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Research Article

THE EFFECT OF INCREASED HEART RATE DUE TO THE INJECTION OF ATROPINE ON THE OXYGEN SATURATION OF THE ARTERIAL AND VENOUS BLOOD OF PATIENTS WITH HEART DISEASE

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The present study was undertaken in an attempt to contribute to the solution of the problem of the effect of rapid heart rate on the oxygen saturation of the arterial blood. In 1921 Barcroft, Bock and Roughton (1) reported that they had found normal saturation of the arterial blood in a patient during an attack of paroxysmal tachycardia. Carter and Stewart (2) later reported a case in which there was a marked decrease in the oxygen saturation of the arterial blood during attacks of paroxysmal auricular tachycardia, and Dieuaide (3) had the same experience in a patient during attacks of paroxysmal ventricular tachycardia. Meakins (4) found normal saturation of the arterial blood with oxygen during regular and irregular tachycardia artificially induced in dogs. These dogs were under paraldehyde anesthesia and since the chests were open they were kept alive by artificial respiration. The author has had occasion to repeat these experiments under the same conditions and has confirmed these observations (5). More recently, Stewart, Crawford and Hastings (6) in their study of the effect of rapid heart rate on the blood flow in normal unanesthetized dogs found that the oxygen saturation of the arterial blood remained unchanged during rapid auricular fibrillation and was usually unchanged during regular tachycardia.

METHODS

The following observations were made in cardiac patients who had been in the hospital at absolute rest for a long while and who, at the time the observations were made, showed no signs of decompensation. Patients having auricular fibrillation as well as those having normal rhythms were available; in one patient the

TABLE 1

The effect of increased heart rate on the oxygen saturation of the arterial and of the venous arm blood of patients with heart disease

Case number	Age	Rhythm	Time with reference to atropine injection	O_2 content		O_2 consumed	O_2 capacity arterial blood	O_2 saturation		Coefficient of utilization	Summary of change in coefficient of utilization*	Ventricular rate per minute	Radial rate per minute	Pulse deficit	Digitalis	Diagnosis
				Arterial blood	Venous blood			Arterial blood	Venous blood							
	years			*mM*	*mM*	*mM*	*mM*	*per cent*	*per cent*	*per cent*						
1	30	A. F.†	Before	9.49	7.51	1.98	9.72	96.7	76.8	19.9		93	85	8	+‡	Mitral insufficiency; chronic myocarditis
			16 min. after	9.40	7.61	1.79	9.62	96.7	78.6	18.1	0	149	99	50		
2	20	A. F.	Before	9.66	5.60	4.06	9.76 (9.41)§	98.0	59.1	38.9		75	68	7	+	Mitral stenosis and insufficiency; cardiac hypertrophy
			50 min. after	9.34	6.38	2.96	9.53 (9.45)§	97.0	67.1	29.9	—	134	92	42		
		A. F.**	Before	9.63	5.14	4.49	9.89	96.4	51.5	44.9		93	71	22	+	Mitral stenosis and insufficiency; cardiac hypertrophy
			24 min. after	9.83	8.26	1.51	10.14	96.0	81.1	14.9	—	187	72	115		
3	27	A. F.	Before	8.17	5.19	2.98	8.45	95.6	60.9	34.7		77	77	0	+	Mitral stenosis and insufficiency; cardiac hypertrophy
			25 min. after	8.18	5.52	2.66	8.56	94.5	64.0	30.5	—	112	78	34		
4	20	A. F.	Before	8.66	6.15	2.51	8.96	95.6	68.2	27.4		83	81	2	+	Mitral stenosis and insufficiency; aortic insufficiency; cardiac hypertrophy
			20 min. after	8.72	6.81	1.92	8.88	97.3	76.2	21.1	—	127	85	42		

