channel of a bronchoscope wedged in a segment of the collapsed region. A pressure gauge is connected by a three-way adaptor, and peripheral airway pressure kept below 30 cmH_2O, or 10 cmH_2O above previous airway pressure. Rapid and complete radiological resolution, accompanied by a substantial decrease of the alveolo-arterial oxygen gradient over the following 24 h, was obtained in 12 of 14 ICU patients. There were no complications [44]. Further validation of this technique is required.

3) Hemoptysis

Bronchoscopy is a very efficient diagnostic tool in patients with hemoptysis [2, 3, 10, 45]. In about 10% of cases, hemoptysis is massive [46]. In such situations, the rigid bronchoscope is often preferable, as it allows better vision, provided that the source of bleeding is proximal, and more efficient aspiration [3, 10, 45]. However, the flexible bronchoscope has been used successfully in cases of more distal bleeding sources [47–50].

When massive hemoptysis occurs, bronchoscopy, in addition to serving diagnostic purposes, can also be therapeutic, by guiding either endobronchial tamponade [45, 47–49] or hemostasis by application of fibrin precursors [50]. Endobronchial tamponade, as classically described, consists of passing a Fogarty balloon catheter through the bronchoscope's aspiration channel, and inflating it to occlude the bleeding zone [51]. This either stops the bleeding or buys enough time to perform a thoracotomy [49]. Once the bleeding has stopped, the main problem is determining both the balloon's inflation pressure and the time during which it stays inflated, tyring to avoid tracheal mucosal damage [52, 53]. In one series, a period of 24 h is mentioned, but neither inflation pressure nor the presence or absence of mucosal damage are outlined [49]. We have been able to document these aspects in 2 of our patients:

(i) *Patient 1* was a 63-year-old male, alcoholic, cirrhotic, heavy smoker, intubated because of a hemoptysic episode of 600 ml in 12 h. The source of bleeding was in a right antero-superior sub-segment. Persistent hemoptysis despite a continuous infusion of vasopressin would have prompted surgical treatment. However, the patient's general condition was so poor that he was judged inoperable. A Swan-Ganz pulmonary artery balloon catheter was inserted, under bronchoscopic guidance, in the subsegment, and inflated over the bleeding source (Fig. 5). The balloon was left inflated with a pressure not exceeding 40 mmHg, verified by means of a manometer, for 48 hours. The maximal pressure was set approximately, in line with experimental data indicating an increased risk of mucosal injury when 30 mmHg are exceeded [52, 53]. There was no recurrence of hemoptysis after the catheter's withdrawal. However, the patient died 10 days later of severe hepato-renal syndrome. Autopsy showed only a small bronchial parietal hematoma corresponding to the source of the bleeding, but no zones of mucosal necrosis at or near the balloon's position were detected on serial histologic sections.

(ii) *Patient 2* was a 66-year-old male patient suffering from multiple myeloma, with chronic heart and renal failure. He was intubated for acute respiratory distress. The chest X-ray showed bilateral patchy infiltrates. Bron-

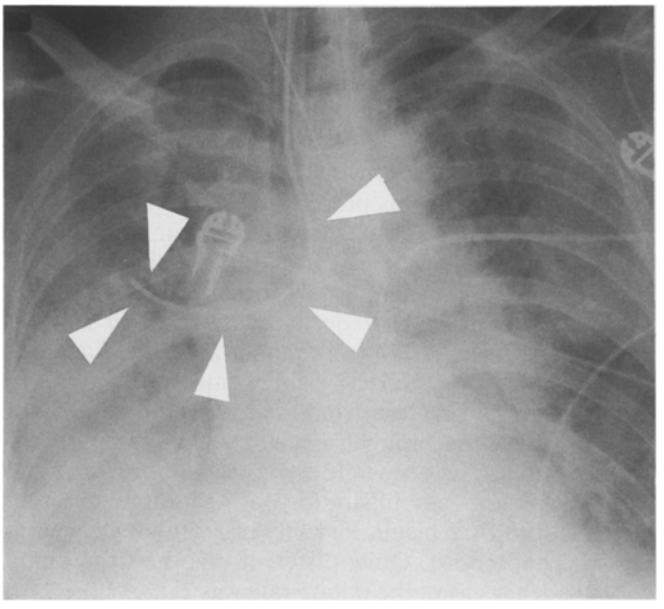

Fig. 5. *Patient 1.* Swan-Ganz plumonary artery balloon catheter, with balloon inflated, positioned in a sub-segment of the anterior segment or the right upper lobe

choscopy with BAL and transbronchial biopsy was performed. Soon thereafter, massive hemoptysis occurred. Using the same technique as in patient 1, a Swan-Ganz catheter was inserted under bronchoscopic guidance into the bleeding segment. The bleeding stopped, with minimal impairement of an already precarious gas exchange situation. The balloon was also left in place for 48 h. The situation momentarily improved, but the patient developed multiple organ system failure (MSOF) and died on the 15th hospital day. As in the preceding case, serial histologic examination did not show evidence of bronchial mucosal damage due to the balloon.

