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Movements of Human Diaphragm During Cardiac Cycle in Respiratory Pause.

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Diaphragmatic "tug," accompanying ventricular systole in man, has been recognized for many years (*cf., e.g.,* Mackenzie,[1]). It has not been analyzed but it has been stated that it "can produce but little movement upward because of the inertia of the heavy abdominal organs." (Hamilton.[2])

By means of the multiple slit roentgenkymograph of Stumpf[3] the component of motion in any given plane of moving boundaries between regions of differing radiographic density can be accurately determined for intervals of time down to about 0.03 second. We

1 Mackenzie, J., *Diseases of the Heart,* 1913, 2nd ed., London.

2 Hamilton, W. F., *Am. J. Physiol.,* 1930, **91**, 712.

3 Stumpf, P., *Fortsch. Röntgenstr.,* 1928, **36**, 3.

have used this procedure for measuring the size change and stroke output of the heart during a single cardiac cycle (Keys and Friedell[4]).

Stumpf[5] noted a cephalad movement of the diaphragm during systole and also stated that this movement may be reversed in the more lateral region of the diaphragm. We have studied a large number of roentgenkymograms (R.K.G.s) to get quantitative measures of these movements as recorded in posterior-anterior, lateral, and oblique views during respiratory pause, in all of which we have made both vertical and horizontal R.K.G.s at a film to target distance of 66 inches. Mathematical analysis of the measurements from tracings on these R.K.G.s was made by regarding the diaphragm as a frustrum of a dome of elliptical section. Rather than assume exact conformity to a simple geometrical form, the displacements recorded were integrated over a large number of steps.

Fig. 1 reproduces the tracing of the horizontal component of motion of both heart and diaphragm in the p.a. view in a typical case (normal young man 5 minutes after moderate exercise). Here, as is generally

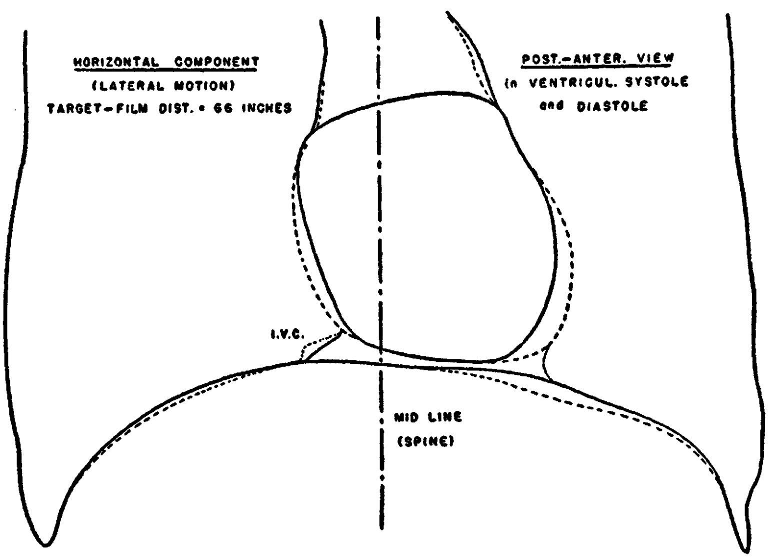

FIG. 1.

Horizontal component of motion of the heart and diaphragm, posterior-anterior view. Exact tracing of position of boundaries at full left ventricular diastole and the next succeeding systole during respiratory pause (moderate inspiration). Normal young man.

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[4] Keys, A., and Friedell, H. L., PROC. SOC. EXP. BIOL. AND MED., 1938, **40**, 267; *Am. J. Physiol.*, 1939, **126**, 741.

[5] Stumpf, P., Weber, H. H., and Weltz, G. A., *Röntgenkymographische Bewegungslehre innerer Organe*, 1936, G. Thieme, Leipzig.

the case, both right and left diaphragms moved laterally in systole and this motion is greatest (maximum 15.5 mm) on the left side near the apex of the heart The vertical component of motion in the p.a. view in this case showed cephalad motion of the left and central diaphragm, greatest near the apex (maximum 4.5 mm), but with apparent paradoxical motion of most of the right diaphragm (maximum 3 mm). This again is generally the case, as is the appearance of paradoxical motion in the posterior portion of the central and left diaphragm when studied in the lateral R.K.G.

