single vessel disease (including two with normal coronary arteriograms) and 19 with multivessel disease. None had involvement of the left main artery, nine triple vessel and ten double vessel disease. Fifteen of the latter completed submaximal stress tests, which were negative in five. During a mean follow-up period of 11.4 months, eight (42 percent) had a recurrent cardiac event including one who immediately elected to undergo bypass surgery. There were two deaths, three recurrent myocardial infarctions and two with unstable angina. Four of the latter five patients underwent subsequent aortocoronary bypass surgery. Thus, five of 19 patients (26 percent) with multivessel disease had a major event (myocardial infarction or death) within one year of their first myocardial infarction.

I am not advocating sending all clinically symptom-free patients (after their first myocardial infarction who harbor multivessel disease) for aortocoronary bypass surgery. However, most would agree that bypass surgery, when performed technically well, does improve myocardial blood flow as demonstrated by exercise testing, radionuclide techniques and follow-up angiographic study of the bypass grafts. If standards for available bypass surgery are such that the mortality is negligible (< 0.5 percent), low incidence of perioperative infarction (< 5 percent) and reliable longterm patency of the saphenous vein grafts this option should certainly be considered in this low risk group, especially in those with critical stenosis in the remaining unoccluded vessels threatening large areas of viable myocardium.

In conclusion, I believe patients who survive their first myocardial infarction need coronary arteriography regardless of symptom status. The presence or absence of multivessel involvement and subsets within this group all have prognostic and therapeutic implications. The same rationale should apply as in evaluating patients with cancer. The pathology must be determined (presence or absence of arteriosclerosis), pathophysiology (obstructed and/or vasospastic vessels) and stage (location and distribution of coronary stenosis). Only then can rational therapeutic objectives be achieved, be it by medical and/or surgical means.

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REFERENCES

1 Rahimtoola SH. Coronary arteriography in asymptomatic patients after myocardial infarction. Chest 1980; 77:53-57
3 Mautner RK, Phillips JH. Coronary arteriography after the first myocardial infarction: clinical, angiographic and prospective observations. Cath Cardiovsc Diag (in press)

Routine Use of Biofeedback in Weaning Patients from Mechanical Ventilation

To the Editor:

We would like to compliment Corson et al. (Chest 76:543-45, 1979) on their concise report on the use of biofeedback in weaning paralyzed patients from ventilators. Although not approaching it as formally as they, we have been utilizing biofeedback routinely in weaning patients from mechanical ventilation. Patients who fail to attain objective weaning criteria are initially placed on IMV at parameters sufficient to maintain normal arterial blood gas levels. Patients with neuromuscular diseases, longstanding COPD, chest wall defects, or blunted central drives are first optimally prepared nutritionally. Electrolytes (including magnesium and phosphorus, and especially bicarbonate) and the patient’s hemodynamic status are stabilized. The physician, nurse, and respiratory therapist then sit down with the patient to assure the individual that he will be receiving adequate ventilation with the mandatory machine breaths and that in between machine-cycled breaths, he will have to work on improving his own spontaneous ventilation. He is told what is expected of him and assured that he will be able to meet our (and his) expectations. The occasional patient who has developed respiratory muscle-diaphragm discoordination must be re instructed in how to breathe. Initially during these weaning trials, one member of the critical care team is continuously present, giving reassurance and positive reinforcement.

We have found the Bourns Bear I adult volume ventilator the easiest ventilator to use on these difficult patients. In the upper left hand corner of the ventilator panel is a numerical display which simultaneously indicates the patient’s exhaled tidal volume (TV) and respiratory rate. The patient is simply turned to face the ventilator and watches his “numbers” on the display panel. As in the article by Corson et al, the goal is to increase the TV and to slow the spontaneous respiratory rate. The Bennett MA-1 or MA-2 and the Foregger can also be used in this way, but only allow the patient to follow his expired TV. We have not employed devices that display only the minute ventilation (MV) because the patient would not be able to determine whether his rate or his TV should be altered. The rare patient who cannot be successfully weaned in the ICU is considered for admission into our home care ventilator protocol. An occasional patient is then able to eventually wean himself off the ventilator when he returns to the comfort and security of his own home environment (one out of eight patients). Our methodology offers an advantage in that no additional equipment needs to be adapted for biofeedback since the patient’s ventilator serves as the bio feedback apparatus. These techniques can also be successfully applied when using the challenge (T-piece) method of weaning by keeping the ventilator “on line.”

