that the pulmonary vascular resistance is potentially always reversible, while in congenital left-to-right cardiac shunts, the increased pulmonary vascular resistance and resultant pulmonary hypertension (Eisenmenger's syndrome) may progress, if untreated, to an irreversible state. The data available concerning pulmonary hypertension in COPD reveal that there is both a reversible and irreversible type. The former is most common in the bronchitic type of COPD; and, therefore, this patient population responds best to oxygen therapy. The evidence suggests that the bronchitic type acts more like other adult acquired forms of pulmonary hypertension, with the pulmonary arterioles developing muscular hypertrophy which is reversible, and that it does not progress to an irreversible state, as in congenital forms of pulmonary hypertension which develop irreversible pulmonary arteriole intimal thickening. The irreversible type of pulmonary hypertension in COPD occurs mostly with emphysema, where the pulmonary vascular bed is irreversibly lost (atrophic). This type of COPD patient (emphysematous) interestingly does not develop right heart failure until late (preterminally) in their disease course. This probably explains the paradox of why some patients with clinical right heart failure and cor pulmonale (presumably a bad prognostic sign) have a better survival rate when treated with oxygen, which reverses their pulmonary hypertension, than some patients treated with oxygen without cor pulmonale (presumably a better prognostic sign) who nevertheless have irreversibly destroyed vascular beds just before their first bout of frank right heart failure.

Therefore, I do not feel that “withholding oxygen therapy” (not prescribing it), even in patients with moderate chronic hypoxia (PaO₂ of 40 to 60 mm Hg) but without bouts of right heart failure, is causing the pulmonary arterioles to become irreversibly diseased. That is, the etiology of COPD is not oxygen deprivation; but, rather, oxygen deprivation (hypoxia) is a result of severe COPD, and it should be treated with long-term oxygen administration when the following symptoms become clinically manifested: right heart failure; exercise limitation secondary to hypoxia; and repeated hospitalization necessary to treat hypoxia. This appears to be the clinical lesson also confirmed in the current paper by Drs. Stewart, Hood, and Block.

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Limitations of Electrocardiograms

While it should be quite obvious that the electrocardiogram per se does not always provide a clear indication of the etiology of abnormalities of rhythm or conduction, almost since its inception, the field of electrocardiography has been ripe with such examples, to wit: upper, middle, and lower atrioventricular nodal rhythms; coronary sinus rhythms; left atrial rhythms; right atrial rhythms; and various forms of bundle-branch block. The articles in this issue of Chest by Suárez de Lezo et al (see page 548) and LaCorte et al (see page 575) provide a valuable service by demonstrating once again the limitations of the ECG in understanding the mechanisms and genesis of various abnormalities in cardiac rhythm and conduction.

Consideration of the right bundle-branch block pattern in a recorded ECG well illustrates these limitations. The diagnoses of incomplete and complete right bundle-branch block on the basis of ECG patterns have been accepted for quite a long time. However, as emphasized by Suárez de Lezo et al, the appearance of the right bundle-branch block pattern in an ECG does not mean necessarily that there is complete block in the main right bundle branch. In fact, it now has been demonstrated that not all right bundle-branch block patterns, complete or incomplete, represent block in the right bundle branch. For instance, Moore et al demonstrated electrophysiologically in the canine heart that the electrocardiographic pattern of incomplete right bundle-branch block was not related in any way to an abnormality in the right bundle branch, but rather was caused by inherited focal hypertrophy of the right ventricle. The extrapolation of these data to man is obvious. Further, a series of studies performed in patients during open-heart surgery has elucidated the genesis of the right bundle-branch block pattern associated with repair of ventricular septal defects of various types. Gelband et al initially demonstrated that the right bundle-branch block pattern was associated with the creation of a right ventriculotomy per se and not with the repair of the ventricular septal defect. Krongrad et al later delineated the course of the right bundle branch to its Purkinje fiber-ventricular muscle junction following creation of a right ventriculotomy and the appearance of a right bundle-branch block pattern in the ECG, thereby demonstrating that despite this ECG pattern, the main right bundle branch was intact. More recently, Krongrad et al extended these studies and demonstrated that the location of the vertical right ventriculotomy may interrupt one of the peripheral branches of the right bundle branch,
resulting in the appearance of a right bundle-branch block pattern in the recorded ECG.

It can be appreciated that these sorts of data are critical in understanding and interpreting the ECG. For instance, it has been suggested that the appearance of the right bundle-branch block pattern with left axis deviation following repair of a ventricular septal defect represents a considerable risk in the development of complete heart block and perhaps even sudden death.8,9 More recently, the clinical significance of the right bundle-branch block pattern with left axis deviation has been called into question.9,10 Clearly, in patients who manifest a right bundle-branch block pattern with left axis deviation following repair of ventricular septal defect, it is not possible simply from an ECG to be certain of the nature of the atroventricular conduction abnormality. As the papers by Suárez de Lezo et al and LaCorte et al remind us, we must be careful not to overinterpret the ECG. Further, we must resist the temptation to accept logical and apparently reasonable interpretations of ECGs until a clear understanding of the underlying pathophysiologic events has been provided by careful experimental studies.

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REFERENCES
1 Massing GK, James TN: Conduction and block in the right bundle-branch--real and imagined. Circulation 45:1, 1972
7 Wolff GS, Rowland TW, Ellison RC: Surgically induced right bundle branch block with left anterior hemiblock; an ominous sign in postoperative tetralogy of Fallot.

Circulation 46:587, 1972

Classification of Diffuse Intrapulmonary Hemorrhage

Diffuse hemorrhage from multiple pulmonary bleeding sites has been classified on the basis of clinical criteria into (1) Goodpasture's syndrome, when both pulmonary hemorrhage and glomerulonephritis are present, and (2) idiopathic pulmonary hemosiderosis (IPH), when only pulmonary manifestations are present. In recent years the clinical category, "Goodpasture's syndrome," has been replaced by the immunopathologic category, "anti-basement membrane antibody-induced glomerulonephritis and pulmonary hemorrhage" (ABMA-induced GN and PH).1 The methods for immunopathologic evaluation of tissue and serum have recently been reviewed by Wilson and Dixon.2 It is the purpose of this discussion to suggest that three categories of diffuse pulmonary hemorrhage can now be distinguished on the basis of immunopathologic studies: one in which ABMA is present, a second in which immune complexes are found, and a third in which neither can be demonstrated.

The presence of ABMA in practically all cases of Goodpasture's syndrome is now well established. Gamma-globulin antibody against basement-membrane antigen can be demonstrated in a linear pattern along the capillary basement membrane of both the lung and kidney by immunofluorescence microscopy.1 Alteration of the basement membrane induced by antibody is thought to be the cause of the diffuse pulmonary hemorrhage. Complement is found in a similar location in two-thirds of such cases. Immune complexes are not present. In 90 percent of such patients, ABMA can be demonstrated in the serum as well.1

The evidence that immune complexes cause diffuse pulmonary hemorrhage is less certain. The occurrence of diffuse pulmonary hemorrhage in a few cases of both immune complex nephritis5,6 and lupus erythematosus4,5 suggests that rarely, immune complexes lead to pulmonary hemorrhage. A search for immune complexes in the lung utilizing immunofluorescence microscopy has not yet been reported in such cases. However, in one case of