ATRIAL FUNCTION AND ITS CLINICAL IMPLICATIONS

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I. Introduction

An attempt to evaluate the importance of atrial function in cardiovascular dynamics seems appropriate in view of two recently developed therapeutic measures which have in common the restoration of a properly timed atrial contraction. These therapeutic methods are (1) the treatment of atrial fibrillation by the use of direct current countershock, and (2) the treatment of complete heart block by implanting a pacing electrode, which is excited by atrial depolarization, on the ventricle.

II. Treatment of Atrial Fibrillation by Cardioversion

1. Zoll, P. M. and Linenthal, A. J.: Treatment of refractory tachycardia by external countershock. Circulation 25:596, 1962.

2. Paul, M. H. and Miller, R. A.: External electrical termination of supraventricular arrhythmias in congenital heart disease. Circulation 25:604, 1962.

3. Lown, B., Amarasingham, R. and Neuman, J.: New method for terminating cardiac arrhythmias. Use of synchronized capacitor discharge. J.A.M.A. 182:548, 1962.

4. Lown, B., Neuman, J., Amarasingham, R. and Berkovits, B. V.: Comparison of alternating current with direct current electroshock across the closed chest. Am. J. Cardiol. 10:223, 1962.

5. Lown, B., Perlroth, M. G., Kaidbey, S., Tadaaki, A. and Harken, D. E.: "Cardioversion" of atrial fibrillation. New England J. Med. 269:325, 1963.

6. Lown, B., Bey, S. K., Perlroth, M. G. and Abe, T.: Cardioversion of ectopic tachycardias. Am. J. M. Sc. 246: 257, 1963.

7. Killip, T.: Synchronized DC precordial shock for arrhythmias. J.A.M.A. 186:1, 1963.

8. Lown, B., Bey, S. K., Perlroth, M. and Abe, T.: Cardioversion of ectopic tachycardias. Trans. Assoc. Am. Phys. 76:262, 1963.

From these various studies, both in dogs and man, it appears that direct current countershock or cardioversion is a relatively safe and reliable treatment of atrial fibrillation. However, this method is not without morbidity and possible mortality. Therefore, an estimation of the expected improvement in cardiovascular dynamics when atrial function is restored must be made.

III. Treatment of Heart Block by Cardiac Pacemakers

9. Weirich, W. L., Gott, V. L. and Lillehei, C. W.: The treatment of complete heart block by the continued use of a myocardial electrode and an artificial pacemaker. Surg. Forum 8:360, 1957.

10. Weirich, W. L., Paneth, M., Gott, V. L. and Lillehei, C. W.: Control of complete heart block by use of an artificial pacemaker and a myocardial electrode. Circulation Res. 6:410, 1958.

11. Nathan, D. A., Center, S., Wu, C. and Keller, W.: An implantable synchronous pacemaker for the long term correction of complete heart block. Am. J. Cardiol. 11: 362, 1963.

12. Nathan, D. A., Center, S., Wu, C. and Keller, W.: An implantable, synchronous pacemaker for the long-term correction of complete heart block. Circulation 27:682, 1963.

13. Center, S., Nathan, D., Wu, C., Samet, P. and Keller, W.: The implantable synchronous pacer in the treatment of complete heart block. J. Thor. and Cardiovas. Surg. 46:744, 1963.

14. Nathan, D. A., Center, S., Wu, C. and Keller, W.: Long term correction of complete heart block: clinical and physiologic studies of a new type of implantable synchronous pacer. Prog. Cardiovas. Dis. In press.

15. Nathan, D. A., Center, S., Wu, C. and Keller, W.: Application of an implantable synchronous pacer for the correction of complete heart block. Ann. New York Acad. Sci. In press.

The use of electrical stimulation of the ventricle has become a beneficial method of treating complete heart block. Lillehei and his group have treated complete heart block by use of an electrode placed on the ventricle and an artificial pacemaker. The ventricle is then paced at a fixed rate and the proper relation between atrial and ventricular systole is not preserved. More recently Nathan and his co-workers have developed an implantable pacemaker in which atrial depolarization excites the ventricular pacing device. This method (1) allows the ventricular rate to respond to autonomic nerve activity and (2) places atrial and ventricular contractions in the proper time sequence.

