Duplicate Publication

Editor’s Note:

The following two letters are self-explanatory. Instead of punishing these authors by banning them from publishing in CHEST, I offered the alternative of publishing their names. They have chosen this option, which alerts future authors that transgressions may become public knowledge. The editors of Thorax have chosen their own method of dealing with this problem, and it will be described in an editorial in that journal. Duplicate publication will not be tolerated by medical journal editors.

A. Jay Block, MD, FCCP
Editor-in-Chief
CHEST

Dear Dr. Girault:

The editors of Thorax have called my attention to a possible duplicate publication in Thorax (1997; 52:690–696) and CHEST (1997; 111:1639–1648). You are the author of both publications, and they were submitted to both journals at about the same time.

I have recently written about duplicate publication and the punishments for it in CHEST (1999; 114:951). I enclose a copy of this publication. I also enclose a copy of the “Instructions to Authors” checklist, published every month in our journal, that you should have read. It states, “No part of the work has been published previously other than in abstract form and the manuscript has not been submitted concurrently to another journal.” You have clearly violated this requirement.

I have read both manuscripts and I see that you stressed the comparison of assist-control ventilation (ACV) to spontaneous breathing in the Thorax paper and there was no pressure support ventilation (PSV) group. In the CHEST paper, you stressed the comparison between the two types of nasal intermittent positive pressure ventilation (ACV and PSV). Nevertheless, the patients studied were identical, as were the measurements made. To be completely ethical, you should have published one paper with all the data in it. This is an example of “salami” publication, where one study is sliced up to make multiple publications. You did not even note in either bibliography that the other study was pending publication in the other journal, suggesting that you wished to hide this fact.

I will give you a chance to explain your actions. The enclosed editorial suggests various punishments for duplicate publication. I do not think these manuscripts are exactly duplicate, but you have not been forthright with the various editors of these journals. An apology is certainly in order.

A. Jay Block, MD, FCCP
Editor-in-Chief, CHEST
Emeritus Professor of Medicine
University of Florida
Gainesville, FL

Dear Dr. Block:

We the authors acknowledge the receipt of your letter regarding the possible duplicate publication in Thorax (1997; 52:690–696) and CHEST (1997; 111:1639–1648). You will also find enclosed, herewith, a copy of our letter in reply to the questions raised by Dr. John Britton and Dr. Alan Knox.

First, we sincerely apologize for any misunderstanding that may have been caused by the above mentioned publications. It was never our intention to produce a duplicate publication because in fact, the original objectives of the two manuscripts were different. In the article published in Thorax, we compared the physiological effects and the influence on respiratory comfort of assist-control ventilation (ACV) during noninvasive ventilation (NIV) to those of spontaneous breathing. In contrast, in the article published in CHEST, we attempted to determine the most appropriate mode of ventilation during NIV by comparing the two modes used (ACV and pressure support ventilation). This seemed to us to regroup different physiological aspects regarding a vast field of research which constitutes NIV.

Nevertheless, since receiving both letters from Thorax and CHEST, we have read the various articles in the literature regarding redundant publications. We are now conscious that the Editors could interpret our publications as redundant. However, as the material was similar, but the objectives and the statistical method different, therefore, it did not obviously appear to us to specifically inform either journal. As you also pointed out in your letter, with hindsight, our greatest error was certainly to not submit both manuscripts to the same Editor.

We would like to stress that we are particularly disconcerted and deeply upset by this correspondence. We ask you to accept our sincere apology for any inconvenience caused by the publication of these articles. We would also like to reassure you that this error was completely unintentional. We remain at your disposal for further information.

Christophe Girault, MD
Medical Intensive Care Unit
Charles Nicolle University Hospital
Rouen, France

Correspondence to: Christophe Girault, MD, Service de Réanimation Médicale, Hôpital Charles Nicolle, Centre Hospitalier et Universitaire de Rouen, 76031 Rouen cedex, France
Tuberculosis and Sarcoidosis

To the Editor:

Patients presenting features of both sarcoidosis and tuberculosis (whether or not in causative relationship) can be quite puzzling to the pulmonary physician, as was pointed out in a recent issue of CHEST by Wong and colleagues (August 1998) and, in a splendid accompanying editorial, by Dr. Banghman.

