other cardiomyopathies are underway.

**Other Potential Uses**

Nifedipine may be useful in treatment of systemic hypertension and also in hypertensive emergencies. These agents may be the most efficacious and logical choices as adjunctive drugs for systemic hypertension, especially when patients are being treated with the medications for other indications. Further comparative studies using calcium antagonists for treatment of systemic hypertension are needed. Verapamil and nifedipine have been shown to reduce pulmonary pressure in patients with pulmonary hypertension. In patients with chronic air flow obstruction and acute respiratory failure, nifedipine vasodilates pulmonary vessels constricted by hypoxia without causing a reduction in arterial oxygenation. Although nifedipine and verapamil do not have intrinsic bronchodilator properties in unstimulated airways, they may blunt the development of airway obstruction following exercise in asthmatic patients. We hope the role of these drugs in asthma and acute and chronic pulmonary hypertension will be better defined in the near future.

The vasodilating properties of the drug may be useful in treatment of congestive heart failure, aortic and mitral insufficiency, although careful monitoring of ventricular performance is required, in view of the potentially negative inotropic effect. Verapamil and nifedipine may interfere with platelet function. The role of these drugs in sudden cardiac death is not yet defined. Non-cardiopulmonary uses such as for treatment of cerebral vasospasm, gastrointestinal, obstetric and gynecologic disorders requires further investigation.

**Conclusions**

This new class of drug has some clear advantages over currently used medications for treatment of specific cardiovascular disorders. One of the major advantages may be that these drugs are useful in the presence of chronic obstructive lung disease or in situations where beta-adrenergic blocking agents cannot be used. Calcium channel blockers may allow more effective treatment of paroxysmal atrial tachycardia and angina pectoris in patients with chronic obstructive lung disease. The agents may produce fewer unpleasant side effects than beta-adrenergic blocking drugs or nitrates, although this needs further confirmation. Initial euphoria must be tempered by the proof of substantial improvement in morbidity and mortality. Nevertheless, the use of this new class of drugs is a welcome addition to our armamentarium.

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**High Frequency Ventilation**

A Passing Fancy?

Over the last two years, interest in high frequency ventilation (HFV) has increased considerably. The technique is not really new, having been described in Sweden some 15 years ago. Originally developed to aid ventilation during bronchoscopy and laryngeal procedures, HFV now has much broader implications.

All modes of HFV are not the same. The different systems of delivery have distinct characteristics. High frequency jet ventilation, described in this issue by Schuster and colleagues (see page 682), delivers each breath through a narrow orifice such as a 14- or 16-gauge intracath. The accelerated flow, operating on the Bernoulli principle, entrains humidified gas, which also enhances tidal volume. The usual frequency is 100-900/min. High frequency positive pressure ventilation is similar, but operates without entrainment. Both methods require a circuit with minimal compressible volume to minimize the loss of gas in the circuit.

High frequency oscillation differs greatly from those two methods. Usually, a piston pumps a set volume of gas into the airway, first in one direction, then in the other. There is no bulk flow. Oxygen is added at a rate consistent with metabolic demands; an absorber or a bypass circuit removes carbon dioxide. This system has so far performed best at rates between 900 and 2,000/min.

The principle effects of HFV compared to the conventional ventilation are reductions of peak inflation and mean airway pressure. Possible benefits include minimal pulmonary barotrauma and less interference with cardiac output.

Several theories attempting to explain the mechanism of HFV have been advanced. However, precise information is still scarce. The rapid rate, small volume, and high flow make difficult the tracing of gas distribution.

Although heralded as a major technologic break-
through and the ventilatory technique of the future, little evidence exists to support such claims; an example of this is the report in this issue of *Chest.* HFV was apparently partially successful, but the data do not preclude that continuing conventional mechanical support could have also improved gas exchange. The authors correctly point out that compared to conventional ventilation, HFV may have actually developed an even higher mean airway pressure (PAW). Other work would indicate that PAW and not PEEP alone is a major determinant of oxygenation. Additionally, the MA-1 ventilator will stall at the high inflation pressures of 70 cm H₂O described in the article. No doubt the reduced rate of flow, as well as the compressible circuit, contributed to the reduction in the patient's actual tidal volume compared to that developed by the machine. The result would be the difficulty described with CO₂ elimination.

The future of HFV mandates identification of patients most likely to benefit from this new modality. Controlled studies of homogeneous clinical and experimental populations need to be undertaken. Further progress will be limited until we gain a clearer understanding of the actual mechanisms of HFV. Because the ventilatory profile varies with the delivery, certain diseases may better lend themselves to particular systems of delivery.

Rarely has any new medical modality not produced its own set of complications; HFV will not be any different. The identification and assessment of these factors will provide the eventual role of HFV.

HFV is at center stage now; however, let us temper our enthusiasm. More data are needed before the method can stand the test of time and eventually take its place in respiratory therapy.

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