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Research Article

PULMONARY FUNCTION STUDIES IN MITRAL STENOSIS BEFORE AND AFTER COMMISSUROTOMY

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Decreased arterial oxygen saturation may occur in the blood of patients with mitral stenosis. This unsaturation might be due to impairment of diffusion of oxygen across the alveolar membrane or to impairment of ventilation-perfusion relationships in the lung. Both such abnormalities have been found in patients with mitral stenosis (1, 2, 3), but it is highly unlikely that appreciable unsaturation could occur during the breathing of room air as a result of impaired diffusion. This report deals with the pulmonary diffusing capacity and ventilation-perfusion relationships in nine patients with mitral stenosis before and after mitral commissurotomy.

MATERIAL AND METHODS

The patients were from the wards of Walter Reed Army Hospital and demonstrated evidence of uncomplicated mitral stenosis which had led to progressive dyspnea and/or hemoptysis. In each instance the diagnosis was confirmed at the time of commissurotomy.

The patients were studied in the fasting state during the inspiration of room air and a low oxygen mixture. Expired air was collected for a three-minute interval in the midst of which arterial blood was allowed to flow from an indwelling radial arterial needle for a one-minute period into a syringe containing mercury and a minimum of heparin. Arterial gas tensions were measured immediately by the direct method of Riley, Proemmel, and Franke (4) which has been standardized in this laboratory (5). Expired gas volumes were measured in a Tissot spirometer and the composition was measured by means of the Scholander micro-gas analyzer (6). Alveolar oxygen tension was calculated from the alveolar equation, substituting the arterial pCO_2 for the alveolar pCO_2. The oxygen diffusing capacity (D_{O_2}) and the percentage venous admixture ($\dot{Q}va/\dot{Q}t$) as defined in the expanded sense by Riley and Cournand (7, 8) were calculated, assuming a 25 per cent difference in saturation between end-capillary and mixed venous blood ($P^{\circ}_{c}-P_{\bar{v}}$). In Cases 4 and 5 a 30 per cent difference in saturation was assumed because of the large amount of venous admixture.

RESULTS

The results of these studies are presented in Table I. In normal subjects, not shown here, the venous admixture averages 3 per cent, varying from 0–8 per cent. Also, in normal subjects, the alveolar-arterial oxygen gradient during the inspiration of a low oxygen mixture is negligible so that the lower limit of normal for D_{O_2} has arbitrarily been selected as 20 (9). Since the methods employed do not measure gradients with an accuracy of more than 3–4 mm. Hg, and since accurate calculation of the diffusing capacity is impossible in the presence of small gradients, D_{O_2} is simply listed as "normal" if the effective alveolar-end-capillary gradient ($P^{\circ}_{A}-P^{\circ}_{c}O_2$) during the inspiration of the low oxygen mixture is less than 4 mm. Hg. In all but three patients the per cent venous admixture was increased, ranging from 8–18 per cent. In all but one instance this became normal after pulmonary congestion was relieved by commissurotomy. Since the arteriovenous difference may become less after operation, and since the estimated amount of venous admixture would err by 3 per cent if the arteriovenous difference varied by 10 per cent from the assumed value, only a change in venous admixture of over 3 per cent may be considered significant. Thus, four out of the six patients with increased venous admixture preoperatively showed a significant fall after operation.

Five of the patients showed resting hyperventilation which, in four, disappeared after commissurotomy. In the patient (Case 7) in whom the hyperventilation and elevated venous admixture effect persisted there was indication of psychological abnormality such that the hyperventilation appeared to have been functional and may account for part of the increase in venous admixture (5).

Oxygen diffusing capacity was not measured in three of the patients preoperatively. In the others, it was reduced in two instances. In Case 2 there was a postoperative increase in diffusing capacity whereas diffusing capacity fell postoperatively in Case 1. The other patients showed slight or no change in this function.