The authors and the readers of CHEST may be interested in a paper published in the European Journal of Internal Medicine, in which we described the case histories of three patients with “overlap features” of both disorders, and in whom extensive diagnostic workup (though not including polymerase chain reaction search for mycobacterial DNA) and response to therapy did not allow unequivocal classification into diagnosis of either sarcoidosis or tuberculosis. In this paper, we also refer to several other reports in the literature on “concomitant” sarcoidosis and tuberculosis that were not quoted by Wong and colleagues, but which may be of interest in this debate.

Finally, although I fully acknowledge the diagnosis of sarcoidosis at the second admission of Wong’s patient, would not the authors expect a search for mycobacterial DNA in pulmonary tissue, 13 months after a documented tuberculous infection, to be positive in every case?

Marc Noppen, MD, PhD, FCCP
Academic Hospital AZ-VUB
Brussels, Belgium

Correspondence to: Marc M. P. Noppen, MD, FCCP, Respiratory Division, Academic Hospital AZ-VUB, 101 Laarbeeklaan, Brussels 1090, Belgium; email: pnennm@az.vub.ac.be

REFERENCES
2 Banghman RP. Can tuberculosis cause sarcoidosis (editorial)? Chest 1998; 114:363–364

The Fourth Heart Sound

To the Editor:

I thank Dr. Noppen for his interest in our case report. I am sure the three cases described in his paper published in the European Journal of Internal Medicine would be of great interest. However, I did not have the chance to read this article because it is not available on MEDLINE.

I agree that diagnosis of sarcoidosis can be very difficult, especially in areas where tuberculosis has a high prevalence. Usually, one can confidently diagnose sarcoidosis only after extensive mycobacteriologic studies have failed to reveal the presence of Mycobacterium tuberculosis. Alternatively, sarcoidosis can be diagnosed in a patient who has received a course of antituberculous treatment with no improvement, as in our reported case.

Concerning the detection of mycobacterial DNA by polymerase chain reaction (PCR) in patients with treated or inactive tuberculosis, it has been well reported that PCR can be positive in the respiratory specimens from patients with treated or inactive tuberculosis. A study conducted in our unit and recently published may also give some data on this issue. In the study, among 82 patients not suffering from active pulmonary tuberculosis, bronchial aspirate for PCR was positive in 22 cases. Among these 22 PCR-positive cases, 12 had evidence of previous tuberculosis whereas in the PCR-negative counterparts, 22 of 60 had evidence of previous tuberculosis. This showed that PCR can be positive in some, but not all, cases of treated tuberculosis.

In our reported case, the patient had well-documented tuberculous lymphadenitis but not pulmonary tuberculosis. As stated in the case report, we believed that the patient had sarcoidosis involvement of the lungs and other organs.

Chi Fong Wong, MBBS, FCCP
Grantham Hospital
Aberdeen, Hong Kong

REFERENCES

The Fourth Heart Sound

To the Editor:

As always, I enjoyed Dr. Spodick’s recent comments. It has been 25 years since we thrust and parried in the literature over the audibility of the fourth heart sound. It remains my contention that differentiation of a split S1 from an S2-S4 is difficult unless the S4 is palpated as a presystolic outward movement of the chest wall on the upstroke of a left ventricular heave. A split S1 is common at any age and an S2 in an older person usually indicates disease, e.g., left ventricular hypertrophy or fibrosis. As stated in my editorial, a recorded S4 does not necessarily equate with an audible S4. Dr. Spodick seems to agree. Indeed, that is the main thesis of my editorial.

