ERYTHROCYTE SEDIMENTATION IN CARDIAC FAILURE

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It is generally believed that the erythrocyte sedimentation (E.S.R.) is retarded and tends to fall to normal when decompensation occurs in rheumatic fever and other heart diseases known to be associated with elevated values.^{2, 4, 5, 7, 9} Observations by McGinnis and co-workers,⁶ who excluded patients who presented evidence of other conditions known to lead to elevation of the E.S.R., however, disclosed that the majority of the patients with cardiac failure exhibited elevated values both initially and after recovery from failure. Their data indicated that the E.S.R. tends to be elevated in both acute and chronic congestive failure. Further adequate data on the subject do not seem to be available in the literature. The purpose of this paper is to report a study of the E.S.R. in 190 patients with cardiac failure.

Material and Methods

One hundred and ninety patients admitted to the hospital for treatment of cardiac failure of varied etiology have been included in this study; patients who died were excluded. There were 124 male and 66 female patients. The age and sex distribution of the patients is given in Table 1. Rheumatic, emphysema, coronary, and hypertensive heart disease accounted for 34, 20, 19 and 7%, respectively, of the total cases.

E.S.R. determinations were made by the method of Westergren, initially within 24 hours of admis-

sion and subsequently at intervals of five to seven days until after the relief of failure; 1.6 cc. of blood were drawn from an arm vein in a syringe containing 0.4 cc. of 3.8% sodium citrate solution. For the purpose of this study, a first hour reading of 15 mm. or more and/or a second hour reading of 30 mm. or more was considered definitely pathological and abnormally elevated. In repeat estimations a rise of 5 mm. or more from the initial value was considered a significant elevation. This elevation was divided into three categories: further rise from initial abnormal values, and rise from initial normal values either to abnormal values or to one still less than the normal limit of 15 mm.

The E.S.R. was also estimated on admission in another 125 cardiac patients, but without failure, to serve as control.

Results

The incidence of abnormally elevated and normal E.S.R. values on admission and of significant rises from initial values after relief of failure in various types of heart disease is given in Table 2. The E.S.R. values at the end of one hour in individual cases are given in Table 3. An abnormally elevated E.S.R. was present on admission in 96 (50%) cases, with values of 30 mm. or more at the end of one hour in 47 (25%) cases. The highest value recorded was 125 mm. Cases with

TABLE I
AGE AND SEX DISTRIBUTION OF 190 PATIENTS ACCORDING TO TYPE OF HEART DISEASE

	Sex		Age (years)							
	M	F	1-10	11-20	21-30	31-40	41-50	51-60	61-70	Total
Rheumatic	39	26	3	22	19	13	8	—	—	65
Emphysema	30	9	—	—	—	10	15	11	3	39
Coronary	25	11	—	—	—	4	13	13	6	36
Hypertensive	9	5	—	—	2	4	5	3	—	14
Syphilitic, pericarditis and miscellaneous	21	15	—	13	10	5	5	3	—	36
Total	124	66	3	35	31	36	46	30	9	190

TABLE 2

INCIDENCE OF ABNORMAL AND NORMAL E.S.R. ON ADMISSION AND OF SIGNIFICANT ELEVATION AFTER COMPENSATION ACCORDING TO TYPE OF HEART DISEASE

Type of Heart Disease	Total Cases	Abnormal E.S.R. on Admission		Normal E.S.R. on Admission		
		Total Cases	No. with further Elevation after Compensation	Total Cases	No. with Elevation after Compensation	
					Up to 15 mm.	Abnormal
Rheumatic	65	37	14	28	5	15
Emphysema	39	13	7	26	1	8
Coronary:						
No infarction ..	8	4	1	4	0	1
Old	10	4	0	6	2	2
Recent	18	13	5	5	0	5
Hypertensive:						
Benign	6	1	0	5	2	1
Malignant	4	3	0	1	0	0
Renal	4	4	3	0	0	0
Syphilitic	6	3	2	3	0	3
Pericarditis:						
Benign	3	3	2	0	0	0
Tuberculous ..	3	3	0	0	0	0
Constrictive ..	3	0	0	3	0	0
Haemorrhagic ..	1	0	0	1	0	0
Congenital	4	0	0	4	1	0
Idiopathic cardiomegaly	6	0	0	6	0	3
Acute left heart failure						
in acute nephritis ..	4	3	0	1	0	1
Disseminated lupus ..	2	2	0	0	0	0
Bacterial endocarditis	2	2	0	0	0	0
Polyarteritis	1	1	1	0	0	0
Fibroelastosis	1	0	0	1	0	0
Total	190	96	35	94	11	39

