is emphasized in the following statement: "Secondary and tertiary asthma treatment should address several basic nonpharmacologic principles. First, asthma triggers, including irritants and allergens, should be avoided."

However, complete avoidance of all substances that might trigger an asthma attack is not always possible, particularly for persons who are highly allergic to numerous common substances. While allergic asthmatics can do many things to avoid or reduce exposures in their own homes, they cannot always control exposures at work or school, in social situations, or in public places. It is only prudent to be prepared for inadvertent exposures by having appropriate medications available or by taking appropriate medications preventively to reduce chances of having an attack or to reduce the severity of an attack if one occurs.

To use Dr. Unger's example, an asthmatic who is allergic to cat dander obviously should not have a cat for a pet. While the asthmatic can ask friends and relatives who own cats to put their cats outdoors during visits, cat dander is still present in the carpet or on furniture, and it may not be practical or possible to avoid entering every house where a cat has been present.

A goal of asthma therapy is to allow persons with asthma to participate in normal activities to their fullest abilities. Restrictions on activities or exposures known to trigger attacks are imperative. However, an overly restrictive life-style calculated to avoid every possible trigger may not be desirable or even possible.

William C. Bailey, M.D., F.C.C.P.,
Division of Pulmonary and Critical Care Medicine,
University of Alabama School of Medicine,
Birmingham

Needle in the Heart

To the Editor:

An intracardiac needle can be removed surgically. This may not be necessary if the patient is asymptomatic. We briefly report two cases involving intracardiac needles.

Case 1: An intracardiac needle was first discovered in a 49-year-old woman undergoing a radiologic examination of her stomach. We saw her 8 years later, and found no organic heart disease. The needle was located in the apex of the left ventricle, as evidenced by a chest x-ray film (Fig 1), an echocardiogram, and a computed tomographic scan. Her condition is stable, with the needle in the same position 13 years after its fortuitous discovery.

Case 2: A 74-year-old woman suddenly complained of acute chest pain. A loud pericardial friction rub was heard, and the patient died within minutes. Autopsy revealed a 2-cm-long tear in the right atrium caused by a sewing needle, with cardiac tamponade. A second needle was found in the liver.

Intracardiac needles should be removed surgically in symptomatic patients. However, they may be surprisingly well tolerated over prolonged periods of time.

Frans Hagemeijer, M.D., Ph.D., F.C.C.P, and Rob van Mechelen. M.D., Ph.D.,
Department of Cardiology,
Sint Franciscus Gasthuis,
Rotterdam, The Netherlands

References

2. Schechter DC, Gilbert L. Injuries of the heart and great vessels due to pins and needles. Thorax 1969; 24:246-53

Chirping Rales in Bird-Fancier's Lung

To the Editor:

Widespread and profuse high-pitched end-inspiratory wheezes accompanied by rales were encountered in two of three patients with bird-fancier's lung. This distinctive feature of extrinsic allergic alveolitis (EAA) is rarely adverted to; it does not appear to occur with diffuse interstitial lung disease due to other causes. No attenuation of end-inspiratory flow was evident on close scrutiny of the spiromgrams of the affected individuals.

Lâënnec described a late inspiratory wheeze as "le cri d'un petit oiseau." In 1982, the term inspiratory "squawk" was coined for this finding. In a phonocardiographic study of pulmonary fibrosis of varying etiologies, the pattern of late, high-pitched inspiratory wheezes initiated by rales was observed only among those with EAA. In "stork"-ing an explanation for this distinctive finding, the authors "rhea"-soned that a partially obstructing bronchiolitic component to the alveolitis might be responsible, and conjectured that late opening of distal airways, particularly when partially

Figure 1. Details of posteroanterior and lateral chest x-ray films showing an intracardiac needle.
obstructed, accounted for the timing, small caliber and high transmural pressures, for the character.