In an era when only dinosaurs like Dr. Spodick and I care enough about an S4 to belabor its recognition, it isheartening that we now seem to agree on the following: (1) a recorded S4 does not necessarily equate with audibility; (2) an S4 virtually always indicates heart disease; (3) most filters used in recording systems pass inaudible low-frequency vibrations; (4) an audible S4 is loud and palpable; and (5) I concur that blinded auscultators provide
the best chance for objectivity, but clearly their accuracy depends on their bedside experience.

Now as for the S₃ gallop…

Robert J. Adolph, MD
Professor Emeritus of Medicine
University of Cincinnati
Cincinnati, OH

Correspondence to: Robert J. Adolph, MD, Room 3468, Medical Science Building, University of Cincinnati, Cincinnati, OH 45267

REFERENCE

1 Spodick DH. Audibility of fourth heart sound. Chest 1999; 115:1218–1219

**Infective Exacerbations of COPD**

To the Editor:

We read with great interest the article by Eller and colleagues (June 1998) in which they showed that there was a correlation between deterioration of lung function (as measured with FEV₁ levels) and the bacteria isolated from patients with infective exacerbations of COPD, in that a significantly higher rate of Enterobacteriaceae and *Pseudomonas aeruginosa* were isolated from patients with more severe disease. We would like to extend these observations with our own experience.

We retrospectively reviewed the records of COPD patients with severe exacerbation hospitalized according to the American Thoracic Society standards between January 1995 and June 1997. We excluded the following individuals: patients with evidence of other disease that can cause chronic cough and sputum production (bronchiectasis, asthma) or radiographically apparent pneumonia; those who were unable to produce any sputum or whose sputum was not of good quality (>25 leukocytes, <10 epithelial cells/100× magnified field); or those who were admitted to the ICU and a nonprotected endotracheal suction material was obtained. COPD patients who had a nosocomial respiratory infection or whose sputum was obtained after antibiotics were given were also not evaluated. Data for 232 patients (35 were female and 194 were male; mean age, 64.4 ± 9.0 years) who matched these criteria are presented here. These were all current or ex-smokers with a mean duration of COPD of 13.1 ± 7.5 years and a mean FEV₁ level of 1.14 ± 0.69 L.

The microbiologic examination of the sputum revealed normal flora in 108 (46.6%) of the patients. The most frequently isolated bacteria were as follows: *Streptococcus pneumoniae* (37 cases, 15.9%), *Haemophilus influenzae* (26 cases, 11.2%), Enterobacteriaceae (20 cases, 8.6%), *Pseudomonas aeruginosa* (16 cases, 6.9%), *Moraxella catarrhalis* (14 cases, 6.0%), Acinetobacter spp (3 cases, 1.3%). The cases were evaluated in two groups: group A included the cases whose sputum examination revealed Enterobacteriaceae, *Pseudomonas aeruginosa* or Acinetobacter spp (n = 39). Group B (n = 193) contained all others whose sputum examination revealed either normal flora or any other bacteria not included in group A.

There was no difference between the two groups as to the following: age; duration of COPD; levels of PaCO₂, hematocrit, and serum creatinine; number of exacerbations during the previous year; and use of inhaled steroids. The two groups differed, however, in that group A cases had lower levels of FEV₁ (0.86 ± 0.45 vs 1.23 ± 0.76 L, p < 0.01) and PaO₂ (51.7 ± 17.7 vs 56.7 ± 17.1 mm Hg, p = 0.053), higher rate of hypalbuminemia (albumin levels <3.5 g/dL in 9.5% vs 27.9%, p < 0.05), and a history of more frequent hospitalizations (5.7 ± 3.2 vs 4.0 ± 2.8, p < 0.01). Group A bacteria were also more frequently isolated from those who were using H₂-antagonists than from those who were not (29.3% vs 14.1%, p < 0.05).

As in the study by Eller and colleagues, we also observed a trend for increasing rates of isolation of group A bacteria with decreasing FEV₁ levels (Fig 1). A similar trend was seen with decreasing levels of PaO₂ (data not shown).

