Descending necrotizing mediastinitis (reply)
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Descending necrotizing mediastinitis

H Esteva

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Usefulness of Collecting Routine Cytologic Specimens During Fiberoptic Bronchoscopy for Endoscopically Visible and Nonvisible Lung Carcinoma

To the Editor:

We read with great interest the article by Govert et al, published in the February 1996 issue of CHEST. We forward some comments based on local experience and a supplementary review of the pertinent literature.

Between April 1994 and March 1996, we had performed 1,387 fiberoptic bronchoscopy examinations for various usual indications. We used a slim-bodied fiberoptic bronchoscope (Pentax FB 15X) and forceps (Pentax KW 1811S) for obtaining endobronchial and transbronchial biopsy specimens and a cytology brush (Mill-Rose Laboratory Product 149) for bronchial brushings. Among 1,387 cases, 329 lung carcinomas were diagnosed. Two hundred eight cases were endoscopically visible (group A); 121 cases were endoscopically nonvisible (group B). The most common abnormal findings in group B cases were extrinsic compression or mucosal swelling of bronchopulmonary segmental orifices.

In the group A cases, 97.1% of the cases were diagnosed by biopsy specimen. Only 2.9% were diagnosed solely by cytologic examination of bronchial washings, bronchial brushings, or a combination of the two techniques. This contrasts greatly with the findings of the earlier published series from Mak et al, respectively. These increases in yield obtained are quite comparable. Fluoroscopic guidance also seems to exert no major impact on the relative contribution to the diagnostic yield of the three techniques. However, utilization of fluoroscopic guidance might be expected to enhance the overall diagnostic yield of a lung carcinoma that is not endoscopically visible by the fiberoptic bronchoscope. Unfortunately, discrepancies have still been noted among different series with overall diagnostic yield rates that ranged widely from about 20% to 50%. Lately another rather interesting article by Chechani published in the March 1996 issue of CHEST, revealed an overall diagnostic rate of 80% for nonendoscopically visible tumors by fiberoptic bronchoscopy under biplane fluoroscopic screening. Bronchial brushings, transbronchial forceps biopsies, and transbronchial needle biopsies each had a yield of about 50% and a combination of these three techniques was advocated for optimizing diagnosis for these cases. However, routine fluoroscopic guidance and use of disposable transbronchial biopsy cytology needles might be exceedingly demanding on physical and human resources in departments that have bronchoscopy service with high throughput like ours. Therefore, in our setting, we still consider routine cytologic examination of bronchial washings and brushings in addition to transbronchial forceps biopsies conducive to a significant escalation of the diagnostic yield of endoscopically nonvisible lung carcinoma. In particular, each cytologic examination in Hong Kong costs only $25 to $65 in contrast to $30 to $250 in the United States, as cited by Govert et al. We currently only perform fiberoptic bronchoscopy under fluoroscopic guidance in...
selected cases, especially those that represent repetition of previously unyielding procedures.

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REFERENCES

To the Editor:

We appreciate the comments of Dr. Chau et al, and value the opportunity to respond by emphasizing a few important points. In addition to the report by Chau et al, a recently published abstract by Chittock et al confirms that routine collection of washings and brushings for endobronchial lesions is not cost-effective. The fact that these reports from three different countries on two continents all reach the identical conclusion speaks to the robustness of the conclusions; namely that it is unnecessary to collect both brushings and washings during bronchoscopy for endobronchial lesions. We still continue to believe that collection of either washings or brushings is cost-effective at our institution. However, this conclusion may not be universally true because any cost analysis is based on the relative costs at individual institutions.

The findings by Chau et al that brushings and washings are the only diagnostic modality in 7.8% and 9.5% of endoscopically nonvisible tumors is quite interesting. These findings coupled with the findings of Chechani appear to confirm our suspicion that there is a very real role for cytologic specimens in endoscopically nonvisible tumors. However, it remains unclear which combination of cytology specimens is the most cost-effective. It would be quite interesting to know the overall sensitivity of bronchoscopy for the endoscopically nonvisible lesions in the series by Chau et al compared to that found by Chechani. If the sensitivities were similar, it would bring into question our routine use of fluoroscopy to guide the biopsy of endoscopically nonvisible lesions.