5	38	A. F.	Before	9.54	5.58	3.96	9.91	95.3	55.9	39.4		86	70	16	+	Chronic myocarditis
			35 min. after	9.39	5.53	3.86	9.89	94.0	55.5	38.5	0	125	76	49		
		N. R.††	Before	8.13	5.66	2.47	8.69	92.5	64.7	27.8		64			+	Chronic myocarditis
			24 min. after	8.22	5.75	2.47	8.51	95.5	67.1	28.4	0	88				
6	18	N. R.	Before	9.61	7.42	2.19	10.06	94.6	73.4	21.2		80			+	Chronic myocarditis
			40 min. after	9.78	8.74	1.04	10.18	95.2	85.5	9.7	−	120				
7	20	N. R.	Before	9.24	7.69	1.55	9.70	94.3	78.9	15.4		88			0‡	Mitral stenosis and insufficiency; cardiac hypertrophy
			28 min. after	9.10	6.64	2.46	9.39	96.0	70.3	25.7	+	136				
8‡‡	24	N. R.	Before	3.61	1.64	1.97	3.79	92.9	42.2	50.7		84			0	Mitral insufficiency; cardiac hypertrophy; arterial hypertension; chronic nephritis; simple anemia
			75 min. after	3.62	1.56	2.06	3.89	90.7	39.1	51.6	0	118		75		

* 0, −, and + in this column indicate no change, decrease and increase respectively.

† A. F. = auricular fibrillation.

‡ + indicates that the patient was under the influence of digitalis at the time the test was carried out; 0 indicates that the patient was not under the influence of digitalis at the time the test was carried out.

§ The oxygen capacity of the venous as well as of the arterial blood was estimated in this test.

** These observations were made one year after the first ones.

†† N. R. = normal rhythm. These observations were made 6 days after the ones made during auricular fibrillation.

‡‡ This patient was given 1.7 mgm. of atropine sulphate. The other patients were given 2 mgm.

observations were made during auricular fibrillation and later after the rhythm had returned to normal following the administration of quinidine sulphate. All the patients with auricular fibrillation as well as patients 5 and 6 (table 1) with normal rhythms were under the influence of digitalis at the time the observations were made. The plan was to study the oxygen saturation during a period of a slow cardiac rate and shortly afterward during a period of more rapid rate. The rapid rate resulted from the injection of atropine sulphate (2.0 mgm.) intravenously. The test was started at least 2 hours after the preceding meal. The patients lay quietly in bed for ½ hour before the test was begun. Several counts of the heart rate were then made at 5 minute intervals. A sample of arterial blood was taken from a radial or brachial artery and a sample of venous blood without stasis from a cubital vein. The patient continued to lie quietly while these blood samples were analyzed for their oxygen content. In the meantime several more control heart rates were taken. Atropine sulphate 2.0 milligrams was then given intravenously and after the maximum increase in heart rate had been present for a varying length of time second samples of arterial and of venous blood were taken. In drawing this sample of venous blood the needle was always inserted at the same point in the same vein from which the first sample had been obtained. Heart rates were counted at 5 minute intervals following the injection until the rate returned to normal, in order to be certain that the second blood samples were taken while the heart was still beating at the maximum rate. In patients with auricular fibrillation both the apex heart rate and the radial rate were counted and the pulse deficit plotted. The oxygen content of the blood samples was estimated by the Van Slyke and Neill manometric method (7). The oxygen capacity of the arterial blood was used in calculating the oxygen saturations.

OBSERVATIONS

There are six observations in 5 patients with auricular fibrillation and four in 4 patients with normal rhythm; in one of these (case 5) similar observations were made also during the presence of auricular fibrillation.

The effect of the increased heart rate on the oxygen saturation of the arterial blood. In no instance was there a conspicuous increase or decrease in the oxygen saturation of the arterial blood during the increased heart rate either in patients with normal rhythms or in those with auricular fibrillation (table 1). The heart rate rose as high as 187 per minute; the greatest increase in rate took place in the patients with auricular fibrillation. In one patient (case 2) similar results were observed in the two tests made approximately 1 year apart.

The effect of the increased ventricular rate on the oxygen saturation of

the venous arm blood. In 3 patients with auricular fibrillation (cases 2, 3 and 4) (tables 1 and 2) and mitral stenosis the venous oxygen saturation was increased during the period of rapid rate, while in two patients without mitral stenosis but with auricular fibrillation (cases 1 and 5) it was unchanged. One patient (case 7) with mitral stenosis and a normal rhythm showed a slight decrease in the venous oxygen saturation during the period of increased heart rate. Of the other 3 patients having a normal rhythm one without valvular disease (case 5) and another with mitral insufficiency (case 8) showed no change in venous saturation during the time of increased heart rate, while in a third patient without valvular disease (case 6) venous saturation was increased during the period of faster rate (table 2).