The only other study on the microbiologic findings of severe COPD exacerbations showed similar results. These data support and extend the observations by Eller and colleagues that Enterobacteriaceae and *P. aeruginosa* are isolated more frequently than other organisms and should be taken into account in infective exacerbations of patients with severe disease, as reflected by low levels of FEV₁, deeper hypoxemia, malnutrition, and more frequent hospitalizations.

Abdullah Saginer, MD
Nurcan Okbay, MD
Ipek Unsal, MD
Nesrin Colpan, MD
Ege University Medical School
Izmir, Turkey

Correspondence to: Abdullah Saginer, MD, Ege Universitesi Tip Fakültesi, Göğüs Hast. A.D., Bornova, Izmir 35100, Turkey; e-mail: asaginer@med.ege.edu.tr

REFERENCES


CHEST / 115 / 5 / MAY, 1999 1481
Ipratropium Bromide in Acute Asthma

Small Beneficial Effects?

To the Editor:

We read with great interest the article by Lanes and colleagues1 regarding the effect of adding ipratropium bromide (IB) to salbutamol in treating acute asthma (August 1998). In this paper, the authors conducted a pooled analysis of three previously published randomized double-blind clinical trials. The studies enrolled 1,064 patients with acute asthma. The authors found that patients receiving IB and salbutamol therapy had greater improvements in FEV1 on average than patients receiving salbutamol alone, but this difference was small (<10%). Also, they found that the small improvement in lung function was associated with a nonsignificant reduced risk of hospitalization (relative risk [RR], 0.80; 95% confidence interval, 0.61 to 1.06).

In an unpublished meta-analysis about IB in acute adult asthma (nine studies, 1,416 subjects [G. Rodrigo, MD, C. Rodrigo, MD; unpublished data; 1998]), we obtained a pooled effect size equivalent to 10.3% (4.3 to 16.3%) greater in pulmonary function compared with the control group (effect size, 0.13; range, 0.03 to 0.23). Additionally, this meta-analysis revealed that the addition of IB to β2-agonists significantly reduced the admission rate of 36% (odds ratio = 0.64, 0.45 to 0.92). However, we must emphasize that all of these beneficial effects have been obtained with the utilization of “unrealistic” therapeutic protocols consisting of small doses of IB (eg, in the pooled analysis, a combination of nebulized 2.5 mg salbutamol plus 0.5 mg IB administered only at baseline, and in the US study, at 45 min). Nevertheless, there is substantial evidence that patients with acute asthma respond to increasing doses of bromidolators,2 and that this thought can be applied to IB. In fact, in a preliminary study, we have shown an important additional therapeutic effect (25% increase in peak expiratory flow rate) from adding IB to salbutamol, when both drugs are administered in high and cumulative doses (0.24 mg of IB every 30 min) through metered-dose inhaler and spacer,3 with minimal adverse effects. Finally, a recently published systematic review4 about the use of anticholinergics added to β2-agonists for treating acute childhood and adolescent asthma indicates that a multiple dose anticholinergic protocol reduces the hospital admission rate by 30% (RR, 0.72; 95% confidence interval 0.53 to 0.99) and improves pulmonary function significantly (effect size, −0.66; range, −0.95 to −0.37).

In conclusion, data highlight the need for new and more “realistic” clinical trials examining the effect of higher and cumulative doses of IB.

Gustavo Rodrigo, MD
Carlos Rodrigo, MD
Asociación Española 1a de Socorros Mutuos
Montevideo, Uruguay

Correspondence to: Carlos Rodrigo, MD, Centro de Terapia Intensiva, Asociación Española 1a de Socorros Mutuos, Bulvar Artigas 1465, Montevideo 11300, Uruguay; e-mail: gurodrig@cardia.rev.edu.uy

REFERENCES
2 Rodrigo C, Rodrigo G. Therapeutic response patterns to high and cumulative doses of salbutamol in acute severe asthma. Chest 1998; 113:593–598
3 Rodrigo C, Rodrigo G. Treatment of acute asthma: administration of high doses of salbutamol and ipratropium bromide delivered by metered dose inhaler with a spacer (Volumatic) [abstract]. Am J Respir Crit Care Med 1996; 153:A60
4 Plotnick LH, Ducharme FM. Should inhaled anticholinergics be added to β2-agonists for treating acute childhood and adolescent asthma? A systematic review. BMJ 1998; 317:971–977