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REFERENCES
Minocycline Sclerosis for Malignant Pericardial Effusions

To the Editor:

Dr. Markiewicz and his group, who have published many outstanding investigations, reported in CHEST (June 1996) on successful minocycline sclerosis for malignant pericardial effusions. 1 Half their patients had severe pain (which also occurs with other sclerosants 2, 3 ) two had diffuse ST-T changes suggesting pericardial or subepicardial injury. Both of these raise questions of whether this effective agent is also optimal for sclerotherapy. Indeed, the distinguished authors recognized that “a randomized study is required.” One wonders why this was not done, since they included “all patients with compressive malignant pericardial effusions,” implying a prospective investigation. They also overlooked another basic concept in prospective randomized controlled trials—the ethical imperative to give all patients a “50-50 chance” 3 not to get the investigational therapy lest it be worse than placebo or established therapy. 1 This is accomplished by randomizing the first patient, even while gaining experience in dosing and collateral effects (the discredited “pilot” studies). 3 Moreover, in our experience (still under investigation), indwelling pericardial tubes—soft, flat, multihole—stimulate dense adhesions without irritative agents. 3 Finally, a description of the ST-T changes in their patients may be enlightening to suggest their nature and for comparison with other therapies.

Despite the foregoing considerations, the authors 1 can be complimented for solidly adding minocycline to the large list of pericardial sclerosants 2 with the hope that they may do the randomized study they mention.

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REFERENCES


To the Editor:

We read with interest Dr. Spodick’s letter and fully agree with his comments. Randomized studies are needed to evaluate whether agents causing sclerosis of the pericardium are more effective than drainage alone in preventing recurrence of malignant pericardial effusion. Excellent randomized studies address this issue in patients with pleural effusion. 1,2 Similar studies are sorely lacking for pericardial effusion, possibly owing to the small number of patients with malignant pericardial effusion seen in any given center, and to the assumption that agents effective for the pleura are probably effective for the pericardium. Our study in the dog with a normal pericardium indicates that instillation of minocycline (Minocin) through a pericardial drain is more effective in causing adhesions than instillation of normal saline solution. 3 This experimental study does not necessarily demonstrate the effectiveness of the drug in the human with pericardial disease.

ECG changes following intrapericardial injection of minocycline in two patients consisted of marked upwardly concave elevation of the ST-T segment in leads L1, L2, aVL, aVF, V4-V6, associated with severe inspiratory chest pain, but no obvious hemodynamic changes. ECG returned to baseline within 6 h, together with resolution of chest pain. No enzymatic or ECG evidence of myocardial infarction was noted. One of the two patients with ECG changes died suddenly of circulatory collapse 24 h later.

This patient had terminal cancer and his death was not unexpected. Postmortem examination was not performed and the possibility that minocycline contributed to death cannot be excluded. We recommend that patients receiving minocycline intrapericardially be followed up closely for possible untoward side effects.

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Buddha

To the Editor:

There is an alternative interpretation to the Buddhist teachings surrounding death and dying, which emphasizes the importance of life as well as maintaining and prolonging a meaningful life. The single purpose of Buddhism is to demonstrate to mankind the path to enlightenment; it essentially has no other purpose. Furthermore, the path to enlightenment cannot be pursued effectively except while enmeshed in the world of karma, that is, during the present lifetime here on earth. Unlike other religions that emphasize the importance of the afterlife, the Buddhist philosophy tends to deemphasize it. Indeed, Buddha often discouraged speculation on the afterlife, existence of God, or other theological topics that are the cornerstones of many other religions. The reason for this is that the urgency to achieve enlightenment in Buddhism is so great that there is no time to waste energy on other less critical objectives. Buddha often used this analogy of a burning building: if one were trapped in a burning building, would one take the time to inquire as to who set the fire, why it happened, what kind of fire it was, etc? Certainly, these questions can wait until after exiting the building. In Buddhism, human life is taught as extraordinarily precious precisely because only it offers the promise of achieving enlightenment and permanently escaping from the “fires” of desire, greed, and ignorance.