syphilitic heart disease, recent myocardial infarction, malignant and renal hypertension, tuberculous and benign idiopathic pericarditis, and acute nephritis with acute left ventricular failure usually showed abnormal initial values. Cases with benign hypertension, constrictive pericarditis and congenital heart disease usually showed normal values.

The E.S.R. remained normal, initially as well as subsequently, in 55 (29%) cases, including 13 cases of rheumatic heart disease, 18 of emphysema, 7 of coronary heart disease without recent infarction, 4 of benign hypertension, and all the four cases of congenital heart disease and three of constrictive pericarditis. After relief of failure a significant rise of E.S.R. from initial values was seen in 85 (45%) cases. Of the 96 cases with initial abnormal values, there was further elevation in 35 (39%) of them. Of 94 cases with initial normal values, there was abnormal elevation in 39 (42%) of them, and there was elevation of 5 mm. or more with values that still remained normal in 11 (12%) of them. The rise of E.S.R. from the initial values varied between five and 78 mm. with an average of 38 mm. Of cases with initial normal and abnormal E.S.R. values, respectively, 70% and 40% with rheumatic heart disease and 34%

and 54% with emphysema heart disease showed significant elevation after relief of failure.

Discussion

In the present study the influence of cardiac failure and of establishment of compensation on erythrocyte sedimentation has been determined in a variety of heart diseases, known to be associated with both normal as well as abnormal E.S.R. Westergren's method of E.S.R. determination was used as it is considered to be more sensitive sometimes than Wintrobe's method,⁸ superior for serial examinations,³ and the method of choice for general purposes.⁷ In 96 cases (50%) the E.S.R. remained abnormally elevated despite the presence of cardiac failure, often severe, and values as high as 125 mm. were seen. It was, however, significant that, during decompensation, there was normalization of E.S.R. in 39 cases (20%) in whom the E.S.R. became abnormal after compensation, and that significant elevation of E.S.R. after establishment of compensation was noted in a total of 45% cases. These figures for elevation may be considered as somewhat less than the actual. It is likely that in some cases therapy directed against the etiological factor, or regression of the disease process concomitantly with the

TABLE 3

E.S.R. VALUES AT THE END OF ONE HOUR, ON ADMISSION AND AFTER COMPENSATION. IN EACH DISEASE CASES SHOWING SIGNIFICANT ELEVATION OF E.S.R. AFTER COMPENSATION ARE NUMBERED FIRST