Continuous Gastric Mucosal Capnometry Is Affected by Enteral Nutrition

Potential for Misinterpretation of Tissue Oxygenation

To the Editor:

The editorial by Fink (September 1998)1 provided excellent guidelines on the use of tissue capnometry in critical care. In addition to the mechanisms by which carbon dioxide can enter the intestinal lumen that are listed in his editorial, it has been shown that carbon dioxide can be formed during enteral feeding.2 As an example of this frequent problem in clinical practice, we describe a patient in whom mucosal hypercapnia was induced by enteral feeding. This case is a 68-year-old man who underwent elective correction of an infrarenal abdominal aortic aneurysm. He subsequently developed septic shock and acute renal failure. Continuous intra gastric capnometry (Tonocap; Datex-Ohmeda; Helsinki, Finland) was begun due to suspected gut hyperperfusion. Severe mucosal hypercapnia and an increase in the gap between end-tidal carbon dioxide and mucosal carbon dioxide was observed (Fig 1). This was associated with the administration of standard enteral feeding formula (Pre-Nutrison; N.V. Nutricia; Zoetermeer, Holland). During the period described, enteral nutrition was first infused at a rate of 25 mL/h, followed later with a higher rate of 38 mL/h (Fig 1). Furthermore, lactacemia and systemic acidosis were absent during the periods of mucosal hypercapnia. Discontinuation of enteral feeding led to normalization of the mucosal carbon dioxide level.

We agree with Dr. Fink that intraluminal capnometry has an important role in cases where oxygen delivery in the microvasculature is the problem. The new technique of continuous monitoring of intraluminal carbon dioxide should be very advantageous in these unstable patients under resuscitation. Continuous monitoring allows an easy and rapid method for observing the patient’s response to therapeutic interventions by detecting short-term changes in mucosal carbon dioxide level.3 This type of real-time measurement is not possible using intermittent measurements; this is especially true in the case of capnometry, which is relatively time consuming to perform. Following the initial stabilization, however, early enteral feeding is recommended in critically ill patients.4 In these patients already on enteral feeding, the monitoring of the intragastric carbon dioxide level will have a limited role in the detection of gut dysoxia.

Tero I. Ala-Kokko, MD, PhD, EDIC
Jouko Laurila, MD
Department of Anesthesiology
Oulu University Hospital
Oulu, Finland

1482 Communications to the Editor
To the Editor:

I read with interest the correspondence between Crausman and Al-Bilbeisi1 and the Editor-In-Chief of CHEST2 on “Code 99—Who’s Watching?”, and I would like to add an international perspective.

India is a developing country where a lot of change in medicine is economy driven. Cardiopulmonary resuscitation (CPR) occupies the bottom rung of the ladder of priorities. In a city like Mumbai with a population of 10 to 12 million, only one hospital has an organized CPR team.3 This is in a city where the average daily coronary artery bypass surgery numbers vary between 20 and 40. It is probable that the total number of CPR teams in our country (with a population of about 1 billion) is less than 10 or 20. In this scenario, the only way to get any sort of success in CPR is with active consultants and ICU staff participation.

Despite an intense, widely disseminated training program at the initiation of our CPR service, the skills of non-ICU medical and nursing staff remain poor. A rapid turnover adds to the problems, and one occasionally still faces a chaotic situation where a young, newly appointed doctor performs an intracardiac injection when venous access is available and freely uses steroids, bicarbonate, and so on. The more experienced nurses stand by helplessly watching the protocols being ignored. All this gets rectified only after a consultant arrives.