These cases document the fact that endobronchial balloon-catheter tamponade is feasible without causing added mucosal damage, for up to 48 h, with an inflation pressure not exceeding 40 mmHg. Furthermore, our choice of the Swan-Ganz rather than the Fogarty catheter merits comment. The Fogarty, inserted through the channel of the fiberoscope, cannot, for reasons of size, occlude less than a lobe, or even sometimes an entire lung. The Swan-Ganz, inserted beside the bronchoscope, is smaller in diameter and can be manoeuvered into subsegmental bronchi. This can be helpful to preserve gas exchange as much as possible, as patient 2 illustrates.

Though their description is beyond the scope of this article, other techniques are potentially useful to manage certain bleeding lesions such as iced-saline lavage [54], endobronchial electrosurgery and laser therapy through the bronchoscope [55], and the spraying of fibrin precursors on the site of hemorrhage [50].

It should nevertheless be remembered that surgery has been advocated as the treatment of choice in massive hemoptysis [46, 48], and should always be considered. However, an attractive alternative in experienced hands is bronchial artery embolization, and might be the preferred technique in patients at high surgical risk [56].

4) Diagnosis of nosocomial pneumonia

The incidence of nosocomial pneumonia during mechanical ventilation varies from one study to another, from 20% in a general population of ICU patients to 70% in the adult respiratory distress syndrome (ARDS) [57–59]. In the latter, it must be noted that mortality directly attributable to respiratory failure is only 16%, the principal cuase of death being sepsis evolving into multiple organ failure (MOF) [60]. The entry point leading to sepsis is most often the lung [60]. Excess mortality resulting from nosocomial pneumonia is difficult to evaluate, but is estimated at 15% [61]. Rapid diagnosis is thus important. Unfortunately, the usual clinical, laboratory and radiological workup has an insufficient predictive value [57, 61]. A bacteriological examination is thus necessary. The great difficulty in obtaining good quality samples in a mechanically ventilated patient by simple tracheal aspiration [62, 63], has lead to the development of improved techniques of bacteriologic sampling [59, 61, 64–76]. We will briefly discuss the most often used bronchoscopic techniques:

a) Protected specimen brush (PSB). This technique was initially described in 1979 [74]. Its value was subsequently confirmed by Chastre in 1984 [72], in a study showing a good correlation between cultures of PSB and those obtained from lung tissue samples, considered to be the gold standard. However, the method's excellent sensitivity of 100% was accompanied by a specificity of only 80% [72]. In a subgroup of patients already receiving antibiotic treatment, specificity was a very poor 42% [72]. A subsequent study by the same group [71] reported a sensitivity of 100% and a specificity of 90%. Nonetheless, a certain number of technical pitfalls must be kept in mind:

(i) the volume of secretions obtained is very small, around 0.001 ml [74]. This requires very precise sample-handling. It also explains why in most studies other than Chastre's [72] the sensitivity of PSB was lower than that of BAL, which retrieves a larger volume of fluid and explores a larger alveolar volume, estimated at 5–20 million alveoli [64];

(ii) PSB entails an added risk of bleeding and pneumothorax [77].

b) Broncho-alveolar lavage (BAL). BAL has been studied as an alternative to PSB, with improved specificity and less risk of complications [59, 66–68, 78]. It is unfortunately inevitable that some degree of contamination by proximal airway secretions occurs. To minimize its consequences, various criteria have been used [63, 66, 67, 69, 78, 79]. Specificity was shown to be very good when more than 10^5 colony forming units (CFU) were present in the culture [78]. It is poor, however, when there are less CFU's [78]. It is possible to improve specificity by taking into account the epithelial cell count [67] and/or a bacterial index from quantitative cultures [72]. Studies which have compared BAL and PSB do not reach a definite conclusion as to the superiority of one over the other [61, 63, 69]. An important factor to be considered is time: rapid results are needed, in order to decide whether antibiotic treatment should be initiated or modified. Chastre [79] showed that, in the microscopic examination of BAL fluid, the presence of micro-organisms in more than 7% of macrophages and neutrophils was diagnostic of pneumonia with a sensitivity of 86% and a specificity of 96%. In another study, the Gram stain of a cytocentrifuged BAL sample showed very good correlation with the results of BAL cultures, in predicting the nature of the organism involved [78]. In terms of complications, bleeding and pneumothorax are more frequent with PSB [77]. BAL causes moderate transient hypoxemia, prompting continuous monitoring of gas exchange and an increase the FiO_2 to 1.0 during the procedure [17, 19, 68].

A summary of the different techniques for the study of material retrieved by BAL and PSB, as well as their sensitivity and specificity, is shown in Table 3.

c) Transbronchial biopsy (TBB). Compared to BAL and PSB, TBB offers the added advantage of a histologic examination of the lung parenchyma [10, 80]. Its superiority in the diagnosis of nosocomial pneumonia has not been shown, nor has it proved better than BAL in diagnosing opportunistic lung infections in immunosuppressed patients [81, 82]. It carries a 7–14% risk of pneumothorax [83, 84], as will be discussed in the section on complications.

