Monitoring Intrapulmonary Hemorrhage In Goodpasture's Syndrome*

Michael Addleman, M.D.; Alexander S. Logan, M.D.; and Ronald F. Grossman, M.D., F.C.C.P.

Diffusing capacity of carbon monoxide (Dco) was measured frequently in a patient with recurrent intrapulmonary hemorrhage secondary to Goodpasture's syndrome. This simple test was found to be a sensitive and useful indicator of the presence or absence of recurrent intrapulmonary hemorrhage. As this is now the major cause of mortality in this disease, we recommend that frequent measurements of the Dco be an important part of its management.

The diffusing capacity of carbon monoxide in the lung (Dco) is a function of both intravascular and extravascular hemoglobin. In the presence of intrapulmonary hemorrhage, the stagnant pool of extravasated blood will pick up carbon monoxide and an elevation of the Dco will be found. This phenomenon has been shown to occur in patients with Goodpasture's syndrome. We describe the usefulness and ease with which we monitored recurrent intrapulmonary hemorrhage in a patient with this disease.

**CASE REPORT**

The patient, a previously healthy 25-year-old man, was admitted to the hospital with acute renal failure associated with dyspnea and hemoptysis. Renal biopsy showed crescentic glomerulonephritis and immunofluorescent staining characteristic of Goodpasture's syndrome. His course was monitored with daily hemoglobin measurements, frequent chest x-ray examinations, and Dco measurements of the Dco.

The Dco was measured in the usual way and was corrected for hemoglobin and total lung volume. The results are shown in Figure 1. The patient was noted to be dyspneic on admission, the 20th and the 44th hospital day. Small amounts of hemoptysis were noted on days 1 to 5 and moderate amounts on day 16 with decreasing amounts until day 25. Moderate hemoptysis again occurred on day 44 with decreasing amounts over the following four days. Episodes of dyspnea and hemoptysis correspond reasonably well to elevations in the Dco indicating that these rises did represent hemorrhage. Interestingly, it appears from the episode on day 44 that the rise in the Dco preceded any clinical signs of hemorrhage.

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*From the Department of Medicine, Mount Sinai Hospital and University of Toronto, Toronto, Canada.

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**Figure 1.** The clinical course of the patient is shown throughout the hospital admission. The diffusing capacity of carbon monoxide (Dco) is seen graphically and three peaks are apparent—on admission, day 21 and day 44. Note that the increases in Dco correspond well to episodes of dyspnea (D) and subsequent hemoptysis. In the last episode, the increase in Dco precedes any clinical event. The therapies instituted in the patient's management are also shown.
**Discussion**

This patient with Goodpasture's syndrome was observed to have dyspnea on three separate occasions: on admission, on day 20 and on day 44. These episodes corresponded well to marked elevation of the DCO at the time. Because of the complicating factors of repeated transfusions and fluid shifts from dialysis, the hemoglobin measurements were not a useful indicator of hemorrhage.

Frequent chest films were also inadequate as a monitor of hemorrhage in this patient because of persistence of infiltrates after clinical improvement, confusion with pulmonary edema and variations in roentgenographic technique.

The DCO provides a simple objective measurement of the presence of intrapulmonary hemorrhage. In our patient, in one instance, the DCO had begun to increase before the onset of symptoms. Had daily measurements been done, we may have found this always to be the case. Only rarely was the patient too ill to be taken to the pulmonary function laboratory. On these occasions, the clinical diagnosis of pulmonary hemorrhage was obvious either because of the presence of hemoptysis or because the DCO had been seen to be increasing in the preceding days. We cannot draw conclusions from our data about the amount of hemorrhage from the degree of elevation of the DCO; however, theoretically the rise in the DCO should parallel the amount of blood lost through hemorrhage.

Of interest is a follow-up DCO done approximately one year after discharge, which showed a value of 80 percent of predicted. This may indicate slight lung damage due to episodic pulmonary hemorrhage and subsequent fibrosis.

Renal failure associated with Goodpasture's syndrome has been treated successfully with dialysis and kidney transplantation. Thus, pulmonary hemorrhage represents the immediate threat to life in this disease. With time, this threat tends to abate and aggressive supportive therapy is crucial. Many different therapies have been investigated in the treatment of Goodpasture's syndrome. Monitoring the DCO also provides an easy method for evaluating the effects of these treatments on pulmonary hemorrhage.

In conclusion, we believe that the DCO is the best test for monitoring these patients for intrapulmonary hemorrhage because of its safety, ease and sensitivity.

**References**

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**Underdrive Suppression of the Sinus Rhythm in Man**

József Tenczer, M.D.; László Littmann, M.D.; and Miklós Rohde, M.D.

This report demonstrates unusual responses of the sinus rhythm to atrial pacing. The sinus rhythm failed to become manifest when the heart was driven at a rate slower than the inherent sinus rate. Sinus rhythm returned only after termination of underdrive pacing with the recovery time longer than twice the cycle length of the control sinus rhythm. The largest difference between underdrive and sinus cycle lengths measured 600 msec. To the best of our knowledge, underdrive suppression of the sinus rhythm has not been previously reported in man.

In recent years, abnormalities in sinus node function have received widespread attention. Experimental and clinical studies have shown that overdrive suppression is a valuable aid in assessing sinus node automaticity. In contrast to underdrive suppression, there are no data available on the occurrence of underdrive suppression of the sinus rhythm in man. Based on observations by Rosenblueth, Loeb et al were the first, in 1979, to systematically study the effects of underdrive pacing on automatic rhythms in dogs. They have shown that supraventricular escape rhythms in the canine heart could be suppressed by electrical pacing at a rate slower than the rate of the escape rhythm. We demonstrate herein underdrive suppression of the sinus rhythm in man.

**Case Report**

A 72-year-old man was admitted because of anterior myocardial infarction. During the second week of hospitalization, recurrent episodes of drug-resistant ventricular tachycardia occurred. After withholding antiarrhythmic therapy for three days, invasive electrophysiologic study was performed including evaluation of the sinus node function. With atrial cycle lengths of 480 msec or longer, maximal sinus node recovery time was 2,600 msec. Atrial pacing at a cycle length of 480 msec or less induced an atrial escape rhythm containing one-five beats. It was clearly demonstrated that the atrial escape rhythm prolonged onset of the sinus activity. These observations suggested that the slow atrial escape rhythm was capable of suppressing the faster sinus rhythm.

To test this interesting phenomenon, slow atrial pacing was initiated at a rate slower than the basic sinus rate during spontaneous sinus rhythm and during the post-overdrive pacing period. It was found that slow atrial pacing prevented emergence of sinus beats. Sinus rhythm returned only after termination of slow stimulation with the recovery time longer than twice the cycle length of spontaneous sinus rhythm. The sinus rate returned to control levels without secondary pauses. Illustrative tracings in Figures 1 and 2 demonstrate these findings. Figure 1, line 1, shows an example of overdrive depression of sinus nodeautomaticity at a driving cycle length of 460 msec. Sinus rhythm returned after one atrial and one junctional escape beat. When slow atrial pacing at a cycle length of 1,600 msec was initiated in the postoverdrive pacing period, the sinus rhythm emerged only after cessation of slow stimulation (Fig 1, lines 2-4). The phenomenon of sinus node underdrive suppression was also observed during sinus rhythm. Figure 2 illustrates the effects of intermittent slow atrial stimulation of different duration.

*From the 3rd Department of Medicine, Semmelweis University Medical School, Budapest, Hungary.*