Despite all of these problems, and after a dismal start, we have finally achieved results compatible with international data.3 These results have been sustained; in the last year, the CPR team in our 320-bed hospital received 193 CPR calls. Eighteen were candidates who should have had advanced do not resuscitate orders, and CPR was rapidly terminated. Of the 175 who received CPR, 132 survived the initial CPR, but only 44 of these were finally discharged home. At the present time, these results could not have been obtained without the active participation of the ICU team and consultants.

Unfortunately, the prevailing medical culture among non-ICU consultants is that CPR is a job for juniors and nurses. In this depressing setting, there is no way in which CPR services will be created and evolve without someone senior watching.

Farhad N. Kapadia, MD
Consultant Physician and Intensivist
Hinduja National Hospital and Medical Center,
Mumbai, India

REFERENCES

Code 99—An International Perspective

To the Editor:

I read with interest the correspondence between Crausman and Al-Bilbeisi1 and the Editor-In-Chief of CHEST2 on “Code 99—Who’s Watching?”, and I would like to add an international perspective.

India is a developing country where a lot of change in medicine is economy driven. Cardiopulmonary resuscitation (CPR) occupies the bottom rung of the ladder of priorities. In a city like Mumbai with a population of 10 to 12 million, only one hospital has an organized CPR team.3 This is in a city where the average daily coronary artery bypass surgery numbers vary between 20 and 40. It is probable that the total number of CPR teams in our country (with a population of about 1 billion) is less than 10 or 20. In this scenario, the only way to get any sort of success in CPR is with active consultants and ICU staff participation.

Despite an intense, widely disseminated training program at the initiation of our CPR service, the skills of non-ICU medical and nursing staff remain poor. A rapid turnover adds to the problems, and one occasionally still faces a chaotic situation where a young, newly appointed doctor performs an intracardiac injection when venous access is available and freely uses steroids, bicarbonate, and so on. The more experienced nurses stand by helplessly watching the protocols being ignored. All this gets rectified only after a consultant arrives.

Despite all of these problems, and after a dismal start, we have finally achieved results compatible with international data.3 These results have been sustained; in the last year, the CPR team in our 320-bed hospital received 193 CPR calls. Eighteen were candidates who should have had advanced do not resuscitate orders, and CPR was rapidly terminated. Of the 175 who received CPR, 132 survived the initial CPR, but only 44 of these were finally discharged home. At the present time, these results could not have been obtained without the active participation of the ICU team and consultants.

Unfortunately, the prevailing medical culture among non-ICU consultants is that CPR is a job for juniors and nurses. In this depressing setting, there is no way in which CPR services will be created and evolve without someone senior watching.

Farhad N. Kapadia, MD
Consultant Physician and Intensivist
Hinduja National Hospital and Medical Center,
Mumbai, India

REFERENCES

To the Editor:

I read with interest the correspondence between Crausman and Al-Bilbeisi1 and the Editor-In-Chief of CHEST2 on “Code 99—Who’s Watching?”, and I would like to add an international perspective.

India is a developing country where a lot of change in medicine is economy driven. Cardiopulmonary resuscitation (CPR) occupies the bottom rung of the ladder of priorities. In a city like Mumbai with a population of 10 to 12 million, only one hospital has an organized CPR team.3 This is in a city where the average daily coronary artery bypass surgery numbers vary between 20 and 40. It is probable that the total number of CPR teams in our country (with a population of about 1 billion) is less than 10 or 20. In this scenario, the only way to get any sort of success in CPR is with active consultants and ICU staff participation.

Despite an intense, widely disseminated training program at the initiation of our CPR service, the skills of non-ICU medical and nursing staff remain poor. A rapid turnover adds to the problems, and one occasionally still faces a chaotic situation where a young, newly appointed doctor performs an intracardiac injection when venous access is available and freely uses steroids, bicarbonate, and so on. The more experienced nurses stand by helplessly watching the protocols being ignored. All this gets rectified only after a consultant arrives.