IV. Atrial Function

In making clinical decisions in regard to these two methods of therapy it is important to attempt to estimate the expected improvement in cardiovascular dynamics when atrial function is restored. In order to do this one must understand the normal function of the atrium.

Atrial function relates both to the part of the circulation to which it forwards blood (the ventricle), and to the portion from which it receives blood (the venous bed). On the one hand, the contribution of atrial systole to ventricular filling is a determinant of ventricular end-diastolic pressure or volume, which, in turn, is a determinant of ventricular stroke work (Frank-Starling mechanism or heterometric autoregulation). On the other hand, mean atrial or central venous pressure is that level which must on the average be exceeded if blood is to return to the heart.

The functions of the atrium may be outlined as follows:

A. Conduit between venous bed and ventricle.

B. Booster pump action.

   1. Enhancement of ventricular filling.

   2. Maintenance of a low mean atrial pressure in relation to ventricular end-diastolic pressure.

C. Atrioventricular valve closure before ventricular systole.

A. Conduit function

16. Henderson, Y.: The volume curve of the ventricles of the mammalian heart, and the significance of this curve in respect to the mechanisms of the heart beat and the filling of the ventricles. Am. J. Physiol. 16:325, 1906.

It is obvious that the atrium serves as a conduit or feed line between the venous bed and the ventricle. In fact, some investigators have felt that this is the only role that the atrium plays in cardiovascular dynamics. Henderson, in 1906, studied the volume changes of the ventricle of dogs with a cardiometer and found that atrial systole played a negligible role in ventricular filling. He stated that "the mammalian auricles are to be regarded as elastic reservoirs rather than as force pumps." However, if this were the only function of the atrium, then the mean pressure in this chamber would determine both the pressure level to be overcome by inflowing blood and the pressure level that could be achieved in the ventricle at the end of diastole. This is probably analogous to the situation that is present during atrial fibrillation.

B. Booster pump action

17. Gesell, R. A.: Auricular systole and its relation to ventricular output. Am. J. Physiol. 29:32, 1911.

In this study it was stated that an important function of the atria is to provide "an adequate filling of the ventricles with a comparatively low venous pressure - thus preventing a continued strain upon the venous system."

18. Mitchell, J. H., Gilmore, J. P. and Sarnoff, S. J.: The transport function of the atrium. Factors influencing the relation between mean left atrial pressure and left ventricular end-diastolic pressure. Am. J. Cardiol. 9:237, 1962.

This group suggested that in the circulation, the atrium performs a function much like a booster pump. In this role the atrium is able to augment the transfer of blood from the venous bed into the ventricle and to maintain a low mean atrial pressure relative to ventricular end-diastolic pressure.

1. Enhancement of ventricular filling

19. Harvey, W.: Anatomical Studies on the Motion of the Heart and Blood. 3rd ed., p. 40. Springfield, Ill.: Charles C. Thomas, 1949.

The contribution of atrial systole to ventricular filling was first described by William Harvey in 1628: "With the auricles still pulsating after the heart has stopped, it is noteworthy that a finger placed on the ventricles perceives the separate pulsations of the auricles for the same reason as the beat of the ventricles in the arteries is felt, because, as was said before, of the distension from the impact of blood. At this same time when the auricles above are beating, if you cut off the tip of the heart with scissors, you will see blood gush out on each beat of the auricles."

20. Roy, C. S.: On the influences which modify the work of the heart. J. Physiol. 1:452, 1879.

21. Roy, C. S. and Adami, J. G.: Heart-beat and pulse-wave. Practitioner 44: , 1889.

22. Roy, C. S. and Adami, J. G.: Contributions to the physiology and pathology of the mammalian heart. Phil. Trans. (Lond.) 183(B):199, 1892.

The work of Roy and Adami showed that in the frog heart atrial activity was important in filling the ventricle.

16. Loc. cit.

23. Straub, H.: The diastolic filling of the mammalian heart. J. Physiol. 40:378, 1910.

The previously mentioned studies of Henderson, which utilized a cardiometer method, seemed to show that atrial systole was relatively unimportant in the filling of the mammalian ventricle. Straub, who used more accurate instrumentation, found that ventricular volume was augmented up to 60 per cent by atrial systole.