Table 3. Most widely used bronchoscopic techniques for the diagnosis of nosocomial pneumonia

Technique	Sample analysis/ diagnostic criteria	Reference	Sensitivity	Specificity
BAL	>7% Macrophages/PMN's containing bacteria	[79]	86%	96%
	Gram stain, cytocentrifuged sample ≥1 micro-organism/ 100× field	[78]	73%	100%
	Quantitative cultures (Bacterial index ≥ 5)	[69]	74%	92%
		[78]	88%	100%
		[66]	71%	86%
	Quantitative cultures (>10^5 *CFU*/ml + ≤% SEC)	[67]	100%	100%
PSB	Gram stain	[113]	58%	N.A.
	Quantitative cultures (>10^3 *CFU*/ml)	[72]	100%	77%
		[113]	70%	100%
		[71]	100%	90%

BAL, Bronchoalveolar lavage; *PSB*, protected specimen brush; *SEC*, squamous epithelial cells; *CFU*, colony forming unit; *N.A.*, information not available; *Bacterial index*, sum of the log number of the concentration of the different bacteria identified (example: *Klebsiella pneumoniae* 10^3/ml and *Pseudomonas aeruginosa* 10^4/ml. Bacterial index = 3 + 4 = 7)

d) Immunosuppressed patients. TBB or open-lung biopsy has been considered as the method of choice for diagnosing opportunistic lung infections in this patient population [10]. A study performed by Williams [81] showed, however, that bronchoscopy with BAL and PSB had a diagnostic sensitivity for infection of 90%. Furthermore, when BAL and PSB were negative, TBB was of no added benefit, while open lung biopsy was marginally contributive [81]. This confirmed results from a previous study by Stover [85]. In patients with the acquired immunodeficiency syndrome (AIDS), both BAL and TBB were initially performed, as they were shown to be complementary [86]. As techniques of microbial identification, in particular of *Pneumocystis carinii,* improved, TBB was abandoned, as BAL was shown to have sufficient yield by itself [82]. BAL is thus usually the preferred technique, except for the diagnosis of the lymphocytic interstitial pneumonia of AIDS, where TBB is needed, and coccicioidomycosis, where BAL and TBB combined have a higher yield [87].

e) Non-bronchoscopic lavage (NBL). There seems to be a promising future for non-bronchoscopic lavage [75, 76, 88–90]: a recent study from our institution confirms that a simple catheter, introduced blindly in the tracheo-bronchial tree can have diagnostic accuracy comparable to bronchoscopic BAL, irrespective of the segment in which it is located [89]. Another recent study [90] comparing the yield of PSB with that of aspiration from a plugged telescopic catheter showed that the latter was as accurate as PSB in diagnosing nosocomial pneumonia in ventilated patients. Furthermore, whether the catheter was guided into place by bronchoscopy or introduced blindly into the tracheo-bronchial tree, its yield was the same [90].

After reviewing the available data, defining the optimal diagnostic approach to the diagnosis of nosocomial pneumonia in ventilated patients remains a difficult matter. Deciding whether to use BAL, PSB or NBL is often a matter of personal experience. We favor bronchoscopic BAL, as it allows inspection of the tracheo-bronchial tree. A Gram stain of a cytocentrifuged sample and a count of bacterial-containing and squamous epithelial cells provides rapid diagnosis. Quantitative bacterial cultures with a cut-off value of 10^5 Cfus/ml allow diagnostic confirmation, bacteria identification, and antibiotic adjustment.

5) Evaluation of tracheo-bronchial lesions during high-frequency jet ventilation (HFJV)

The use of HFJV has been described in laryngeal surgery, during bronchoscopy, in the treatment of broncho-pleural fistulas, and to improve oxygenation in infant or adult respiratory distress syndrome [91–93]. Clinical and experimental studies have shown that HFJV can cause tracheal and main bronchi damage in the form of mucosal edema and congestion, leading to erosions evolving into hemorragic necrosis [94–100], in turn causing airway obstruction [96, 98, 99]. We have studied 7 patients undergoing HFJV for severe respiratory distress [101]. The 7 patients died. A detailed histologic examination showed dramatic tracheal wall lesions in all the patients. The severity of histological damage correlated with the number of jet pulses administered (Fig. 6). These data should prompt caution as to duration and frequency when administering HFJV. Humidification, either too little or too much seems to be an important contributor to the development of tracheal lesions, and should also be monitored carefully [102, 103]. Some authors recommend repeated bronchoscopic evaluations, possibly with tracheal biopsies, during HFJV, to prevent the transition to severe lesions [98, 99]. We try to abide by these recommendations. However, it is not always feasible to perform