TABLE 2

The effect of increased heart rate on the oxygen saturation of the venous arm blood

Number of cases	Rhythm and valve lesion	Venous saturation during increased heart rate
3	A. F.*with mitral stenosis	Increased
2	A. F. without mitral stenosis	Unchanged
1	N. R.† with mitral stenosis	Decreased
3	N. R. without mitral stenosis	Unchanged or increased

* A. F. = auricular fibrillation.

† N. R. = normal rhythm.

The results of these observations may then be summarized as follows:

A. In patients with heart disease, tachycardia *per se*, whether with regular or irregular rhythm, does not change the degree of *arterial* oxygen saturation.

B. During the time of rapid rate the degree of *venous* oxygen saturation is

(a) *unchanged* in cases of valvular disease (other than mitral stenosis) irrespective of the rhythm.

(b) *increased* (1) in mitral stenosis if auricular fibrillation is present and

(2) with undamaged valves if normal rhythm is present.

(c) *decreased* in mitral stenosis if normal rhythm is present.

DISCUSSION

In patients with heart disease, tachycardia whether regular or irregular did not decrease the arterial oxygen saturation *per se*. In one instance (case 2, 1925) the ventricular rate increased from 93 to 187 per minute, an increase of 100 per cent; in spite of this the oxygen saturation of the arterial blood remained unchanged (fig. 1). The saturation of the venous arm blood increased from 52 to 81 per cent. How increased venous saturation occurred in this patient during tachycardia as well as in patients 3 and 4 is not easy to understand, since the number of effective beats during the period of rapid rate did not change. The point has been made by Goldschmidt and Light (8) that the oxygen content of the blood in the veins of the forearm is affected to some extent by variations in temperature and position, but these conditions remained unchanged in our observations.

The patient (case 5) in whom the test was first made when auricular fibrillation was present gave a similar response (fig. 2) on repeating the test a few days later when the heart rhythm was normal. The venous saturation was higher during the normal rhythm as Stewart (9) has previously shown.

The increased venous saturation which occurs during increased ventricular rate in auricular fibrillation is not to be confused with the decreased saturation of the mixed venous blood which Stewart, Crawford and Hastings (6) have found to take place when the normally beating heart is made to fibrillate. In the latter case (decreased saturation) a change in rhythm has occurred. The increases in heart rate which followed the injection of atropine may have been within the limits to which the heart responds effectively by increased or unchanged minute volume output. Increases beyond these limits or increases in rate lasting for a longer time may be accompanied by failure on the part of the heart to maintain saturation. These increases in ventricular rate took place moreover without an increased demand on the heart by the organism.

These studies throw no light on the cause of arterial anoxemia in the cases reported by Carter and Stewart (2) and Dieuaide (3). Arterial saturation remains unaltered in the presence of extreme myocardial and valvular disease although the ventricular rate is very rapid,