17. Loc. cit.

24. Gesell, R. A.: Auricular systole and its relation to ventricular output. Am. J. Physiol. 29:32, 1911.

25. Gesell, R. A.: The effects of change in auricular tone and amplitude of auricular systole on ventricular output. Am. J. Physiol. 38:404, 1915.

26. Gesell, R. A.: Initial length, initial tension and tone of auricular muscle in relation to myo and cardiodynamics. Am. J. Physiol. 39:239, 1916.

27. Gesell, R. A.: Cardiodynamics in heart block as affected by auricular systole, auricular fibrillation and stimulation of the vagus nerve. Am. J. Physiol. 40:267, 1916.

Gesell examined the role of atrial function in an ingenious series of investigations. He utilized a heart block dog preparation and found that the production of atrial fibrillation caused a decrease in arterial blood pressure up to 20 per cent. Also by gradually altering the time relation between atrial and ventricular systole, marked alterations in arterial blood pressure occurred. Later he examined the importance of atrial contraction in determining cardiac output by use of "auriculo-ventricular interference waves". By this method it was found that atrial systole increased cardiac output about 50 per cent over that maintained by venous pressure alone.

28. Lewis, T.: Fibrillation of the auricles; its effects upon the circulation. J. Exper. Med. 16:395, 1912.

When atrial fibrillation was produced, there was a decrease in heart volume, cardiac output and arterial pressure and an increase in venous pressure. Lewis attributed these changes to the increased ventricular rate and not to the absence of an effective atrial contraction.

29. Stewart, H. J. and Gilchrist, A. R.: Studies on the effect of cardiac irregularity on the circulation. II. The estimation of cardiac output in dogs subject to artificial auricular fibrillation. J. Clin. Invest. 5:335, 1928.

The cardiac output of unanesthetized dogs decreased 22 to 29 per cent when atrial fibrillation was artificially produced.

30. Wiggers, C. J. and Katz, L. N.: Contour of the ventricular volume curves under different conditions. Am. J. Physiol. 58:439, 1921-22.

These investigators, utilizing the cardiometer technique, found that the contribution of atrial systole to ventricular filling ranged from 18 to 60 per cent with an average of about 35 per cent. They stated that the relative importance of atrial systole for ventricular filling depended upon (1) the time that atrial systole comes in diastole, (2) the completeness of ventricular filling at the time of atrial systole and (3) the vigor of atrial systole.

31. Jochim, K.: The contribution of the auricles to ventricular filling in complete heart block. Am. J. Physiol. 122:639, 1938.

The effect of individual atrial systoles on ventricular volume curves in dogs with heart block was correlated with the interval between the initiation of atrial and ventricular contraction (As-Vs interval). Effective atrial contribution reached a maximum at an As-Vs interval of 80 msec.

32. Lind, J., Wegelius, C. and Lichtenstein, H.: The dynamics of the heart in complete A-V block. An angiographic study. Circulation 10:195, 1954.

These investigators demonstrated in human patients with heart block the filling of the ventricle secondary to atrial systole by injecting contrast media and taking rapid x-rays.

33. Linden, R. J. and Mitchell, J. H.: Relation between left ventricular diastolic pressure and myocardial segment length and observations on the contribution of atrial systole. Circulation Res. 8:1092, 1960.

Changes in the length of a segment of left ventricular myocardium were recorded simultaneously with left ventricular pressure. This study indicated both the magnitude of fiber length elongation in the ventricle that can be produced by atrial systole, and the circumstances in which it was likely to produce the greatest effect.

34. Snyder, J. and Wood, E. H.: Effect of heart rate on atrial contribution to cardiac performance in dogs with complete heart block. Fed. Proc. 21:137, 1962. Abstract.

35. Sellers, F. J., Donald, D. E. and Wood, E. H.: Atrial contribution to stroke volume in dogs with chronic cardiac denervation. Physiologist 5:211, 1962.