Buddha always preached about the importance of maintaining health, since without a healthy body one’s path to enlightenment was obstructed; the body is, after all, an important tool to use toward the achievement of enlightenment. This was why prior to his enlightenment, Buddha so carefully nursed himself back to

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health after leaving the ascetic colony where he had spent 6 years studying. The irony of the editorial “CPR or DNR: Lessons From Buddha” is that the authors attempt to use an exception to prove the rule. That is, they are using the example of the Buddha (an enlightened being) as a model for how we (mostly unenlightened beings) should behave. According to Buddhist doctrine, from the very moment that Buddha became enlightened, his life was no longer necessary for his development. He could have entered nirvana then and there (which would have meant instant death to his earthly body) and not suffered any karmic repercussions. Buddha chose to stay on earth for many more years out of a profound compassion for all of mankind and a desire to liberate as many people as he could. Since he had already achieved enlightenment, it made no difference to him (from a Buddhist perspective) when he died since his remaining life was essentially a gift to all mankind. The sense of urgency regarding achieving his enlightenment no longer existed.

This interpretation of Buddhist teachings leads me to a different conclusion regarding an approach to DNR and CPR. I thoroughly agree with the authors that the fear of the unknown and the fear of death cannot be acceptable reasons for unrealistic and unaffordable treatment. However, such types of decisions must always be tempered with an appreciation of the enormous value of human life. The way I interpret Buddhism’s philosophy toward this issue is that “where there is life there is hope.” Buddhism teaches that within each one of us there exists a latent enlightenment. One may only think back to the self-immolation of the Buddhist monks in Vietnam, the zen samurai warriors adhering to their creed of busido, or the less well-known Mahayanist sacrifices (ritual suicide is described in the Lotus Sutra chapter XXII. Charles Eliot in Japanese Buddhism, argues against the practice and the Buddha’s condoning of it1 to see how the doctrine was later interpreted thousands of miles from the site and over 2,000 years after the founder’s sermons.

Charles Stimler, MD
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REFERENCE

To the Editor:

Universal religious teachings such as Buddhism are treasured for their great wisdom. Such systems of thought appeal to a vast populace, with each individual understanding the points of the doctrine in his own unique way. It is hard to define a religious truth of any tradition that will find complete consensus. This is all the more true for Buddhism, which was orally transmitted for over 500 years before the teachings were written down, and by that late date, no unani mity could be found. Consequently, there never was a codified “bible,” rather a compilation of sacred Buddhist scriptures that number in the tens of thousands. (Columbia University has a copy of the thousands of sacred volumes of the Taisho edition of the Tripitaka.)

Certainly no one could argue against the great compassion of the Buddha. This was the central tenet of his teaching. The fact that he did not address the question of an afterlife or of a god is a natural corollary to his doctrines. Indians believe in karma—infinite rebirth. The Buddhist understanding was that life is inherently painful, and that infinite rebirth propels pain infinitely. Since the ancient Vedic gods of India (it was not a monothestic society) were also subject to karma, deities were useless in the pursuit of enlightenment. This is why in the scriptural tradition when Mara, god of death and desire, offers the meditating Buddha-To-Be a high place in the ranks of the god, he is rejected.2 If one teaching can be firmly associated with the Buddhist doctrine, it is the one encapsulated in the Four Truths—attachment leads to pain.3 This truth can be expressed more concretely as the attachment to life, for it alone leads to rebirth. Extinguishing the desire to live is the means to nirvana.

Dr. Stimler takes exception to “using the example of the Buddha as a model for how we should behave,” but it is important to take enlightened people, such as the Buddha, as models, though we ourselves may fall short of that blessed state. It is only by studying the wisdom of those who understand the human condition and what is frankly possible that reasonable goals be set and hopefully achieved. Further, one cannot apply the Buddha’s death bed sermon urging healthy people to be aware of their mortality and to try and detach themselves from the karmic conditions of the wheel of suffering as an excuse to prolong the pain-filled life of terminally ill people. As Dr. Stimler himself quotes the Buddha as saying, “A sick body is a hindrance to enlightenment.”