Type of Heart Disease and Serial No.	E.S.R. Values		Type of Heart Disease and Serial No.	E.S.R. Values		Type of Heart Disease and Serial No.	E.S.R. Values	
	On Admission	After Compensation		On Admission	After Compensation		On Admission	After Compensation
RHEUMATIC			RHEUMATIC—			Old infarction—		
1	2	8	<i>contd.</i>			<i>contd.</i>		
2	3	10	63	90	40	12	3	11
3	3	10	64	90	48	13	1	1
4	3	12	65	112	90	14	15	10
5	4	10				15	15	4
6	2	35	EMPHYSEMA			16	15	10
7	2	35	1	3	10	17	20	17
8	2	45	2	5	22	18	20	15
9	3	33	3	7	20			
10	4	34	4	8	42	Recent infarction		
11	5	20	5	9	21	19	2	32
12	5	35	6	10	32	20	3	50
13	5	40	7	10	33	21	5	40
14	5	42	8	10	50	22	5	40
15	5	43	9	13	45	23	10	60
16	8	23	10	15	90	24	15	40
17	10	18	11	22	90	25	16	40
18	10	40	12	25	55	26	22	100
19	11	42	13	30	85	27	30	60
20	12	42	14	45	60	28	65	90
21	15	60	15	60	65	29	16	10
22	15	68	16	60	65	30	20	15
23	16	40	17	2	4	31	25	10
24	16	40	18	3	5	32	25	25
25	17	42	19	3	5	33	30	23
26	19	24	20	3	5	34	37	20
27	20	28	21	4	5	35	78	55
28	20	50	22	4	6	36	80	60
29	25	36	23	5	5			
30	25	62	24	5	3	SYPHILITIC		
31	27	80	25	8	7	1	10	60
32	28	72	26	8	12	2	11	30
33	30	42	27	9	5	3	13	23
34	50	120	28	9	8	4	20	38
35	3	5	29	10	12			
36	4	6				5	25	82
37	5	4	30	12	8	6	15	15
38	5	5	31	12	10			
39	5	7	32	12	10	PERICARDITIS		
40	5	8	33	12	3	Benign		
41	10	5	34	24	17	1	35	60
42	12	8	35	29	11	2	45	55
43	18	7	36	35	30	3	32	30
44	19	20	37	38	17			
45	20	5	38	40	25	Tuberculous		
46	20	10	39	45	30	4	25	22
47	20	10				5	45	35
48	22	8	CORONARY			6	89	52
49	23	12	No infarction					
50	23	18	1	11	25	Constrictive		
51	24	6	2	20	70	7	5	9
52	25	7	3	2	4	8	11	9
53	25	12	4	2	6	9	12	12
54	30	19	5	4	6			
55	38	19	6	15	12	Haemorrhagic		
56	40	2	7	15	10	10	13	6
57	40	10	8	20	16			
58	52	40				HYPERTENSION		
59	55	30	Old infarction			Benign		
60	60	12	9	5	40	1	2	8
61	65	12	10	5	48	2	3	10
62	75	40	11	3	10			

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Type of Heart Disease and Serial No.	E.S.R. Values		Type of Heart Disease and Serial No.	E.S.R. Values		Type of Heart Disease and Serial No.	E.S.R. Values	
	On Admission	After Compensation		On Admission	After Compensation		On Admission	After Compensation
Benign— <i>contd.</i>			Congenital— <i>contd.</i>			Systemic lupus		
3	11	25				13	40	25
4	5	8	2	3	6	14	45	40
5	7	9	3	5	5			
6	18	18	4	7	6	Bacterial endocarditis		
Renal			Idiopathic cardiomegaly			15	90	65
7	65	140				16	65	50
8	82	100	5	3	20	Acute left failure in acute nephritis		
9	125	140	6	10	40			
10	35	23	7	13	25	17	14	35
Malignant			8	3	4	18	25	20
11	11	8	9	4	4	19	30	30
12	55	50	10	8	7	20	35	30
13	60	40	Fibroelastosis					
14	103	60	11	6	10			
MISCELLANEOUS			Polyarteritis					
Congenital			12	40	58			
1	4	12						

relief of failure, tended to cause decrease or normalization of the E.S.R. and therefore opposes the elevation that might have occurred after compensation. For example, aspirin and penicillin were administered from the beginning in cases of rheumatic heart disease with elevated E.S.R. on admission, but in those with normal E.S.R. on admission they were not administered unless and until the E.S.R. became elevated subsequently. Again, antibiotics were administered routinely to all cases with emphysema heart failure. This may also explain the lesser incidence of subsequent elevation in cases with abnormal initial values in comparison with those with normal initial values. The elevation of E.S.R. after establishment of compensation was often marked, being more than 30 mm. in 39 and more than 40 mm. in 18 cases, the maximum being as high as 78 mm. The average E.S.R. in these 85 cases was 18 mm. on admission and 56 mm. after compensation.