Wood and his group have studied the change in cardiac output that occurred in closed-chest dogs when atrial systole was made ineffective. An ineffective atrial systole was found to produce a 5 to 10 per cent decrease in cardiac output over a wide range of heart rates (60-200 beats/min). Also in dogs with chronic cardiac denervation but without heart block a 15 per cent decrease in cardiac output occurred when atrial systole was made ineffective over a comparable heart rate range.

36. Brockman, S. K.: Dynamic function of atrial contraction in regulation of cardiac performance. Am. J. Physiol. 204:597, 1963.

In this study which utilized a heart block preparation, ventricular beats at the optimum As-Vs interval were compared with those in which the As-Vs interval was such that there was no atrial contribution. Under the conditions of this experiment the maximal increase in fiber length was 40 per cent and in stroke volume 27 ml.

37. Skinner, N. S., Jr., Mitchell, J. H., Wallace, A. G. and Sarnoff, S. J.: Hemodynamic effects of altering the timing of atrial systole. Am. J. Physiol. 205:499, 1963.

Changing the interval between left atrial and left ventricular systole (As-Vs interval) produced a decrease in left ventricular end-diastolic pressure, mean aortic pressure and aortic flow. The absence of atrial systole produced similar changes. It was concluded that "the atrium contributes significantly to ventricular filling, that autonomic nerve activity can importantly influence the extent of the atrial contribution, that atrial activity can induce closure of the mitral valve prior to the onset of ventricular systole, and that a vigorous and properly timed atrial systole will keep mean atrial pressure at a lower level for any given ventricular end-diastolic pressure produced than would otherwise occur."

38. Skinner, N. S., Jr., Mitchell, J. H., Wallace, A. G. and Sarnoff, S. J.: Hemodynamic consequences of atrial fibrillation at constant ventricular rates. Am. J. Med. 36:342, 1964.

In areflexic dogs with heart block and constant ventricular rates, atrial fibrillation resulted in an increase in mean left atrial pressure, both absolute and relative to left ventricular end-diastolic pressure, and a decrease in aortic flow and aortic pressure. These effects are due to a loss of the contribution of atrial systole to ventricular filling and to mitral regurgitation.

39. Mitchell, J. H., Gupta, D. N. and Payne, R. M.: Influence of atrial systole on effective ventricular stroke volume. Fed. Proc. 23:464, 1964.

Effective ventricular stroke volumes were determined for both ventricular ejections preceded by an atrial systole and those not preceded by an atrial systole. When ventricular beats were not preceded by atrial systole, the average drop in effective ventricular stroke volume was 19 per cent at low heart rates and 34 per cent at high heart rates.

2. Maintenance of a low mean atrial pressure in relation to ventricular end-diastolic pressure

The pressure in the atrium at any given time must be exceeded if blood is to return to the heart and this, on the average, is

represented by mean atrial pressure. Also the pressure level generated in the atrium during atrial contraction influences the left ventricular end-diastolic pressure. Thus from the point of view of integrating the heart with the total organism, it is important to know those conditions which affect the relation between mean atrial pressure and ventricular end-diastolic pressure.

40. Braunwald, E. and Frahm, C. J.: Studies on Starling's law of the heart. IV. Observations on the hemodynamic functions of the left atrium in man. Circulation 24: 633, 1961.

It is stated that the relation between mean atrial pressure and ventricular end-diastolic pressure is quite meaningful since "the ventricular end-diastolic pressure may be considered to be the hemodynamic 'stimulus' which determines the force of ventricular contraction, the mean atrial pressure may be considered the hemodynamic 'price' which the organism must pay for this stimulus to be provided."

18. Loc. cit.

Increased left atrial contractility lowers the mean left atrial pressure for any given left ventricular end-diastolic pressure and a decreased left atrial contractility raises the mean left atrial pressure for any given left ventricular end-diastolic pressure as the heart rate is increased above a certain critical level which is determined by the functional condition of the left ventricle.

41. Wallace, A. G., Mitchell, J. H., Skinner, N. S., Jr. and Sarnoff, S. J.: Hemodynamic variables affecting the relation between mean left atrial and left ventricular end-diastolic pressures. Circulation Res. 13:261, 1963.