Prolonging life at any cost is not a Buddhist value. In fact, the practice of suicide was sometimes considered a symbolic act of enlightenment. One may only think back to the self-immolation of the Buddhist monks in Vietnam, the zen samurai warriors adhering to their creed of busido, or the less well-known Mahayanist sacrifices (ritual suicide is described in the Lotus Sutra chapter XXII. Charles Eliot in Japanese Buddhism, argues against the practice and the Buddha’s condoning of it1 to see how the doctrine was later interpreted thousands of miles from the site and over 2,000 years after the founder’s sermons.

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Measurement of Pleural Fluid Cholesterol Levels

To the Editor:

In a letter to the editor in CHEST (July 1996), Romero et al1 report that the criteria of pleural cholesterol level >45 mg/dL and pleural lactate dehydrogenase (LDH) level >200 IU, proposed by us for the identification of pleural exudates,2 have been inaccurate when applied to their patients, especially regarding specificity. Searching for an explanation for this discrepancy, we have become aware that we are using different methods for the determination of LDH activity, so that the upper normal limits for serum are not the same. Therefore, the cutoff point of 200 IU does not have the same meaning for both groups. In our article,
we made the mistakes of not pointing out that with our method our upper normal value in serum is 225 IU, and of not expressing the proposed cutoff point as 88.8% of that value, instead of the absolute number of 200 IU, which is valid only for laboratories using exactly the same method we use.

As the normal upper limit for serum for the group of Romero et al. is 460 IU, the cutoff point of 200 IU, which represents only 43% of their normal upper limit, obviously results in a very low specificity, only partially improved when they use 307 IU, equivalent to 66.6% of their normal upper limit. We presume that with a cutoff point of 408 IU or 88.8% of their upper normal limit in serum, the results in their patients may be similar to ours. Specificity should increase while a good sensitivity is assured by the low cutoff point of 45 mg/dL for cholesterol.

We think the same explanation is valid for the letter of Drs. Garcia-Pachon and Padilla-Navas, but we cannot be sure because they do not report their upper normal value.

Without pretending to justify our mistakes, we think it is necessary to point out that similar errors are, surprisingly, present in most of the articles reviewed by us: only four report their upper normal limits for serum, which range from 237 to 460 IU,4,6,7 and only two explicitly express the cutoff point for LDH as a proportion of the upper normal limit in serum.3,5 In another three, this proportion may be calculated4,6,7 and in only three the criteria for pleural LDH of two-thirds the normal upper limit for serum, stated by Light8 in 1983, are applied exactly.3,5

We will not go into further detail because we think that these aspects will be fully considered in the meta-analysis that is being conducted by John E. Heffner, who has requested from us and other groups all the data concerning laboratory methods and the individual results of patients.

To conclude, we would like to emphasize that all future studies involving pleural LDH should be required to report the upper normal limit for serum of that particular laboratory and to express the cutoff point as a proportion of that value.

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Pseudoaneurysm of the Left Ventricle After Mitral Valve Replacement

Surgical Repair or Medical Follow-up

To the Editor:

I read with great attention and interest the article by Fazia and coworkers (February 1996)1 and the case reports of Blinc and coworkers (February 1996)2 concerning long-term survival without emergency surgical repair of the four patients with left ventricular pseudoaneurysm following myocardial infarction and mitral valve replacement.

Treatment of these patients, especially with a large pseudoaneurysm, by heart transplantation prior to decompensation, seems to be a very safe solution of the problem for centers with a cardiac transplant program.

I would like to describe herein a patient who developed subannular pseudoaneurysm of the left ventricle after mitral valve replacement. In this case also, “unexpected” 6-year medical follow-up was possible without any surgical attempt.

