Effects of pirbuterol and sodium nitroprusside on pulmonary haemodynamics in hypoxic cor pulmonale

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important in the pulmonary circuit when there is no estimate of flow through it. D W GREEN

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**The authors reply below.—Ed, BMJ.**

SIR,—Dr D W Green is, of course, technically correct. In any segment of the circulation the resistance is defined as the drop in pressure across the segment divided by the rate of volume flow through it. Thus the pulmonary vascular resistance is expressed as the difference between the mean pulmonary arterial pressure and the mean left atrial pressure divided by the cardiac output.

In stating this, however, Dr Green has ignored the controversy that has existed for the past 20 years or more as to whether measurement of pulmonary arterial pressure accurately reflects mean left atrial pressure in patients with severe chronic bronchitis and emphysema, such as those studied in our paper. Fishman states: "Pulmonary capillary wedge pressure does not reliably reflect left atrial pressure in patients with severe chronic bronchitis: fact or fancy." Clin Sci 1969;37: 155-8.

It is for these reasons that we chose to measure total pulmonary vascular resistance, avoiding the effect of a possible error in the measurement of resistance. As pulmonary arterial pressure in our patients was high and we have assumed that left atrial pressure was low (as all of our patients had well preserved left ventricular function) this is not dissimilar to the approach in calculating systemic vascular resistance, where the right atrial pressure is not usually considered. It was for similar reasons that total pulmonary vascular resistance was measured in our study as "wedge pressure" minus arterial oxygen pressure. It is possible that in the future, the largest single series of measurements of pulmonary haemodynamics in patients with obstructive lung disease does not mention measurements of pulmonary capillary wedge pressure. We cannot therefore agree with Dr Green that "most workers would argue that the formula used in this study was wrong." 3

Furthermore, it is not valid to assume that long term administration of pirbuterol had beneficial effects, when no estimate is made of cardiac output. Thus a reduction purely in systolic pressure does not seem clinically important in the pulmonary arterial pressure and an increase in right ventricular ejection fraction. Cardiac output was not measured in order to reduce the number of invasive measurements made during the chronic study. As the increase in right ventricular ejection fraction paralleled the increase in cardiac output during the acute study and as a consistent increase in right ventricular ejection fraction occurred in the chronic study, we have assumed that cardiac output would also increase.

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Recognising placental steroid sulphatase deficiency

SIR,—The determination of the steroid sulphatase activity in leucocyte homografts (23 July, p 293) certainly meets the demand for an "easy diagnostic method for X-linked ichthyosis suitable for routine clinical biochemistry laboratories" put forward in the leading article by Dr R A Harkness and others (7 July, p 221). The authors found a considerable variation of the steroid sulphatase activity among obligate carriers of the steroid sulphatase deficiency trait. This limits the ability of their simple method1 to recognise heterozygotes, which can be needed urgently in cases of low urinary steroid excretion during pregnancy as this may be caused by steroid sulphatase deficiency.

The results of the steroid sulphatase activity determination proved the heterozygosity for this trait in only 14 out of 18 carriers (figure). The much greater variation in the carrier group than in the healthy normal group is not likely to be due to technical differences, as all assays were carried out in the same period by one person.

Scientially, these results are most interesting as they indicate that no complete escape from X-chromosomal inactivation of the steroid sulphatase locus seems to occur in leucocytes. This, however, differs from the results given with cultured skin fibroblasts reported by Shapiro et al for four obligate carriers2 and reported by Müller et al for five obligate carriers,3 which suggested complete escape of inactivation. The reported steroid sulphatase activity in leucocytes and in lymphocytes4 of normal persons and further work of Shapiro's group on cultured fibroblasts from carriers of the steroid sulphatase deficiency trait5 indicate, though, that a differential expression of the steroid sulphatase locus.

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SIR,—I would like to take issue with the methodology used by Dr W MacNee and others (22 October, p 1169) for measurement of pulmonary vascular resistance and the effects of variables of the two drugs pirbuterol and sodium nitroprusside.

The authors state: "Left atrial pressure is not always easy to assess from pulmonary artery wedge pressure in these patients." The formula for measuring pulmonary vascular resistance was then derived, as it includes no estimate of the pressure difference across the pulmonary circuit. Whether one believes in the concept of "resistance" in the pulmonary circuit or "impedance," I am sure that most workers would argue that the formula used in this study was wrong.1

Furthermore, it is not valid to assume that long term administration of pirbuterol had beneficial effects, when no estimate is made of cardiac output. Thus a reduction purely in systolic pressure does not seem clinically