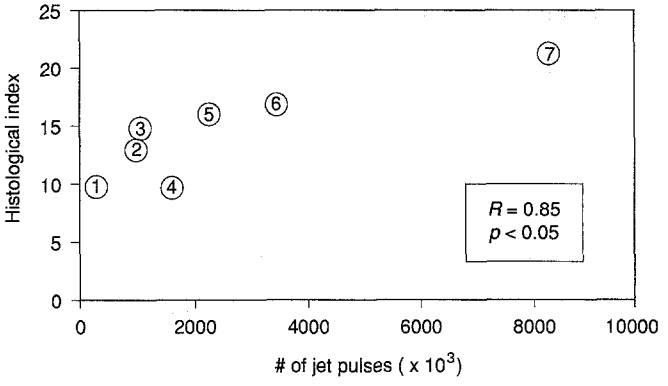

Fig. 6. Correlation between the number of administered jet pulses (jet frequency per min × 60 × number of hours of jet ventilation) and a histological severity index of tracheal wall damage, in 7 patients having received high frequency jet ventilation (HFJV). Reproduced by permission from [101]

bronchoscopy in the often disastrous gas exchange conditions of these patients. Furthermore, it remains to be proved that the early detection of lesions can lead to avoidance of the evolution to necrotizing tracheobronchitis.

6) Other indications

a) Trauma. In a series of 53 patients having sustained chest trauma, bronchoscopy performed within 3 days revealed lesions such as tracheal or bronchial transections, lacerations or contusions, hemorrhage or mucuous plugging in 53% of the patients [104]. Thus, this examination should be part of the diagnostic work-up of chest trauma patients [104, 105].

b) Airway obstruction. Bronchoscopy is a useful tool in respiratory failure stemming from bronchial obstruction due to foreign bodies [106, 107], or tumoral tissue growth. In the latter situation, endobronchial electrosurgery [108] or laser therapy [109] have been successfully used.

Complications of bronchoscopy in the ICU

No study has so far been designed to specifically evaluate the complication rate of bronchoscopic examination in ICU patients. In a recent series of 198 such procedures [31], 76% of which took place during mechanical ventilation, there was a 4% incidence of complications, consisting of either arrythmia, transient hypoxemia, or increased fever. No deaths occured. The same rate and nature of complications were documented in an older series comprising 446 procedures [7]. There was a 3% incidence of malignant arrythmia or cardiac arrest directly attributable to the procedure [7]. Transbronchial biopsies can be performed during mechanical ventilation, with a good diagnostic yield [83, 84], but indications should be restrictive in view of a 7% – 14% risk of pneumothorax reported in these series. In fact, mechanical ventilation has been considered by some as a contraindication to this procedure [110]. One intriguing event is the occurrence of self-limiting fever immediately following the procedure [111]. The causative mechanism is still poorly understood. Bacterial translocation into the bloodstream has been hypothesized, but disproved [112]. A possibility could be the translocation of endotoxin or release of inflammatory mediators. However, in 6% of the patients in Pereira's study [111], fever was accompanied by radiological signs of parenchymal infiltrate, suggesting that, in some patients, the procedure might be complicated by intrapulmonary infection.

Finally, it should be noted that premedication and local anesthesia may be sources of complications: in a survey of over 24,000 patients [22], excessive premedication resulted in 4 episodes of serious respiratory depression, in one instance requiring intubation. Local anesthesia caused 7 episodes of major side effects (respiratory depression, seizures) and one death (cardiovascular collapse). These figures are quite low, but it must be outlined that the procedures were not all carried out in ICU patients.

Thus, present data suggest that the rate of complications is low and that few are life-threatening.

Conclusion

Flexible fiberoptic bronchoscopy has become an indispensable tool in the optimal management of ICU patients with both diagnostic and therapeutic goals. Its feasibility during mechanical ventilation, with a low complication rate, has been amply demonstrated. Nonetheless, precise knowledge of the particular pathophysiological and technical aspects of performing bronchoscopy in a mechanically ventilated patient is mandatory, as is the presence of a skilled operator. Our policy regarding this last point is to always have an experienced pneumologist available during bronchoscopic examinations.