A 47-year-old woman was first operated on 16 years ago for closed mitral valvotomy. She underwent reoperation in 1989 and the heavily calcified mitral valve was replaced with 29-mm mechanical prosthesis (Medtronic). A De Vega annuloplasty was also done for the insufficient tricuspid valve. The patient had an uneventful recovery. In July 1990, however, she presented as New York Heart Association class IV, and a chest radiograph revealed right lateral enlargement of the cardiac silhouette. Echocardiographic studies and cardiac catheterization showed a subannular posterior left ventricular pseudoaneurysm formation and paravalvular leakage. Her functional status improved with medical therapy (New York Heart Association class II to III) and the patient did not want surgery. Cardiac catheterization was repeated in July 1995. On the follow-up, the sizes of the pseudoaneurysm were not changed, and there is (+/+-++) mitral regurgitation due to the paravalvular leakage (Fig 1).

We collected 26 similar cases reported between 1969 and 1994.3-5 The time intervals elapsed from the last surgical procedure on mitral valve to the diagnosis of the pseudoaneurysm vary from 2 weeks to 7 years. One of them was found at autopsy.4 Fifteen cases were treated surgically. Rereplacement of the prosthetic mitral valve and closure of the pseudoaneurysm with a pericardial or polytetrafluoroethylene or Dacron or Teflon patch were generally done. Five of the patients did not survive. Another one survived with a residual channel from left ventricle to pseudoaneurysm. Ten patients were followed-up medically from 5 months to 10 years, without any catastrophe, and there are two reported spontaneous closures.5,6

Prompt surgical repair is said to be the first choice of therapy. Recommended surgical technique of repair is removal of the previous prosthesis, internal closure of the orifice of the pseudoaneurysm using a patch, and reimplantation of a new valve. However, especially for patients who had several previous surgical interventions, medical follow-up and echocardiographic survey of the dimensions of the pseudoaneurysm may be safer.

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Is Aging a Risk Factor for Silent Ischemic Cardiopathy?

To the Editor:

One of the most credited theories about the origin of silent myocardial ischemia is the raising of the pain threshold. The relationship between silent ischemia and old age has not yet been defined clearly, but age is often considered a risk factor for this disease.

In our study, we assessed the course of pain threshold and pain tolerance in subjects affected by silent ischemia and attempted to define the role played by age.

We studied 23 male subjects (mean age 64.7 ± 8.5 years) affected by silent ischemic cardiopathy and 20 male patients (mean age 68.5 ± 8.1 years) with symptomatic cardiac ischemic disease and evaluated the pain threshold in three different sites (forearm, shoulder, precordium), using electromyographic equipment with a protocol that provides short (0.05 ms) and low-frequency (0.5 Hz) endermic electric stimuli. This was obtained by taking five measurements and calculating the mean of the three central ones.

Pain tolerance was then determined by increasing the stimuli. Exclusion criterion was the presence of significant mental disorders such as excessive anxiety or depression and this was determined using the Hamilton test and the Assessment Scale of Emotivity and Wellbeing in the Elderly (cutoff point was, respectively, at 18 and 6).

Values of pain threshold were assessed using the same method in a group of 40 healthy subjects, five for each age decade, between 10 and 90 years.

Our data show a significant difference of pain threshold and tolerance between subjects with silent and symptomatic cardiopathies (34.7 ± 12.6 mA vs 25.2 ± 12.5 mA; p<0.001 for the threshold; and 68.5 ± 21.2 mA vs 46.0 ± 22.3; p<0.001 for tolerance).

The fact that the significance of our results is superior to other studies can be due to the particular method of stimulation used and to the uniformity of the sample studied (sex, age, exclusion of subjects with anxiety and depressive symptoms). No difference
was found in pain threshold regarding age. It seems probable that the difference found between subjects with silent and symptomatic cardiopathies is due to the different modulation of the perception of pain at a central level, independent of the age factor.

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Treatment Algorithm for OSA

To the Editor:

I read with interest the review article by Hudgel (May 1996) on obstructive sleep apnea (OSA) treatment. The article includes a treatment algorithm for initial therapy for OSA in which patients are first treated with nasal continuous positive airway pressure (CPAP) or weight loss plus medication. According to studies described below, weight loss plus medication should not be considered an effective treatment for OSA in most cases.