Despite all of these problems, and after a dismal start, we have finally achieved results compatible with international data.3 These results have been sustained; in the last year, the CPR team in our 320-bed hospital received 193 CPR calls. Eighteen were candidates who should have had advanced do not resuscitate orders, and CPR was rapidly terminated. Of the 175 who received CPR, 132 survived the initial CPR, but only 44 of these were finally discharged home. At the present time, these results could not have been obtained without the active participation of the ICU team and consultants.

Unfortunately, the prevailing medical culture among non-ICU consultants is that CPR is a job for juniors and nurses. In this depressing setting, there is no way in which CPR services will be created and evolve without someone senior watching.

Farhad N. Kapadia, MD
Consultant Physician and Intensivist
Hinduja National Hospital and Medical Center,
Mumbai, India

REFERENCES
Brachial Artery Puncture for Blood Sampling

To the Editor:

Recently, Okeson and Wulbrecht (September 1998)\(^1\) reported their experience with 6,185 brachial arterial punctures for arterial blood sampling, demonstrating a very low complication rate. Hematoma formation occurred in 4 patients (0.06%), immediate pain or paresthesias in 66 patients (1.1%), and delayed pain or paresthesias in 57 patients (0.9%). Of the few subjects who experienced immediate pain in the distribution of the median nerve, only one had discomfort that lasted > 48 h, and full resolution occurred within 2 months of the procedure. Only one subject with delayed pain or paresthesias had persistent symptoms, and again full resolution occurred within 4 months. Although the complication rates related to brachial artery puncture are low in this study, there is a risk of progressive median nerve damage, as demonstrated by the following example.

A 63-year-old man with chronic respiratory failure secondary to combined COPD and obstructive sleep apnea was admitted to the hospital in April 1997 with acute decompensation following a respiratory tract infection. Arterial blood gas sampling was successfully completed using the right brachial artery without immediate pain or paresthesias. Within 24 h, however, the patient complained of paresthesias in the median nerve distribution of the right hand. No hematoma was detected at the site of the brachial artery puncture, and spontaneous recovery over a period of weeks to months was predicted. However, the sensory symptoms persisted, and there was associated progressive muscle weakness and wasting in the distribution of the right median nerve.

Electromyographic and nerve conduction studies performed 6 months after the incident confirmed a severe lesion of the right median nerve with loss of 99% of axons. The sensory nerve action potential was absent, and the distal motor potential was < 2% of predicted. Eighteen months after the incident, the patient remains symptomatic with no clinical or neurophysiologic recovery of right median nerve function. A neurologic consultation suggested acute infarction of the median nerve as the most likely mechanism of nerve injury in this particular situation.

Hospital residency programs sometimes include recommendations to avoid sampling from end arteries, including the brachial artery, in order to avoid potential vascular complications. However, the risk of complications from brachial artery puncture, including median nerve damage, is poorly documented.\(^2,3\) Although the study by Okeson and Wulbrecht\(^1\) is somewhat reassuring, the case reported here demonstrates that significant morbidity can, in fact, occur from this seemingly innocuous medical procedure.

David J. Barnes, MB, BS, FCCP
Royal Prince Alfred Hospital
Sydney, Australia

Correspondence to: David J. Barnes, MB, BS, FCCP, Department of Respiratory Medicine, Royal Prince Alfred Hospital, 100 Carillon Avenue, Newtown, New South Wales 2042, Australia; e-mail: dbarnes@nsw.bigpond.net.au

REFERENCES

1 Okeson GC, Wulbrecht PH. The safety of brachial artery puncture for arterial blood sampling. Chest 1998; 114:748–751

Metalloptysis Expulsion of Wire Stent Fragments

To the Editor:

We would like to report an incidence of breakage and expulsion of a wire mesh stent (Wallstent; Schneider; Minneapolis, MN) in a patient who required multiple stents for idiopathic tracheobronchomalacia.