References

1. Zavala D (1975) Diagnostic fiberoptic bronchoscopy: techniques and results in 600 patients. Chest 68:12–19
2. Landa J (1978) Indications for bronchoscopy. Chest 73:686–690
3. Landa J (1981) Bronchoscopy: general considerations. In: Sackner M (ed) Diagnostic techniques in pulmonary disease. Dekker, New York Basel, pp 655–696
4. Ikeda J, Yanai N, Ishikawa S (1968) Flexible bronchofiberscope. Keio J Med 17:1–18
5. Sackner M (1975) Bronchofiberscopy. State of the art. Am Rev Respir Dis 111:62–88
6. Krell W (1988) Pulmonary diagnostic procedures in the critically ill. Crit Care Clin 4:393–407
7. Barrett C (1978) Flexible fiberoptic bronchoscopy in the critically ill patient: methodology and indications. Chest 73:746–749
8. Milledge J (1976) Therapeutic fiberoptic bronchoscopy in intensive care. Br J Med 2:1427–1429
9. Lindholm C, Ollmann B, Snyder J et al (1974) Flexible fiberoptic bronchoscopy in critical care medicine: diagnosis, therapy and complications. Crit Care Med 2:250–261
10. Zavala D (1988) Bronchoscopy, lung biopsy, and other procedures. In: Murray J, Nadel J (eds) Textbook of respiratory medicine. Saunders, Philadelphia London Toronto Montreal Sydney Tokyo, pp 562–596
11. Green C, Eisenberg J, Leong A, Nathanson I, Schnapf B, Wood R (1992) Flexible endoscopy of the pediatric airway. Am Rev Respir Dis 145:233–235
12. Fitzpatrick S, Marsh B, Stokes D et al (1983) Indications for flexible bronchoscopy in pediatric patients. Am J Dis Child 137:595–597
13. Lindholm C, Ollmann B, Snyder J, Millen E, Grenvik A (1978) Cardiorespiratory effects of flexible fiberoptic bronchoscopy in critically ill patients. Chest 74:362–367
14. Matsushima Y, Jones R, King E, Moysa G, Alton J (1984) Alterations in pulmonary mechanics and gas exchange during routine fiberoptic bronchoscopy. Chest 86:184–188
15. Perry L (1978) Topical anesthesia for bronchoscopy. Chest 73:691–693
16. Sanderson D, McDougall J (1978) Transoral bronchofiberoscopy. Chest 73:701–703
17. Ghows M, Rosen M, Chuang M, Sacks H, Teirstein A (1986) Transcutaneous oxygen monitoring during fiberoptic bronchoscopy. Chest 89:543–544
18. Albertini R, Harrel J, Moser K (1974) Hypoxemia during fiberoptic bronchoscopy. Chest 65:117
19. Manchon J, Ferber C, Roche F, Lopez M, Gallet M (1988) Intérêt de la mesure transcutanée des gaz du sang au cours des fibroscopies bronchiques en réanimation. Ann Fr Anesth Réanim 7:42–45
20. Wiedemann H, McCarthy K (1989) Noninvasive monitoring of oxygen and carbon dioxide. Clin Chest Med 10:239–254