The question arises as to why the E.S.R. rises after the establishment of compensation. It has been the general belief that congestive cardiac failure has a retarding influence on the E.S.R., tending to cause its normalization. McGinnis and co-workers⁶ studied the E.S.R. in patients with heart failure due to arteriosclerotic, hypertensive, and syphilitic heart disease. They apparently considered that the E.S.R. is not elevated in these heart diseases, except in the presence of some complication. They found that the E.S.R. was elevated in most patients, initially as well as after compensation. Speculating on the cause of

elevation of the E.S.R. after compensation, they postulated that absorption of the products of tissue necrosis as a result of passive congestion may play a part in the increase of E.S.R. in the period immediately after compensation. In our study the E.S.R. was normal in majority of the patients with benign hypertension. It was elevated in all of the patients with syphilitic aortic valvular disease and in 60% of those with coronary artery disease without recent infarction. Similar results were obtained in control patients without heart failure. Whether the elevation in these two diseases is due to the heart disease itself or to the presence of some other inapparent concomitant complication is uncertain. The E.S.R. has been stated to be increased in syphilitic aortitis and in certain cases of angina.⁹ In emphysema heart disease the E.S.R. was elevated in 54% of the cases. In this disease the failure is often precipitated by some respiratory infection which seems to be the most likely explanation for the elevated values. Age or sex appeared to have no relation to the elevation of E.S.R.

In the present series the E.S.R. was normal on admission in 50% of the cases and after compensation in 40% of the cases. It remained normal, both initially as well as after compensation, in 29% of the cases; this included 20% of the cases with rheumatic heart disease in whom no aspirin or antibiotics were administered, 46% of those with emphysema heart disease, 40% of those with coronary heart disease without recent infarction, most of the cases of benign hypertension, and all the cases of congenital heart disease and con-

strictive pericarditis. The data in our study, therefore, do not support the hypothesis of McGinnis and co-workers⁶ that the E.S.R. tends to be elevated in patients with heart failure. On the contrary, our data strongly support the concept that cardiac failure has a retarding influence on the E.S.R. and that elevation of the E.S.R. after compensation is due to removal of this influence. The cause of this retarding influence requires investigation.

Bland and Jones¹ found evidence which suggested that acute rheumatic fever is frequently an unrecognizable concomitant of cardiac failure in patients with rheumatic heart disease. In our series also, a high E.S.R. was noted initially in 57% of the cases, and subsequently in another 23% of cases. This suggests that the failure is associated with activity of the rheumatic process in a great majority of the cases. The observation is of significance because it suggests that anti-rheumatic therapy is indicated from the beginning in the majority of cases of rheumatic heart disease with failure, even if the initial E.S.R. is normal.

Summary

Erythrocyte sedimentation was studied, on admission and at intervals until after compensation was established, in 190 patients with congestive cardiac failure.

E.S.R. values were elevated in 50% of the cases on admission. Significant elevation of E.S.R. values after recovery from failure was observed in 45% including abnormal elevation from initial normal values in 20%. The E.S.R. remained normal, both initially as well as subsequently, in 29%.

It is concluded that cardiac failure has a definite retarding influence on the E.S.R. and tends to cause normalization of E.S.R.

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REFERENCES

1. BLAND, E. F., and JONES, T. D. (1952), *Ann. intern. Med.*, **37**, 1006.
2. DAVIS, L. J. (1946), *Practitioner*, **157**, 13.
3. GOLDBERG, S., GLYNN, L. E., and BYWATERS, E. G. L. (1952), *Brit. med. J.*, **1**, 202.
4. HARRISON, T. R. (1954), 'Principles of Internal Medicine', pp. 485 and 880. New York: The Blackiston Co. Inc.
5. KEITH, J. D., ROWE, R. D., and VLAD, P. (1958), 'Heart Disease in Infancy and Childhood', p. 626. New York: The Macmillan Co.
6. MCGINNIS, A. E., LANSCH, W. E., and GLASER, R. J. (1953), *Amer. J. med. Sci.*, **225**, 599.
7. SCHLESINGER, B. (1946), *Practitioner*, **157**, 38.
8. SINTON, R. J. (1948), *Brit. med. J.*, **1**, 391.
9. WHITBY, L. E. H., and BRITTON, C. J. C. (1957), 'orders of the Blood', p. 129. London: J. & A. Churchill.

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