A 69-year-old man with tracheobronchomalacia had two Wallstents inserted in the trachea and left main bronchus (LMB) in February 1996. He did well for approximately 1 ½ years and returned with increasing shortness of breath. Flexible bronchoscopy in October 1997 revealed granulomas involving the stents, and the lower end of the tracheal stent was overhanging the LMB. Both of the granulomas and the lower portion of the tracheal stent were ablated with Nd-YAG laser photoresection. A Rusch Y stent was inserted in the trachea after balloon dilation of the existing tracheal stent. The Rusch Y stent had to be subsequently removed in December 1997 because of frequent mucous plugging.

The patient returned 3 months later with an “exacerbation of asthma,” of 1 week’s duration and had coughed up two wire stent fragments (Fig 1). Bronchoscopy at the time revealed wires...

Correspondence to: Gyman C. Okeson, MD, FCCP, Medical Director, Pulmonary Function Laboratory, Scott and White Clinic, 2401 South 31st Street, Temple, TX 76508

REFERENCE

1 Okeson GC, Wulbrecht PH. The safety of brachial artery puncture for arterial blood sampling. Chest 1998; 114:748–751
protruding from an intact tracheal stent (Fig 2) and dynamic collapse of the posterior wall of the trachea. No intervention was done at that time. The patient returned again in June 1998 after having coughed up two more pieces of wire stent fragments while having swallowed the third one.

During both of these episodes, the patient experienced hemoptysis and was fearful of losing the stent and recurrence of his symptoms.

Flexible bronchoscopy is being increasingly used to insert self-expandable metallic stents for management of large airway obstruction.1–3 Though complications of Wallstents like stent migration, granuloma formation, infection, and stent expulsion have been reported, to our knowledge, this is the first incident of spontaneous breakage and expulsion of wire stent fragments. We speculate that damage to the stent occurred during subsequent manipulation through the stent, and spontaneous breakage occurred by the dynamic maneuvers, like coughing.

We highlight this occurrence as a reminder that Wallstents are delicate, and one needs to be careful about forcible manipulation through the stent. It also raises concern about structural manipulation of the Wallstent, for example, by laser ablation. If such manipulations are mandatory, then patients should be warned of later expulsion of wires to decrease their anxiety. This is also likely to lead to loss of stent function, which may require insertion of a second stent through the first stent.

Anjana Aggarwal, MD
Department of Internal Medicine
Ashok Dasgupta, MBBS
Atul C. Mehta, MBBS, FCCP
Department of Pulmonary and Critical Care Medicine
The Cleveland Clinic Foundation
Cleveland, OH

Correspondence to: Atul C. Mehta, MBBS, FCCP, Department of Pulmonary/Critical Care Medicine, A-90, Cleveland Clinic Foundation, 9500 Euclid Avenue, Cleveland, OH 44195

REFERENCES

Errata
In the November 1998 issue, the article “Sympathetic Nervous Response Relative to the Adenosine Triphosphate Supply-Demand Imbalance During Exercise Is Augmented in Patients With Heart Failure” (CHEST 1998; 114:1295–1300), by Kinugawa and colleagues, requires a correction. Rest and peak plasma hypoxanthine (HX) levels, depicted in Table 2 (p 1297), should be increased by a factor of 10. The ratio of change in plasma norepinephrine to change in plasma hypoxanthine ($D_{\text{plasma NE}}/D_{\text{plasma HX}}$), shown on the y axis of Figure 1 (p 1298), should range from 0 to 600 instead of 0 to 6000.

In the February 1999 issue, the article “Asbestosis: A Marker for the Increased Risk of Lung Cancer Among Workers Exposed to Asbestos,” (CHEST 1999; 115:536–549) by Weiss, contained an error. On page 540, in the second paragraph, the sentence beginning “In the age group 40 to 69 years” should read “In the latency group 40 to 69 years.”

In the January 1999 issue, the article “Pharmacokinetics of Rifampin Under Fasting Conditions, With Food, and With Antacids” (CHEST 1999; 115:12–18), by Peloquin and colleagues, requires a correction. Rifampin (RIF) is stable in human serum for > 6 h at room temperature, not > 24 h. Rifampin begins to decay by 24 h. This correction does not change the results in any manner.