21. Trouillet J, Guiguet M, Gibert C, Fagon J, Dreyfuss D, Blanchet F, Chastre J (1990) Fiberoptic bronchoscopy inventilated patients. Evaluation of cardiopulmonary risk under midazolam sedation. Chest 97:927–933
22. Credle W, Smiddy J, Elliott R (1974) Complications of fiberoptic bronchoscopy. Am Rev Respir Dis 109:67–72
23. Pereira W, Kovnat D, Snider G (1978) A prospective cooperative study of complications following flexible fiberoptic bronchoscopy. Chest 73:813–816
24. Surrat P, Smiddy J, Grubert B (1976) Deaths and complications associated with fiberoptic bronchoscopy. Chest 69:747–751
25. Shrader D, Lakshminarayan S (1978) The effect of fiberoptic bronchoscopy on cardiac rhythm. Chest 73:821–824
26. Kendall R (1985) Role of fiberoptic bronchoscopy in asthma. In: Weiss E, Segal M, Stein M (eds) Bronchial asthma. Mechanisms and therapeutics. Little, Brown and Co, Boston Toronto, pp 912–913
27. Sahn S, Scoggin C (1976) Fiberoptic bronchoscopy in bronchial asthma. A word of caution. Chest 69:39–42
28. Djukanovic R, Wilson J, Lai C, Holgate S, Howarth P (1991) The safety aspects of fiberoptic bronchoscopy, bronchoalveolar lavage, and endobronchial biopsy in asthma. Am Rev Respir Dis 143:772–777
29. El Sadr W, Simberkoff M (1988) Survival and prognostic factors in severe Pneumocystis carinii pneumonia requiring mechanical ventilation. Am Rev Respir Dis 137:1264–1267
30. Martin W, Smith T, Brutinel W, Cockerill F, Douglas W (1987) Role of bronchoalveolar lavage in the assessment of opportunistic pulmonary infections: utility and complications. Mayo Clin Proc 62:549–557
31. Olopade C, Prakash U (1989) Bronchoscopy in the critical care unit. Mayo Clin Proc 64:1255–1263
32. Green C (1986) Improved technique for fiberoptic intubation. Anesthesiology 64:835
33. Moorthy S, Dierdorf S (1985) An unusual difficulty in fiberoptic intubation. Anesthesiology 63:229
34. Suarez M, Chediak A, Ershowsky P, Krieger B (1987) Evaluation of a flexible fiberoptic catheter in confirming endotracheal tube placement in the intensive care unit. Respir Care 32:81–84
35. Kronberg F, Goodwin Jr W (1985) Sinusitis in intensive care unit patients. Laryngoscope 95:936–943
36. Shapiro M, Wilson K, Casar G, Bloom K, Teague R (1986) Work of breathing through different sized endotracheal tubes. Crit Care Med 14:1028–1031
37. Schmidt-Nowara W, Altman A (1984) Atelectasis and neuromuscular respiratory failure. Chest 85:792–795
38. Marini J, Pierson D, Hudson L (1979) Acute lobar atelectasis: a prospective comparison of bronchoscopy and respiratory therapy. Am Rev Respir Dis 119:971–978
39. Mahajan V, Catron P, Huber G (1978) The value of fiberoptic bronchoscopy in the management of pulmonary collapse. Chest 73:817–820
40. Clergue F, Pansard J (Update 1990) Respiratory changes induced by upper abdominal and cardiac surgery. In: Vincent J (ed) Update in intensive care and emergency medicine. Springer, Berlin Heidelberg New York Tokyo, pp 166–173
41. Barrett C, Vecchione J, Loomis Bell A (1974) Flexible fiberoptic bronchoscopy for airway management during acute respiratory failure. Am Rev Respir Dis 109:429–434
42. Wanner A, Landa J, Nieman R, Vevaiana J, Delgado I (1973) Bedside bronchofiberscopy for atelectasis and lung abscess. JAMA 224:1281–1283
43. Sunderrajan E, Davenport J (1985) The Guillain-Barré syndrome: pulmonary-neurologic correlations. Medicine 64:333–341
44. Yao Taso T, Huang Tsai Y, Shee Lan R, Bin Shieh W, Huei Lee C (1990) Treatment for collapsed lung in critically ill patients. Chest 97:435–438
45. Selecky P (1978) Evaluation of hemoptysis through the bronchoscope. Chest 73:741–745
46. Crocco J, Rooney J, Fankhusen D et al (1968) Massive hemoptysis. Arch Intern Med 121:495–498
47. Imgrund S, Goldberg S, Walkenstein M, Fischer R, Lippmann M (1985) Clinical diagnosis of masive hemoptysis using the fiberoptic bronchoscope. Crit Care Med 13:438–443