The relation between mean left atrial pressure and left ventricular end-diastolic pressure is independent of stroke volume and of aortic pressure. An increase in heart rate above a critical level, which is determined by the functional state of the ventricle, increases mean left atrial pressure in relation to left ventricular end-diastolic pressure. Stimulation of cardiac

sympathetic nerves decreases mean left atrial pressure in relation to left ventricular end-diastolic pressure and this effect is most prominent at rapid heart rates.

C. Atrioventricular valve closure before ventricular systole

It has generally been accepted that atrioventricular valve closure results from the ventriculo-atrial pressure gradient which is caused by ventricular systole. However, some recent studies have suggested that the atrioventricular valve is closed before the initiation of ventricular systole.

42. Baumgarten, A.: Ueber den Mechanismus, durch welchen die venösen Herzklappen geschlossen werden. Arch. Anat. Physiol. Wiss. Med., Müller, 1843, pp. 463-470.

43. Henderson, Y. and Johnson, F. E.: Two modes of closure of the heart valves. Heart 4:69, 1912.

44. Dean, A. L.: The movements of the mitral cusps in relation to the cardiac cycle. Am. J. Physiol. 40:206, 1916.

These studies suggested methods of valve closure which were not due to pressure generation by the ventricles.

45. Little, R. C.: Effect of atrial systole on ventricular pressure and closure of the A-V valves. Am. J. Physiol. 166:289, 1951.

46. Little, R. C., Hilton, J. G. and Schaefer, R. D.: The first heart sound in normal and ectopic ventricular contractions. Mechanism of closure of the A-V valves. Circulation Res. 2:48, 1954.

47. Schaefer, R. D. and Little, R. C.: First heart sound in ventricular contractions arising from the apex and the base. Proc. Soc. Exper. Biol. and Med. 85:639, 1954.

These studies by Little and his co-workers present strong evidence that tricuspid valve closure is due to right atrial activity.

It is stated that "the increase in ventricular pressure resulting from atrial systole is sufficient to reverse the atrioventricular pressure gradient before the onset of ventricular contraction."

48. Müller, O. and Shillingford, J.: Tricuspid incompetence. Brit. Heart J. 16:195, 1954.

49. Daley, R., McMillan, I. K. R. and Gorlin, R.: Mitral incompetence in experimental auricular fibrillation. Lancet 2:18, 1955.

50. Friedman, B., Daily, W. M. and Wilson, R. H.: Studies on mitral valve function. Effect of acute hypervolemia, premature beats and other arrhythmias. Circulation Res. 4:33, 1956.

These studies have indicated the occurrence of either tricuspid or mitral regurgitation during atrial fibrillation.

51. Sarnoff, S. J., Gilmore, J. P. and Mitchell, J. H.: Influence of atrial contraction and relaxation on closure of the mitral valve. Circulation Res. 11:27, 1962.

In heart block dogs, the mitral valve can be closed solely as the result of atrial activity. Closure of the mitral valve is caused by a ventriculo-atrial pressure gradient. After ventricular diastolic pressure has been elevated by atrial systole, the magnitude and rate of decline of atrial pressure during atrial relaxation contributes significantly to this pressure record.

52. Grant, C., Greene, D. G. and Bunnell, I. L.: The valve-closing function of the right atrium. A study of pressures and atrial sounds in patients with heart block. Am. J. Med. 34:325, 1963.

In patients with heart block, isolated atrial contractions occurring during prolonged ventricular diastoles produced a reversed pressure gradient across the tricuspid valve, showing that the tricuspid valve was closed by atrial activity.

37. Loc. cit.

38. Loc. cit.

A platinum electrode technique demonstrated that mitral regurgitation can occur with either improper timing of atrial systole or atrial fibrillation.

V. Hemodynamic Effects of Atrial Fibrillation in Man

The question of whether atrial fibrillation seriously impairs cardiac performance in man has been debated for years. The hemodynamic consequences of atrial fibrillation in both normal and diseased hearts have been reported by many investigators. Much of this work has been performed in only the resting state but some studies have included mild to moderate exercise stress.