There are few studies of more than five patients that demonstrate weight loss to be effective in the treatment of OSA. Rubinstein et al. used hospitalization and a very low calorie diet to achieve a 26 kg weight loss in 12 OSA patients, with a reduction in the respiratory disturbance index (RDI) from 57 to 14 events/h. A recent study of 39 OSA patients receiving gastrectomy resulted in a 9 kg weight loss, but the RDI decreased only from 67 to 50 events/h. Behavioral therapy is a less intensive therapy and resulted in a 2.2 kg weight loss in 127 obese non-OSA patients at 2 years; however, a mean weight loss of 3.6 kg did not result in a decreased RDI in a study of 19 obese OSA patients by Braver et al.

Medications have not been shown to be effective in the treatment of OSA except in specific cases, such as hypothyroidism. Antidepressants suppress rapid eye movement sleep and may decrease OSA severity, although none of these agents is a clinically useful treatment in most patients. A study by Hanzel et al. using fluoxetine and protriptyline to treat OSA in 12 patients demonstrated a reduction in the RDI from 57 to 34 events/h, but only 17% of these patients had both a posttreatment RDI <20 events/h and a ≥50% reduction in RDI. Other drugs, such as theophylline and progesterone, have shown even less clinical efficacy, according to Hudgel.

Treatment with oral appliances (OA) was not included in Hudgel’s algorithm. A recent prospective, double-crossover study by Ferguson et al. showed that while OA treatment was not as effective as CPAP, it was an effective treatment in 48% of OSA patients, especially those with mild or moderate OSA. In conclusion, weight loss plus medication should not be considered as a first-line treatment for OSA patients. More effective initial treatment alternatives to CPAP include OA and pharyngeal and jaw surgeries.

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The opinions or assertions contained herein are the private views of the authors and do not necessarily represent the opinion of the Department of the Army or of the Department of Defense.

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To the Editor:

I appreciate the opportunity to respond to Dr. Loube’s letter concerning my review of the treatment of obstructive sleep apnea (OSA). Dr. Loube contends that weight loss and medications should not be recommended as a first line of treatment for OSA. He would recommend either nasal continuous positive airway pressure (CPAP) or upper airway surgery as the initial treatment option discussed with a new untreated sleep apnea patient.

In the military where Dr. Loube practices, extremely obese OSA patients likely are not encountered. However, in the civilian population, obesity is quite common in OSA patients. Several studies have shown an improvement in upper airway function and a decrease in apnea with weight loss. These findings are discussed in the review. Obviously, we would be remiss if we did not strongly encourage our obese OSA patients to lose weight.

Although the medical treatment of obstructive sleep apnea is in its infancy, some promising results are available. The relationship between serotonin abnormalities and sleep apnea is not totally clarified, but studies have provided initial promising results. Tryptophan, fluoxetine, and buspirone—agents that increase brain serotonin activity by different mechanisms—all improve sleep apnea in some patients. Although it is not yet clear which patients are the best candidates for this therapy, the use of medications can be more convenient than CPAP, if they are as
effective. Surely, this area needs further research; in the future, we hope to have more specific indications for medications in the treatment of sleep apnea.

The use of oral appliances is addressed in my review, but at this time there is insufficient data to know where to place this mode of therapy in the treatment algorithm. Since my review was submitted, the manuscript by Ferguson et al was published and shows promising results. As supportive evidence grows, this form of therapy may become one of our first line treatments.

Surely, CPAP is often prescribed initially for the treatment of OSA. However, studies documenting that patient compliance is often considerably less than ideal influenced the placement of CPAP in my recommended algorithm. Surgery for sleep apnea is not successful enough to be a first line treatment at this time. New findings about pharyngeal surgery, discussed in the review, are worrisome. First, the pharynx may revert to the original caliber several months after surgery. Second, late uvulopalatopharyngoplasty failures are being reported. Third, pharyngeal surgery is often performed without preoperative identification of the site of obstruction.

In summary, I am aware that there are multiple approaches to treating the OSA patient. In spite of the treatment chosen, the response to a given treatment choice should be objectively measured, be it by monitoring CPAP machine timer, repeat polysomnogram following a few weeks of drug therapy, or documentation of improvement in apnea following surgery. When there is not an adequate response, the therapy should be changed based on this objective evidence. Our goal is to have the sleep apnea patient’s health improved with a form of treatment that is effective over time, and with which the patient is satisfied.