The total net motion of the diaphragm is always such as to reduce the volume of the thorax during systole. Since the 4 films necessary cannot be made simultaneously, exact values cannot be given but in the young man whose R.K.G. tracing is shown here (Fig. 1), the total net volume change of the thorax computed from the p.a. and lateral R.K.G.s was about 50 cc, of which approximately 80% was the resultant of lateral (horizontal) motion and 20% was pure vertical elevation of the diaphragm. The small value for the net vertical component results from the paradoxical vertical motion of the diaphragm. In this illustrative case the mean stroke output (left ventricle) was about 70 cc from our method of calculation (Keys and Friedell[3]). The volume reduction of the thorax during the cardiac cycle always corresponds to at least a large fraction of the stroke output. These observations on the diaphragm lend further support to our contention that the total heart size in man is markedly reduced in systole and that this reduction is closely related to the stroke output.

We have also studied 8 cases of pneumoperitoneum by these methods. In these cases the viscera are removed from the peritoneum by 5 to 15 cm and not only the free diaphragm but also the whole of the caudal portion of the heart are made sharply visible. The results are in full agreement with our observations on normal individuals where the heart and diaphragm are less fully visualized.

It should be noted that these diaphragmatic and cardiac movements in man are not necessarily identical with those in animals (Hamilton and Rompf[6]) which do not maintain the erect posture and which possess a different architecture about the heart (absence of rigid mediastinum, presence of a frenulum attachment to the apex of the heart, etc.). In man, alteration of posture or of the respiratory phase changes the form of the movement. For example, the movements of the diaphragm are more difficult to visualize in expiration and they tend to disappear in the Valsalva experiment. In all cases the total thoracic volume tends to diminish in systole by an amount

[6] Hamilton, W. F., and Rompf, J. H., *Am. J. Physiol.*, 1932, **102**, 559.

which appears to correspond to a large fraction of the systolic discharge. During respiration these passive movements are obscured by the active movements of the diaphragm but if the stroke volume is large and respiration is very quiet traces may still be visible in the R.K.G.

Summary. In man during respiratory pause there are changes in the position of the diaphragm during the cardiac cycle so that in ventricular systole the total thoracic volume is smaller than in diastole. Calculation of the net thoracic changes involved integration of measurements of both vertical and horizontal components of motion in posterior-anterior and lateral projections. The reduction in thoracic volume amounts to a large and apparently rather constant fraction of the cardiac stroke output.

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Production of Bradycardia in Normal Man by Neosynephrin* (l-α-hydroxy-β-methylamino-3-hydroxy-ethylbenzene hydrochloride).

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It is well known that heart rate and blood pressure do not rise in equivalent degree after administration of different sympathomimetic drugs. Ethylnor-suprarenin (3-4 dihydroxy-phenyl-1-amino-2-butanol-1) produces a rise in pulse rate and a fall in blood pressure (Cameron, *et al.*[1]). Neosynephrin (3-hydroxyphenyl-1-methylamino-2-ethanol-1) increases the blood pressure with a relative fall in the pulse rate (Johnson[2]).

The production of relative bradycardia by sympathomimetic drugs has generally been ascribed to reflexes produced by the elevated blood pressure arising in the aortic arch and the carotid sinus. Such an effect can be demonstrated in man when small doses of epinephrine are used; the heart rate and blood pressure rise together but after some minutes the rate may fall while the blood pressure is still

* This work has been supported by a fellowship grant to the University of Minnesota by Frederick Stearns and Company.

1 Cameron, W. M., Crismon, J. M., Whitsell, L. J., and Tainter, M. L., *J. Pharm.*, 1937, **62**, 318.

2 Johnson, C. A., *Surgery, Gyn., Obst.*, 1936, **63**, 35.