48. Gourin A, Garzon A (1975) Control of hemorrage in emergency pulmonary resection for massive hemoptysis. Chest 68:120–121
49. Saw E, Gottlieb L, Yokoyama T, Lee B (1976) Flexible fiberoptic bronchoscopy and endobronchial tamponade in the management of massive hemoptysis. Chest 70:589–591
50. Bense L (1990) Intrabronchial selective coagulative treatment of hemoptysis. Chest 97:990–996
51. Gottlieb L, Hillberg R (1975) Endobronchial tamponade therapy for intractable hemoptysis. Chest 67:482–483
52. Bishop M (1989) Mechanisms of laryngotracheal injury following prolonged tracheal intubation. Chest 96:185–186
53. Nordin U, Lindholm C, Wolgast M (1977) Blood flow in the rabbit trachea mucosa under normal conditions and under the influence of tracheal intubation. Acta Anaesthesiol Scand 21:81–94
54. Conlan A, Hurwitz S (1980) Management of massive haemoptysis with the rigid bronchoscope and cold saline lavage. Thorax 35:901–904
55. Lang N, Maners A, Broadwater J, Shewmake K, Chu D, Westbrook K (1988) Management of airway problems in lung cancer patients using the Neodymium-Yttrium-Aluminium-Garnet (Nd-YAG) laser and endobronchial radiotherapy. Am J Surg 156:463–465
56. Uflaker R, Kämerer A, Picon P et al (1985) Bronchial artery embolization in the management of hemoptysis. Radiology 157:637–644
57. Andrews C, Coalson J, Smith J, Johanson W (1981) Diagnosis of nosocomial pneumonia in acute diffuse lung injury. Chest 80:254–258
58. Craven D, Kunches L, Kilinsky V, Lichtenberg D, Make B, McCabe W (1986) Risk factors for pneumonia and fatality in patients receiving continuous mechanical ventilation. Am Rev Respir Dis 133:792–796
59. Kahn R, Arich C, Baron D et al (1990) Diagnostic des pneumonies nosocomiales en réanimation. 5ème conférence de consensus en réanimation et médecine d'urgence. Réan Soins Intens Méd Urg 6:91–124
60. Montgomery A, Stager M, Carrico C, Hudson L (1985) Causes of mortality in patients with the adult respiratory distress syndrome. Am Rev Respir Dis 132:485–489
61. Fagon J, Chastre J, Domart Y, Trouillet J, Pierre J, Darné C, Gibert C (1989) Nosocomial pneumonia in patients receiving continuous mechanical ventilation. Am Rev Respir Dis 139:877–884
62. Salata R, Lederman M, Schlaes D, Jacobs M, Eckstein E (1987) Diagnosis of nosocomial pneumonia in intubated, intensive care unit patients. Am Rev Respir Dis 135:426–432
63. Torres A, Puig de la Bellacasa J, Xaubert A et al (1989) Diagnostic value of quantitative cultures of bronchoalveolar lavage and telescoping plugged catheter in mechanically ventilated patients with bacterial pneumonia. Am Rev Respir Dis 140:306–310
64. Meduri G (1990) Ventilatory-associated pneumonia in patients with respiratory failure. A diagnostic approach. Chest 97:1208–1219
65. Gaussorgues P, Piperno D, Bachmann P (1989) Comparison of nonbronchoscopic bronchoalveolar lavage to open lung biopsy for the diagnosis of plumonary infections in mechanically ventilated patients. Intensive Care Med 15:94–98
66. Chastre J, Fagon J, Soler P, Bornet M, Domart Y, Trouillet J, Gibert C, Hance A (1988) Diagnosis of nosocomial bacterial pneumonia in intubated patients undergoing ventilation: comparison of the usefulness of bronchoalveolar lavage and the protected specimen brush. Am J Med 85:499–506
67. Kahn F, Jones J (1987) Diagnosing bacterial infection by bronchoalveolar lavage. J Infect Dis 155:862–869
68. Reynolds H (1987) State of the art: bronchoalveolar lavage. Am Rev Respir Dis 135:250–263
69. Johanson W, Seidenfeld J, Gomez P, de los Santos R, Coalson J (1988) Bacteriologic diagnosis of nosocomial pneumonia folowing prolonged mechanical ventilation. Am Rev Respir Dis 137:259–264
70. Faling J (1988) New advances in diagnosing nosocomial pneumonia in intubated patients. Am Rev Respir Dis 137:253–255