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To the Editor:

Recently we examined a 34-year-old woman with signs of Klippel-Feil syndrome: short webbed neck, scoliosis and low hair line. On auscultation of the heart an apical late systolic murmur, grade 2/6, was heard. Echocardiographic M-mode and two-dimensional examinations revealed signs of prolapse of the mitral valve—both leaflets. Udoshi and associates1 demonstrated a high frequency of mitral valve prolapse in
subjects with thoracic skeletal abnormalities.

In Klippel-Feil syndrome there are many skeletal abnormalities and the deformity is believed to result from disordered progression of the formation of cervical centra and discs in the fourth gestational week. The primordia of the mitral valve undergo differentiation to their final form at the same time that the vertebral column and the thoracic cage are beginning their chondrification and ossification. The finding of a mitral valve prolapse in this syndrome may confirm the assumption that there may be connective tissue defect during embryonic development of the bony thoracic cage and atrioventricular valves.

The rate of association of congenital heart diseases with Klippel-Feil syndrome was reported to be 4.2 percent. The most commonly associated congenital cardiovascular defects are ventricular septal defect, atrial septal defect and transposition of the great vessels. Surprisingly, however, prolapse of the mitral valve has not been reported in this syndrome. We believe that the reason for this is that current knowledge about the syndrome of mitral valve prolapse and the diagnostic tools, mainly echocardiography for its diagnosis, were not available at the time the studies on the association of congenital heart diseases with Klippel-Feil syndrome were carried out.

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References

Indoor Air and Tuberculosis

To the Editor:

In the December supplement of Chest, devoted entirely to the International Conference on Tuberculosis and including five articles on control, there is not even a passing reference to environmental control in the form of indoor dissection. This in spite of the fact that environmental control will become more important as buildings are made tighter to save energy. Even the federal government and the National Academy of Science are becoming aware of indoor air quality. By the government’s own statistics (National Health Survey) respiratory infections outnumber all other acute conditions, including accidents. Most of them are not tuberculous, but still, when people are finally recognizing that indoor air is the medium through which airborne infection occurs and when workers in the field of tuberculosis should remember that their disease sparked this recognition, it is too bad that they do not even suggest the possibility of environmental control. Above all, this is important in connection with tuberculosis in institutions. Otherwise, the supplement was great!

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Desquamative Interstitial Pneumonitis

The Intra-alveolar Cells are Macrophages

To the Editor:

In the article, "Desquamative interstitial pneumonitis, characterization of free intra-alveolar cells," in the April, 1980 issue of Chest (77:552-554) Fromm et al stated that the intra-alveolar cells in desquamative interstitial pneumonitis (DIP) are macrophages. Their conclusion was based on the electron microscopic appearance of the cells and their phagocytic and bactericidal activity in vitro. We had studied tissue sections from four patients with DIP and our findings corroborate those of Fromm et al. Ultrastructurally, the cells lining the alveoli were distinct from those in the lumina of the alveoli. The lining cells exhibited surface microvilli and intracytoplasmic osmiophilic lamellar bodies characteristic of type II pneumocytes. The intra-alveolar cells had the features of macrophages with an occasional cell containing phagocytosed lamellar bodies. The macrophage nature of the intra-alveolar cells was also indicated by their reactivity with antibodies to lysozyme by immunofluorescence and immunoperoxidase staining. The cells lining the alveoli, type II pneumocytes, did not react with antilysozyme sera.

The tissue sections were also studied by immunoperoxidase staining for surfactant specific apoprotein. The reactivity of the antibody with alveolar lining cells further confirmed them as type II pneumocytes. The antibody did not react with desquamated intra-alveolar cells, thus indicating that they were not type II pneumocytes.

Our morphologic studies and analysis of specific marker protein corroborate the morphologic and functional studies of Fromm et al, and it is safe to state that the desquamated intra-alveolar cells in DIP are macrophages whereas the cuboidal cells lining the alveoli are reactive type II pneumocytes.

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References