I advocate the term "chirping rales" for this singular finding. (One must caution against the possibility of confusing the term "chirping rales" with "chirps," the latter designating a well-known and regrettably common disorder of amorous songbirds.) More euphonious than "squawk," the onomatopoetic "chirp" more closely emulates the actual sound; it has the sanction of prior usage and encompasses both auscultatory components. Chirping rales of EAA are distinguished from the findings in bronchiolitis obliterans by later occurrence during inspiration, diffuseness, clinical setting, and absence of airway obstruction. It is a melancholy experience for those who would popularize a sign, when they see no way to substantiate its existence by means of three, four, or six imaging techniques. Having no other recourse available, I will now, therefore, humbly propose my own thoughts (the author wishes to acknowledge with deepest gratitude the "Modest" contribution of J. Swift, D.D., to this portion of the manuscript), which I hope will not be liable to the least objection: A manually operated, sonically activated cylindrical auscultating tool (SCAT), convenient for chest scanning, may be helpful in suggesting EAA in patients with diffuse interstitial lung disease. Its many other applications in this field I omit, being studious of brevity. I profess, in the sincerity of my heart, that I have not the least personal interest in endeavoring to promote the use of stethoscope: being the practitioner of a cognitive specialty, my emoluments are too small to finance their manufacture, and my wife is in similar exigous circumstances.

Jerome M. Reich, M.D.,
Bess Kaiser Medical Center,
Portland, Oregon

REFERENCES

4 Forcager P. Crackles and wheezes. Lancet 1967; 508: 203-05

Undetected Superior Laryngeal Nerve Injury Presenting as Noncardiogenic Pulmonary Edema

To the Editor:

Upper airway obstruction is occasionally associated with noncardiogenic pulmonary edema. Recently we had the opportunity to see a patient in whom noncardiogenic pulmonary edema was associated with superior laryngeal nerve injury.

Four months before admission, the patient, a 16-year-old boy, was admitted to our unit with severe cranial trauma after a car accident. A computed tomographic scan showed a right parietal hemorrhagic contusion and brain edema. Intracranial hypertension was controlled with hyperventilation and barbiturates. Mechanical ventilation and orotracheal intubation were used for 10 and 13 days, respectively. The patient was extubated without problem and was discharged 7 days later with hemiparesis on the left side.

Four days before admission the patient began smoking 20 cigarettes daily. He experienced increasing cough and was brought to the emergency room in obvious respiratory distress and stridor. Otorhachal intubation was performed, and mechanical ventilation was initiated. Light vocal cord edema was appreciated at intubation. Chest radiography showed interstitial edema in the right lung. A Swan-Ganz catheter was inserted. The pulmonary artery pressure was 23/12 mm Hg, and the pulmonary capillary pressure was 7 mm Hg. Gram stain of a sputum specimen showed no polymorphonuclear leukocytes and no organisms. Cultures of blood and bronchial secretions were done, and therapy with antibiotics and bronchodilators was initiated.

Twenty-four hours later the chest radiograph was normal, and arterial blood gas values while breathing O2 at a rate of 5 L/min were as follows: PaO2, 163 mm Hg; PaCO2, 43/L; mm Hg; pH, 7.39; bicarbonate, 20 mmol/L. The patient was extubated. An otolaryngologist performed a direct laryngoscopic examination and found minimal vocal cord edema. Bronchoscopy disclosed no abnormalities in the trachea and bronchial tree.

Twenty-four hours later the patient had another episode of stridor and respiratory distress. Otorhachal intubation and mechanical ventilation were again initiated. The pulmonary artery pressure was 34/15 mm Hg, and the pulmonary capillary pressure was 12 mm Hg. A chest x-ray film showed bilateral alveolar edema (Fig 1). No organisms grew in blood and sputum cultures.

Twenty-four hours later the patient was again extubated. A chest x-ray film was normal. Laryngoscopic examination was again performed, and bilateral cord paralysis was observed at phonation, suggesting superior laryngeal nerve paralysis. The patient was discharged 2 days later.

Superior laryngeal nerve injury can be associated with minimal symptoms. In our patient it was undetected until a laryngoscopic examination was performed at phonation. Both episodes of pulmonary edema were associated with normal pulmonary capillary pressures, and chest radiographs were normal after 24 h. Initially, the possibility of pneumonia was entertained, but the rapid chest radiographic resolution, the absence of fever and leukocytosis, and the negative sputum and blood cultures excluded that possibility. The vocal cord changes associated with increasing smoking in the previous 4 days seemed to be the precipitating factor for the upper airway obstruction. The superior laryngeal nerve is a branch of the vagus nerve supplying the motor fibers to the cricothyroid muscle.

Figure 1. Chest x-ray film obtained on the second day shows bilateral interstitial edema.