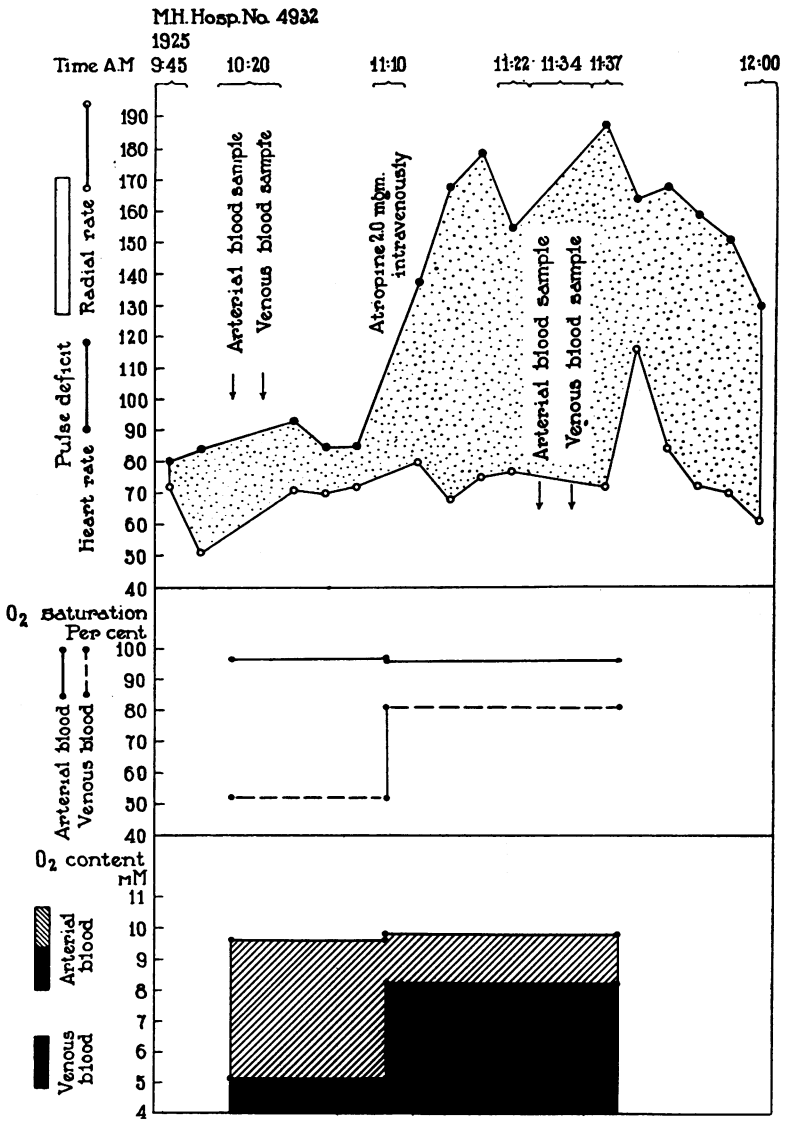

FIG. 1. This figure shows the effect of increased heart rate upon the oxygen saturation and the oxygen content of the arterial and of the venous arm blood in case 2 in 1925. The changes for convenience are represented as taking place immediately after the onset of the increased rate.