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REFERENCES


Descending Necrotizing Mediastinitis

To the Editor:

The report by Dr. Kruyt and colleagues (June 1996) of a case of descending necrotizing mediastinitis (DNM) causing pleuroesophageal fistula, treated without aggressive surgery, is interesting but should not be taken as general advice.

In the last few years, we treated 4 patients with DNM of dental origin, and only the fourth survived. We learned from experience to be as aggressive as the disease is, even if the patient seems to be in good condition. Dental resection, wide open drainage of the oral and cervical process, and thoracotomy are therefore agree with Dr. Esteva that sometimes an aggressive approach long lasting; and what are the most useful tests for postoperative monitoring?

In the young patient with DNM, a small thoracotomy adds no significant functional risk but can be the best approach for the definitive surgical treatment of mediastinal, pleural, and pericardial collections.

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REFERENCES


To the Editor:

We thank Dr. Hugo Esteva for his remarks on our recent article about a case of descending necrotizing mediastinitis (DNM) causing a pleuroesophageal fistula. As indicated by the experience of Dr. Esteva, who describes 4 cases of DNM of whom 3 died, DNM can be a life-threatening infection. We therefore agree with Dr. Esteva that sometimes an aggressive treatment is necessary. If the cause of DNM is adequately treated and the patient is in good condition, a stepwise approach is justified, as described in our case report. Initially, DNM can be treated without aggressive surgical therapy. However, in the case of further deterioration of the patient, more extensive surgical drainage is indicated.

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Lung Volume Reduction Surgery

To the Editor:

In Toronto, Canada, we have had a major interest in the treatment of emphysema by lung volume reduction surgery (LVRS), particularly in the period 1965 to 1975. There were publications and presentations at that time. Some of our data have never been published, but these may be of help toward answering questions that are of present concern: how does one select the best patients for the operation; is the benefit of the operation long lasting; and what are the most useful tests for postoperative monitoring?

In selecting patients, worthwhile results were most likely when the vascular pattern was defined. Space occupation occurs when there is avascular change in the destroyed lung tissue, but it is also important to show that there is good vascularization in the remaining lung. Pulmonary angiograms and radioactive lung scanning were used at that time, but noninvasive high-resolution thin-slice CT scanning will now give equally good information.

We were able to get long-term follow-up in 10 patients who
had had lobectomy and/or plication. Three were alive and well 8 years, one 6 years, and one 2 years after surgery. Two died 4 years after surgery from surgery not related to the chest. Three others died 4, 6, and 8 years after the LVRS, but the cause could not be ascertained. These data suggest that long-term survival and benefit may be obtained even 8 years after lung volume reduction operations.

Age was not a contraindication to surgery and two patients who did particularly well, both symptomatically and in the number of improved test results, were in an older age group, namely 69 years and 54 years of age.

It is difficult to define the most useful tests for postoperative long-term monitoring. We did very extensive testing involving spirometry, ventilation-perfusion relations, and mechanics of breathing as well as blood lactate, all at rest and with exercise. There was considerable variability in the changes in pulmonary function. Some patients showed improvement in the results of ventilatory tests, improvement in diffusing capacity, and blood gases. Other patients might show improvement in the mechanics of breathing. However, in the same patient there might be improvement in one aspect of pulmonary function and either no change or even a worsening in some other aspect of function. I have noted similar variability in some of the recently published reports.

Modern concepts result in an appreciation that it will never be possible to fully predict what will happen in all cases from a treatment. The body functions are nonlinear, chaotic, and dynamic, and one cannot even conceive of all the factors that might have an effect on a particular medical situation. The lungs are an expression of fractal geometry where there are twists and turns and folding and branching. We will never know enough to allow an accurate prediction that will have real validity in the individual patient. There will always be uncertainties and it should not be discouraging if logic is not always fulfilled by results.

In Toronto there is a continued interest in LVRS and it is encouraging that, even after a hiatus of 30 years, this treatment for some cases of emphysema is now receiving an enthusiastic acceptance.

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## Descending necrotizing mediastinitis

H Esteva

*Chest* 1997;111:529-

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