71. Fagon J, Chastre J, Hance A, Guiguet M, Trouillet J, Domart Y, Pierre J, Gibert C (1988) Detection of nosocomial lung infection in ventilated patients. Use of a protected specimen brush and quantitative culture techniques in 147 patients. Am Rev Respir Dis 138:110–116
72. Chastre J, Viau F, Brun P, Pierre J, Dauge M, Bouchama A, Akesbi A, Gibert C (1984) Prospective evaluation of the protected specimen brush for the diagnosis of pulmonary infections in ventilated patients. Am Rev Respir Dis 130:924–929
73. Villers D, Deriennic M, Raffi F (1985) Reliability of the bronchoscopic protected catheter brush in intubated and ventilated patients. Chest 88:527–530
74. Wimberley N, Faling L, Bartlett J (1979) A fiberoptic bronchoscopy technique to obtain uncontaminated lower airway secretions for bacterial culture. Am Rev Respir Dis 119:337–343
75. Matthew E, Holstrom F, Kaspar R (1977) A simple method for diagnosing pneumonia in intubated or tracheostomized patients. Crit Care Med 5:76–81
76. Mann J, Altus C, Webber C (1987) Nonbronchoscopic lung lavage for diagnosing opportunistic infection in AIDS. Chest 91:319–322
77. Tobin M, Grenvik A (1984) Nosocomial lung infection and its diagnosis. Crit Care Med 12:191–199
78. Thorpe J, Baughman R, Frame P, Wessler T, Staneck J (1987) Bronchoalveolar lavage for diagnosing acute bacterial pneumonia. J Infect Dis 155:855–861
79. Chastre J, Fagon J, Soler P, Domart Y, Pierre J, Dombret M, Gibert C, Hance A (1989) Quantification of BAL cells containing intracellular bacteria rapidly identifies ventilated patients with nosocomial pneumonia. Chest 95:190S–192S
80. Andersen H (1978) Transbronchic lung biopsy for diffuse pulmonary diseases. Chest 73:734–736
81. Williams D, Yungbluth M, Adams G, Glassroth J (1985) The role of fiberoptic bronchoscopy in the evaluation of immunocompromised hosts with diffuse pulmonary infiltrates. Am Rev Respir Dis 131:880–885
82. Golden J, Hollander H, Stulbarg M, Gamsu G (1986) Bronchoalveolar lavage as the exclusive diagnostic modality for Pneumocystis carinii pneumonia. Chest 90:18–22
83. Papin T, Grum C, Weg J (1986) Transbronchial biopsy during mechanical ventilation. Chest 89:168–170
84. Pincus P, Kallenbach J, Hurwitz M, Clinton C, Feldman C, Abramowitz J, Zwi S (1987) Transbronchial biopsy during mechanical ventilation. Crit Care Med 15:1136–1139
85. Stover D, Zaman M, Hajdu S, Lange M, Gold J, Armstrong D (1984) Bronchoalveolar lavage in the diagnosis of diffuse pulmonary infiltrates in the immunosuppressed host. Ann Intern Med 101:1–7
86. Broaddus C, Dake M, Stulbarg M, Blumenfeld W, Hadley K, Golden J, Hopewell P (1985) Bronchoalveolar lavage and transbronchial biopsy for the diagnosis of pulmonary infections in the acquired immunodeficiency syndrome. Ann Intern Med 102:747–752
87. Murray J, Mills J (1990) Pulmonary infectious complications of human immunodeficiency virus infection. Part I. Am Rev Respir Dis 141:1356–1372
88. Caughey G, Wong H, Gamsu G, Golden J (1985) Nonbronchoscopic bronchoalveolar lavage for the diagnosis of Pneumocystis carinii pneumonia in AIDS. Chest 88:659–662
89. Pugin J, Auckenthaler R, Mili N, Janssens J, Lew D, Suter P (1991) Diagnosis of ventilatorassociated pneumonia by bacteriologic analysis of bronchoscopic and nonbronchoscopic "blind" bronchoalveolar lavage fluid. Am Rev Respir Dis 143:1121–1129
90. Pham L, Brun-Buisson C, Legrand P, Rauss A, Verra F, Brochard L, Lemaire F (1991) Diagnosis of nosocomial pneumonia in mechanically ventilated patients. Comparison of a plugged telescoping catheter with the protected specimen brush. Am Rev Respir Dis 143:1055–1061
91. Bishop M, Benson M, Sato P, Pierson D (1987) Comparison of high-frequency jet ventilation with conventional mechanical ventilation for bronchopleural fistula. Anesth Analg 66:833–838
92. Schlenkhoff D, Droste H, Scieszka S, Vogt H (1986) The use of high-frequency jet ventilation in operative bronchoscopy. Endoscopy 18:192–194
93. Wetzel R, Gioia F (1987) High frequency jet ventilation. Pediatr Clin North Am 34:15–38
94. Hamilton P, Onayemi A, Smyth J et al (1983) Comparison of conventional and high-frequency ventilation: oxygenation and lung pathology. J Appl Physiol 55:131–138
95. Keszler M, Klein R, McClellan L, Nelson D, Platt M (1982) Effects of conventional and high-frequency jet ventilation on lung parenchyma. Crit Care Med 10:514–516
96. Harris J, Gooch W, Wilson J, Avent J (1984) Necrotizing tracheobronchitis associated with high-frequency jet ventilation. Clin Res 32:132A
97. Ophoven J, Mammel M, Gordon M, Boros S (1984) Tracheobronchial histopathology associated with high-frequency jet ventilation. Crit Care Med 12:829–832
98. Kirpalani H, Higa T, Perlman M, Friedberg J, Cutz E (1985) Diagnosis and therapy of necrotizing tracheobronchitis in ventilated neonates. Crit Care Med 13:792–797
99. Tolkin J, Kirpalani H, Fitzhardinge P, Swyer P, Cutz E, Higa T (1984) Necrotizing tracheobronchitis: a new complication of neonatal mechanical ventilation. Pediatr Res 18:391A
100. Mammel M, Ophoven J, Lewallen P, Gordon M, Boros S (1991) Acute airway injury during high-frequency jet ventilation and high-frequency oscillatory ventilation. Crit Care Med 19:394–398
101. Delafosse C, Chevrolet J, Suter P, Cox J (1988) Necrotizing tracheobronchitis: a complication of high-frequency jet ventilation. Virchows Archiv Pathol Anat 413:257–264
102. Nordin U, Klain M, Keszler H (1982) Electron-microscopic studies of tracheal mucosa after high-frequency jet ventilation. Crit Care Med 10:A211
103. Carlon G, Barker R, Benna R, Guy J (1985) Airway humidification with high-frequency jet ventilation. Crit Care Med 13:114–117
104. Hara K, Prakash U (1989) Fiberoptic bronchoscopy in the evaluation of acute chest and upper airway trauma. Chest 96:627–630
105. Kirsh M, Orringer M, Behrendt D, Sloan H (1976) Management of tracheobronchial disruption secondary to nonpenetrating trauma. Ann Thorac Surg 22:93–101
106. Cunanan O (1978) The flexible fiberoptic bronchoscope in foreign body removal. Chest 73:725–726
107. Fieselmann J, Zaval D, Keim L (1977) Removal of foreign bodies by fiberoptic bronchoscopy. Chest 72:241–243
108. Gerasin V, Shafirovsky B (1988) Endobronchial electrosurgery. Chest 93:270–274
109. Schray M, McDougall J, Martinez A, Cortese D, Brutinel W (1988) Management of malignant airway compromise with laser and lowe dose rate brachytherapy. Chest 93:264–269
110. Fulkerson W (1984) Fiberoptic bronchoscopy. N Engl J Med 311:511–515
111. Pereira W, Kovnat D, Khan M, Iacovino J, Spivack M, Snider G (1975) Fever and pneumonia after flexible bronchoscopy. Am Rev Respir Dis 112:59–64
112. Kane R, Cohen M, Fossieck Jr. B, Tvardzick A (1975) Absence of bacteremia after fiberoptic bronchoscopy. Am Rev Respir Dis 111:102–104
113. Higuchi JH, Coalson JJ, Johanson WG (1982) Bacteriologic diagnosis of nosocomial pneumonia in primates. Usefulness of the protected specimen brush. Am Rev Respir Dis 125:53–57

Dr. J.C. Chevrolet
Soins Intensifs de Médecine
Hôpital Cantonal Universitaire
24, Rue Micheli-du-Crest
CH-1211 Geneva 4
Switzerland