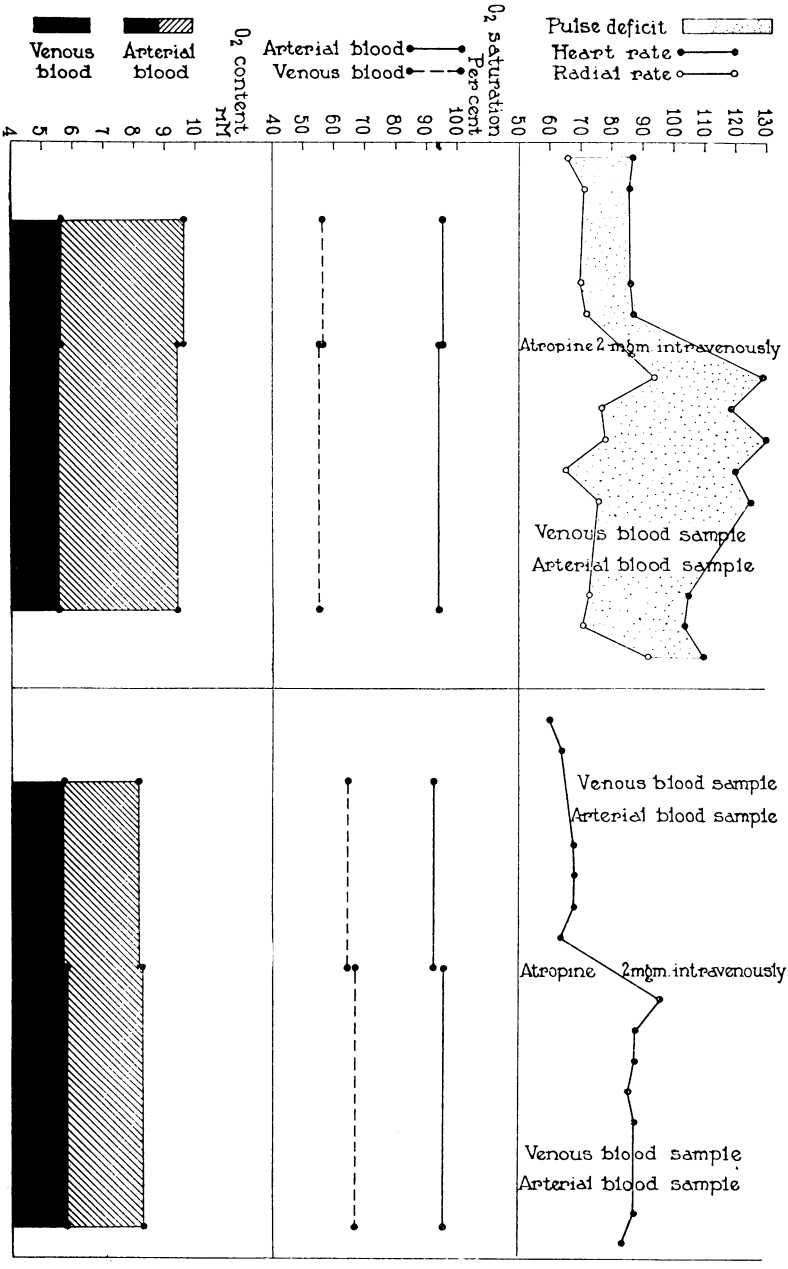

FIG. 2. This figure compares the effect of increased heart rate on the oxygen saturation and the oxygen content of the arterial and of the venous arm blood in case 5, during auricular fibrillation and later during the normal rhythm. For convenience the changes are represented as taking place immediately after the onset of the increased heart rate.

as these and other observations show (case 2, table 1). In the cases mentioned above there may have been pulmonary congestion which interfered with complete oxygenation of the blood in the lungs. There were however no râles over the chest in either of these cases. On the other hand sufficient congestion to interfere with normal oxygenation of the blood may have been present without giving rise to enough moisture to produce râles. The myocardial damage may have been too great for the heart to maintain an adequate circulation during paroxysms of tachycardia, the oxygen unsaturation of the venous blood becoming so marked that it could not be raised to the normal level of arterial blood in the pulmonary circulation time. Once the venous blood has become unduly unsaturated an additional burden is placed on the heart, because of increased viscosity of unsaturated blood (10). The increased viscosity of unsaturated blood may play an important and hitherto unstressed rôle in the onset and continuance of heart failure especially when the large size of the vascular bed is considered.

In patients with mitral stenosis, the difference in behavior during irregular tachycardia and in the presence of the normal rhythm corresponds with the clinical impression that they often seem better after the onset of auricular fibrillation than when the rhythm was normal. On studying from this point of view the data which the author (9) published on oxygen saturation of the arterial and venous arm blood in auricular fibrillation and after restoration of the normal rhythm, it is found that there were only two patients (cases 4 and 9), in whom the venous oxygen saturation did not increase following the return to the normal rhythm; both of these patients had mitral stenosis. One of these patients was subjectively improved following the return to the normal rhythm, while the other one was miserable until fibrillation returned. In the other 7 patients without mitral stenosis the venous saturation increased.

If increased venous saturation may be taken as a measure of improvement in the circulation these observations may be interpreted in the following manner. In mitral stenosis, it is after the onset of auricular fibrillation that improvement takes place as an accompaniment of increased venous saturation. In the absence of mitral stenosis, the circulation is not improved by the onset of auricular

fibrillation; increased venous saturation does not occur. The determining factor then is mitral stenosis; in its presence, auricular fibrillation is an advantage, without it, a disadvantage. This correlation is however too curious to permit its being maintained on the basis of these few observations. Undoubtedly the state of the heart muscle as well as the dynamics of the blood flow must be taken into account before the adoption of definitive opinions.

CONCLUSIONS

1. Tachycardia *per se* whether regular or irregular does not affect the oxygen saturation of the arterial blood in patients with heart disease.

2. In this series of observations it appears that tachycardia may be followed by increased oxygen saturation of the venous arm blood in patients without mitral stenosis in the presence of a normal rhythm and in patients with mitral stenosis in the presence of auricular fibrillation.

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