53. Gossage, A. M. and Hicks, J. A. B.: On auricular fibrillation. Quart. J. Med. 6:435, 1912-13.

54. Parkinson, J. and Campbell, M.: Paroxysmal auricular fibrillation: a record of 200 patients. Quart. J. Med. 23:67, 1930.

55. Brill, I. C.: Auricular fibrillation with congestive failure and no other evidence of organic heart disease. Am. Heart J. 13:175, 1937.

56. Brill, I. C.: Congestive heart failure arising from uncontrolled auricular fibrillation in the otherwise normal heart. Am. J. Med. 2:544, 1947.

57. Brill, I. C., Rosenbaum, E. E. and Flanery, J. R.: Congestive failure due to auricular fibrillation in an otherwise normal heart. Report of a case with twenty-five year follow-up. J.A.M.A. 173:784, 1960.

58. Phillips, E. and Levine, S. A.: Auricular fibrillation without other evidence of heart disease: cause of reversible heart failure. Am. J. Med. 7:478, 1949.

The above studies report the occurrence of congestive heart failure in patients in which atrial fibrillation was the only

demonstrated abnormality. The most extensive study is that of Phillips and Levine who reported 84 such patients and concluded that "auricular fibrillation per se may produce cardiac dilatation and progressive congestive failure in patients with otherwise normal hearts."

59. Stewart, H. J., Deitrick, J. E., Crane, N. F. and Thompson, W. P.: Studies of the circulation in the presence of abnormal cardiac rhythms. Observations relating to (Part I) rhythms associated with rapid ventricular rate and to (Part II) rhythms associated with slow ventricular rate. J. Clin. Invest. 17:449, 1938.

The cardiac output at rest was less in 6 out of 7 patients during atrial fibrillation than after the reversion to normal sinus rhythm. Further the venous pressure was elevated during arrhythmia and fell to normal when sinus rhythm occurred. However, other studies have found no change in the resting cardiac output when conversion was accomplished but an increase in the output during exercise.

60. Kory, R. C. and Meneely, G. R.: Cardiac output in auricular fibrillation with observations on the effects of conversion to normal sinus rhythm. J. Clin. Invest. 30:653, 1951.

The circulatory dynamics of eight patients with atrial fibrillation were studied by cardiac catheterization before and after conversion to normal sinus rhythm with quinidine. Before conversion the resting cardiac output was low and the cardiac output response to exercise was considerably below normal. After conversion the six patients with organic heart disease had an average increase of 43 per cent in both the resting and exercise cardiac outputs. In the two patients with no heart disease, there was no significant change in cardiac output following conversion. It is concluded that conversion to sinus rhythm improves cardiac function in those patients with atrial fibrillation who have organic heart disease.

61. Hecht, H. H., Osher, W. J. and Samuels, A. J.: Cardiovascular adjustments in subjects with organic heart disease before and after conversion of atrial fibrillation to normal sinus rhythm. J. Clin. Invest. 30:647, 1951.

Respiratory and cardiac function were studied at rest and during exercise in 15 patients before and after conversion of atrial fibrillation to normal sinus rhythm by quinidine. The average resting cardiac output was below normal during atrial fibrillation and was changed little by conversion. However, the average cardiac output during exercise improved significantly after conversion. It was concluded that "a coordinate contraction of the cardiac atria aids in cardiac filling during demand periods and that this reserve function is lost in atrial fibrillation."

62. Hecht, H. H. and Lange, R. L.: The hemodynamic consequences of atrial fibrillation. Mod. Con. Cardiovas. Dis. 25:351, 1956.

This later report by Hecht and Lange gave further data on two patients before and after conversion with quinidine. One of these showed a very slight improvement of the cardiac output response to exercise, and the other a marked improvement.

63. Hansen, W. R., McClendon, R. L. and Kinsman, J. M.: Auricular fibrillation. Hemodynamic studies before and after conversion with quinidine. Am. Heart. J. 44:499, 1952.

Right heart catheterization was performed in patients before and after conversion with quinidine. Studies were conducted both at rest and during leg exercise in the supine position. Cardiac output at rest increased significantly in 9 out of 14 patients. During exercise a significant increase in cardiac output occurred in 8 out of 12 patients. These authors state that "the contention that atrial systole plays only a minor role in cardiocirculatory function does not seem tenable in view of these results. After restoration of sinus rhythm the increased rate of blood flow seems to be a constant finding."

