Abnormal distribution of pulmonary blood flow in aortic valve disease

Relation between pulmonary function and chest radiograph

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Wasted ventilatory volume (V_D) and its ratio to tidal volume (V_D/V_T) were measured at rest and during exertion in 17 patients with aortic valve disease. We considered V_D/V_T to indicate abnormal ventilation: perfusion relations if it did not decrease on exertion, or if the exercising value was greater than 40 per cent. Plain chest radiographs were independently examined for evidence of diversion of pulmonary blood to the upper lobes. There was significant agreement ($p < 0.05$) between radiographic and pulmonary function estimations of abnormality. This suggests that the raised pulmonary venous pressure associated with left ventricular failure creates an abnormal pattern of blood flow through the lung, which is responsible for causing inadequate perfusion with respect to ventilation.

Patients with pulmonary venous hypertension often show a characteristic pattern in the plain chest radiograph with increased upper lobe veins (Lavender *et al.*, 1962), suggesting increased upper lobe blood flow. Indeed, such diversion of blood flow to the upper lobes has been shown to occur (West, Dollery, and Heard, 1964; Dawson, Kaneko, and McGregor, 1965). In addition, patients with aortic valve disease who complain of breathlessness (presumably an indication of raised pulmonary venous pressure) are unable to decrease their wasted ventilatory volume:tidal volume ratio normally on exertion (Goodenday, *in press*). As this abnormality did not appear to be related to a decreased tidal volume, it suggested abnormal perfusion to some areas of the lung, creating an abnormal ventilation:perfusion relation. Since both the chest radiograph and this aspect of pulmonary function point to maldistribution of pulmonary blood flow, we decided to see whether or not both signs of abnormality were consistently present or absent in the same patients.

The use of the term 'wasted ventilatory volume' may be confusing at first. Comroe *et al.* (1962), however, suggest that the so-called physiological dead space is 'neither

'physiologic' nor 'space', nor 'dead''. J. A. Nadel therefore has suggested the term 'wasted ventilatory volume' for this measurement (Nadel, Gold, and Burgess, 1968), and we agree that it is more accurately descriptive. We shall continue, however, to use the standard symbols for the term.

Methods

Seventeen patients with aortic valve disease of varying severity underwent routine pulmonary function tests performed by one of the authors. Tests were done before and after replacement of the aortic valve in 3 patients. There was no evidence of non-cardiac lung disease. Wasted ventilatory volume (V_D) and its ratio to tidal volume (V_D/V_T) were measured in the usual manner (Comroe *et al.*, 1962), with the patient seated at rest and then pedalling a cycle ergometer at a low load. We considered V_D/V_T to be abnormal if it did not decrease on exertion, or if the exercising value was greater than 40 per cent (Nadel *et al.*, 1968).

Standard chest radiographs of these patients, taken on inspiration, upright and at rest, near the time of the pulmonary function tests, were examined independently by another author, who determined whether or not diversion of pulmonary blood to the upper lobes was present. An example of upper lobe blood diversion in one of these patients is shown in Fig. 1-3.

We used the χ^2 test (Weinberg and Schumaker, 1962) for statistical significance between the variables.

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Results

Findings are presented in the Table. There was agreement between the chest radiograph and pulmonary function evidence of over-all maldistribution of pulmonary blood flow in 14 of 20 studies. Of the remaining 6, 1 (Case 12) had bilateral pleural effusions which may have affected either the wasted ventilatory volume or interpretation of the chest radiograph, and another (Case 16), though classified as having normal V_D/V_T in the statistical evaluation, actually had a borderline exercise value (1% change). Even with these patients, however, agreement between the tests was statistically significant ($p < 0.05$).

Discussion

The measurement of V_D/V_T depends on the difference between mixed expired carbon dioxide tension ($P_{E\text{CO}_2}$) and mean alveolar carbon dioxide tension ($P_{A\text{CO}_2}$) of perfused alveoli (usually estimated by arterial $P_{a\text{CO}_2}$), and the tidal volume. It is therefore a reflection of the relation between ventilation and perfusion throughout the lung. Hyperventilation, by lowering $P_{E\text{CO}_2}$, causes an increase in V_D/V_T , as does a small tidal volume. This was not a factor in these patients. Normal ventilation to underperfused areas of the lung, however, would create relative hyperventilation of those areas, and again increase V_D/V_T . An abnormally high V_D/V_T on exertion, if ventilation is normal, suggests maldistribution of pulmonary blood flow.

In pulmonary venous hypertension, blood flow to the apices of the lung is increased (West *et al.*, 1964; Dawson *et al.*, 1965). At first, one would think this would create better matching of ventilation and perfusion in the upright posture. Several studies have now shown, however, that V_D/V_T is increased in heart failure (Cosby *et al.*, 1957; Saunders, 1966; Goodenday, in press). In our exercised patients, this was not due to hyperventilation. If, then, distribution of ventilation is not grossly abnormal, there must be areas of decreased blood flow.

This abnormal distribution of flow is probably not as simple as a mechanical model would imply. That is, we cannot say that the apices of the lung are ventilated and perfused in a 1:1 ratio, whereas the bases are ventilated but not perfused. Certainly there are areas of patchy interstitial oedema throughout the lung (though mostly in the dependent portions) which may have blood flow decreased more, in proportion, than ventilation. West (1968) suggests that raising interstitial pressure (as might occur in pulmonary venous

TABLE Wasted ventilatory volume (V_D), its ratio to tidal volume (V_D/V_T), and presence (+) or absence (−) of pulmonary blood diversion in the chest radiograph

Case No.	Rest		Exercise		Pulmonary blood diversion
	V_D (ml.)	V_D/V_T (%)	V_D (ml.)	V_D/V_T (%)	
1 { pre-op.	270	39	350	47	+
1 { post-op.	125	28	202	24	−
2	235	35	508	39	+
3	354	30	181	18	−
4	561	58	374	58	+
5 { pre-op.	190	36	279	39	+
5 { post-op.	356	52	354	40	−
6	415	47	405	33	+
7 { pre-op.	224	39	205	24	+
7 { post-op.	142	22	366	29	+
8	232	33	138	25	−
9	253	21	150	12	−
10	254	32	255	30	−
11	349	24	585	28	+
12	327	31	197	19	+
13	82	19	177	23	+
14	412	54	513	47	−
15	248	42	303	34	+
16	184	37	239	36	+
17	409	45	408	45	+

FIG. 1 Case 5, before operation. Upper arrows point to upper lobe vessels which are larger than corresponding lower lobe vessel opposite arrow.

FIG. 2 Enlargement of left upper zone of Fig. 1.

hypertension) causes increased pulmonary vascular resistance.

In our patients, we found that there was some correlation between an abnormally high wasted ventilatory volume:tidal volume ratio on exertion, and an abnormal diversion of blood from the bases to the upper lobes in chest radiographs. This suggests that this mechanism of blood diversion is one cause of the abnormal ventilation:perfusion relations found in pulmonary venous hypertension.

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FIG. 3 Same patient 6 months after aortic valve replacement by Starr-Edwards prosthesis. Same area as Fig. 2. Slight reduction in size of upper lobe vessel opposite arrow.

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