TABLE I

Physiological data on patients with mitral stenosis pre- and post-operatively

Case	Age	B.S.A. *sq.m.*	Sex	Pre or postop.	$P_{I_{O_2}}$ *mm. Hg*	$P_{a_{CO_2}}$ *mm. Hg*	$P_{a_{O_2}}$ *mm. Hg*	R	$\dot{V}$ *L./min.* BTPS	$P^{e}_{A_{O_2}}$ *mm. Hg*	$P^{e}_{C_{O_2}}$ *mm. Hg*	$\dot{Q}va/\dot{Q}t.$ ×100	D_{O_2} *cc./min./ mm.*	D.S./TV ×100	$\dot{V}_{O_2}$ *L./min.* STPD
1	41		M	Preop.	146	30	89	.84		112	112	9			
					146	30	84	.88		113	113	13			
					95	30	53	.86		62	59	(13)	Normal		
				3 weeks	148	34	95	.91	7.02	111	111	6		10	210
				postop.	96	34	49	.94	8.03	60	51	(6)	11	19	202
2	18	1.38	F	Preop.	150	34	88	.73	9.04	106	105	8		34	253
					97	30	48	.90	10.95	64	50	(8)	9	39	221
				12 weeks	100	40	46	.70	7.86	45	45	(2)	Normal	33	259
				postop.	149	42	91	.72	5.52	94	94	2		30	206
3	19	1.75	M	Preop.	148	37	94	.81	6.48	104	104	5		19	241
					96	34	52	.92	7.63	59	54	(5)	16	14	247
				7 weeks	149	37	92	.84	5.67	106	106	6		16	206
				postop.	100	37	50	.83	5.72	56	52	(6)	15	18	198
					71	28	37	1.01	9.26	43	37	(6)	20	15	222
4	38	1.58	F	Preop.	148	32	73	.78	9.27	109	109	17		38	230
					148	32	57	.70	9.72	105	105	33		40	251
				2½ weeks	149	37	83	.80	5.41	105	105	11		28	173
				postop.	97	31	53	.91		66	58	(11)	8		163
5	27	1.52	F	Preop.	148	37	68	.90	8.26	108	100	18*		32	214
					148	38	70	.86	7.96	105	95	15*		32	227
				3 weeks	149	42	84	.89	6.76	103	92	5		33	190
				postop.	149	43	78	.85	6.75	100	88	7		36	191
					101	36	39	1.20	9.47	70	39	(7)	4	38	165
6	35	1.76	F	Preop.	149	39	90	.75	5.54	100	100	5		23	232
					101	37	62	.92	5.98	61	64	(5)	Normal	23	204
				6½ weeks	148	34	90	.70	5.54	102	102	6		18	213
				postop.	70	29	39	1.00	6.22	41	40	(6)	Normal	22	117
7	25	1.38	F	Preop.	148	30	92	.80	7.25	112	112	8		25	192
					148	32	90	.81	6.52	110	110	9		25	181
				5 weeks	97	31	52	.81	7.47	60	56	(10)	14	29	191
				postop.	149	30	87	.73	8.26	110	110	10		25	199
8	41	1.47	F	Preop.	101	36	55	.98	7.70	61	58	(4)	Normal	30	162
					149	37	96	.80	6.76	105	105	4		28	182
				3½ weeks	149	38	92	.83	4.64	105	105	6		21	113
				postop.	100	36	57	.90	7.11	60	60	(6)	Normal	26	180
					71	31	35	.97	8.51	39	35	(6)	23	26	185
9	24	1.86	M	Preop.	148	33	89	.84	8.66	110	110	9		21	261
					100	33	51	.78	8.01	59	54	(9)	20	24	297
				3 weeks	149	36	90	.73	6.67	103	103	6		20	250
				postop.	90	37	42	.78		43	43	(6)	Normal		263

* $P^{e}_{A}-P\bar{c}_{O_2}$ of 37 assumed for calculation of per cent venous admixture, since that was the mean alveolar-capillary gradient determined postoperatively.

DISCUSSION

Certain effects of longstanding mitral stenosis are apparent from these studies. As reported also by others (2, 3) the pulmonary diffusing capacity for oxygen is frequently reduced, although usually not greatly enough to cause an alveolar-arterial oxygen gradient during the inspiration of room air. Four of our patients showed a very low oxygen diffusing capacity postoperatively, but in only one instance (Case 5) did the impaired diffusion cause an appreciable alveolar-arterial gradient during the breathing of room air. This impairment is not likely to be greatly improved by commissurotomy and may be attributed either to thickening of the alveolar capillaries, as has been shown to occur in mitral stenosis (10) or to reduction in the size of the effective pulmonary capillary bed.

The impaired distribution of blood and gas in

the lung, which is largely eliminated by reduction of the pulmonary hypertension, may be attributed to pulmonary congestion with effective blocking of the small airways by transudate. A similar effect has been observed in experimental pulmonary edema (11), in which there is a large venous admixture effect but no impairment of diffusion across the alveolar membrane. Prolonged rest in the supine position is apt to increase the pulmonary congestion. This is exemplified by Case 4 in whom the venous admixture increased from 17–33 per cent as the patient developed mild dyspnea after lying supine for one hour. There was a good correlation between the size of the venous admixture effect and the clinical symptomatology.

The frequent occurrence of resting hyperventilation which disappears after commissurotomy suggests that the engorged pulmonary bed serves as a reflex stimulus to ventilation.

Ventilatory studies were performed on these patients and revealed normal or slightly reduced maximum breathing capacity and vital capacity in each case. Only in the patient with a 33 per cent venous admixture effect was the lung dead space greatly increased (40 per cent of the tidal volume), and this fell postoperatively. A large dead space effect would be expected when large numbers of alveoli are not effectively ventilated. The small increase in dead space in Cases 2 and 5 was not significantly changed postoperatively.

SUMMARY

Pulmonary function has been studied in nine cases of mitral stenosis before and after commissurotomy. Several cases showed an increase in the effective shunting of venous blood through unaerated or poorly ventilated lung with, in some instances, arterial unsaturation. This abnormality was largely corrected by commissurotomy. Pulmonary diffusing capacity was frequently impaired, little affected by operation but usually not sufficiently reduced to cause lowering of arterial oxygen tension during the breathing of room air. Resting hyperventilation, when due to pulmonary congestion, was diminished after commissurotomy.

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