64. Broch, O. J. and Müller, O.: Haemodynamic studies during auricular fibrillation and after restoration of sinus rhythm. Brit. Heart J. 19:222, 1957.

These authors also studied a group of patients with right heart catheterization before and after restoration of normal rhythm by treatment with quinidine. Their results are as follows:

	no.	mean		p
		AF	SR	
Cardiac output (l/min)				
Rest	17	5.6	6.2	.01
Exercise	11	6.8	8.1	.001
Stroke volume (ml)				
Rest	18	66	78	.02
Exercise	9	51	69	.15
O_2 consumption (ml/min)				
Rest	19	257	269	.20
Exercise	13	527	537	.80
O_2 saturation (pulmonary artery)				
Rest	19	71.8	73.6	.02
Exercise	16	57.6	60.9	.03
Arteriovenous O_2 difference				
Rest	20	4.9	4.2	.01
Exercise	20	7.4	6.3	.01

65. Gilbert, R., Eich, R. H., Smulyan, H., Keighley, J. and Auchincloss, J. H., Jr.: Effect on circulation of conversion of atrial fibrillation to sinus rhythm. Circulation 27:1079, 1963.

In this study treadmill exercise tests were performed in cardiac subjects before and after conversion from atrial fibrillation to normal sinus rhythm with quinidine. During exercise there was marked reduction in heart rate, increase in cardiac output and increase in stroke volume after conversion. The authors conclude that "conversion from atrial fibrillation to sinus rhythm is worthwhile from a functional standpoint in patients with valvular heart disease..."

66. Graettinger, J. S., Carleton, R. A. and Muenster, J. J.: Circulatory consequences of changes in cardiac rhythm produced in patients by transthoracic direct-current shock. J. Clin. Invest. 42:938, 1963. Abstract.

67. Morris, J. J., Jr., Entman, M. L., Thompson, H. K., Jr., North, W. C. and McIntosh, H. D.: Cardiac output in atrial fibrillation and sinus rhythm (P). Circulation 28:772, 1963.

In these two recent studies the cardiac output at rest and during exercise was determined in a group of patients before and after conversion with DC countershock. Graettinger et al. found no significant change either at rest or during exercise. Morris et al. studied the cardiac outputs of 11 patients at rest before, and after "cardioversion". Seven of these showed a mean increase of 26 per cent, three showed no change and one showed a decrease of 11 per cent. Five patients were exercised to the same level of O_2 consumption (about 1 l/min) before and after "cardioversion". All showed an increase in cardiac output of 10 to 30 per cent, with a mean increase of 18 per cent. It is also of interest that after conversion to normal sinus rhythm these five subjects had a lower arteriovenous oxygen difference during exercise at the same level of oxygen intake.

VI. Conclusions

Two recent advances in cardiac therapy have in common the return of a properly timed atrial systole. These are (1) the treatment of atrial fibrillation by the use of direct current countershock and (2) the treatment of complete heart block by a surgically implanted pacing electrode on the ventricle which is excited by atrial depolarization. It is important, therefore, to understand the role of the atrium in cardiovascular dynamics.

The role of the atrium in cardiovascular dynamics has been outlined and data presented to substantiate each of its functions. From this it now seems clear that the atrium makes a definite contribution to the general adaptability of the cardiac output. It seems probable that when the normal heart is deprived of atrial activity, numerous compensatory mechanisms can maintain the cardiac output at rest and during moderate stress such as mild muscular exercise, but the response to severe stress is abnormal. When

the heart with a slight to moderate decrease in function is deprived of atrial activity, the compensatory mechanisms may or may not maintain the cardiac output at rest but usually are not able to adequately increase the cardiac output during mild to moderate exercise. When the heart with a marked decrease in function is deprived of atrial activity, the compensatory mechanisms are unable to maintain the cardiac output even at rest.

Recently, controlled human studies on the quantitative effect of the restoration of atrial function in cardiovascular dynamics have appeared in the literature. In general, these studies have demonstrated the predicted improvement in cardiac output response. A final answer awaits further study.