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Effect of Exertion on Vital Capacity of Normal Subjects.

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Numerous studies have been made of the physiology of exercise, but comparatively little attention has been directed to the immediate effect of severe exertion on the vital capacity of normal subjects. Changes in the lungs, including reduction in vital capacity, are the rule in the dyspnea of congestive heart failure. Whether or not similar changes in the lungs occur in association with dyspnea due to severe exertion in normal subjects has not yet been established.

During the last century it was generally believed that dyspnea of exertion in healthy persons might be related to pulmonary engorgement. More recently, Bohr[1] and Gordon, Levine and Wilmaers[2] have mentioned this possibility. Available reports give conflicting data. Bohr[1] found that muscular activity, including running and work done while sitting and standing, decreased the vital capacity. Levine and Wilson[3] reported insignificant changes in the vital capacity of normal subjects who ran up and down stairs until breathless. Joannides[4] found that vital capacity was decreased in most of his normal subjects after exercising until dyspnea occurred. Gordon, *et al.*,[2] found definite diminution of the vital capacity of most of the runners after a 25-mile race. Harrison, Harrison, Calhoun and Marsh[5] observed little change in the vital capacity of normal subjects and patients with heart disease after mild exertion; their data are difficult to interpret since the nature and severity of the exercise were not noted. Because of these conflicting results and the variability in the conditions under which these studies were undertaken, the following investigation was made.

The vital capacity of untrained healthy adults was measured before and within 30 seconds after the ascent and descent of 4 flights of stairs as rapidly as possible. This exertion was sufficient to produce severe dyspnea which lasted for several minutes in all the sub-

1 Bohr, C., *Deutsches Arch. f. klin. Med.*, 1907, **88**, 385.

2 Gordon, B., Levine, S. A., and Wilmaers, A., *Arch. Int. Med.*, 1924, **33**, 425.

3 Levine, S. A., and Wilson, F. N., *Heart*, 1919, **7**, 53.

4 Joannides, M., *Arch. Int. Med.*, 1924, **33**, 145.

5 Harrison, T. R., Harrison, W. G., Calhoun, J. A., and Marsh, J. P., *Arch. Int. Med.*, 1932, **50**, 690.

jects. All measurements were made in standing posture with the ordinary bell type of spirometer.

In 12 of the 15 subjects the vital capacity was slightly lower after exercise (Table I). No change of more than 4.7% occurred; the average change was less than 2%.

TABLE I.
Effect on Vital Capacity of Running Up and Down Stairs.

	Vital capacity in liters		Change after exercise	
Subject	Before exercise	After exercise	Liters	% of original value
1	5.40	5.30	—0.10	—1.9
2	4.45	4.40	—0.05	—1.1
3	6.50	6.50	0	0
4	3.93	3.82	—0.11	—2.8
5	4.22	4.23	+0.01	+0.2
6	4.19	4.05	—0.14	—3.3
7	4.92	4.82	—0.10	—2.0
8	5.42	5.30	—0.12	—2.2
9	5.24	5.01	—0.23	—4.4
10	2.70	2.68	—0.02	—0.7
11	4.12	4.04	—0.08	—1.9
12	2.58	2.46	—0.12	—4.7
13	2.90	2.82	—0.08	—2.8
14	5.03	5.10	+0.07	+1.4
15	3.78	3.65	—0.13	—3.5

The slight changes found in the vital capacity of normal subjects after exercise are within the limits of error of the measurement. However, so many of the subjects showed changes in the same direction that it might be argued that the observed decreases are significant. The slight decreases are to be explained by the distress and discomfort associated with dyspnea. It is concluded, therefore, that no significant change in the vital capacity of normal subjects occurs after exercise which is strenuous enough to produce severe dyspnea. This is in accord with the findings of Levine and Wilson.[3]

The dyspnea of patients wth congestive heart failure is associated with a decrease in vital capacity which has been explained on the basis of congestion of the pulmonary circuit;[6] pulmonary congestion does not appear to be a factor in the dyspnea of exertion of normal persons. Confirmatory evidence as to the absence of pulmonary congestion in the dyspnea of exertion in normal subjects is the fact that the arterial blood oxygen saturation is not decreased after exercise severe enough to cause dyspnea in such subjects.[7, 8]

[6] Drinker, C. K., Peabody, F. W., and Blumgart, H. L., *J. Exp. Med.*, 1922, **35**, 77.

[7] Himwich, H. E., and Barr, D. P., *J. Biol. Chem.*, 1923, **57**, 363.

[8] Cullen, G. E., Harrison, T. R., Calhoun, J. A., Wilkins, W. E., and Tims, M. M., *J. Clin. Inv.*, 1931, **10**, 807.

Summary. The vital capacity of normal subjects is not diminished by exercise severe enough to cause marked dyspnea. It is concluded that the dyspnea of exertion of normal subjects is not due to impaired pulmonary function consequent to congestion of the lungs.

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The Urinary Pigments in Four Cases of Alcoholic Pellagra.

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Beckh, Ellinger and Spies[1] employed a very simple procedure for detecting and estimating porphyrin in the urine in 20 cases of pellagra. This consisted of extraction of the acidified urine with ether, after which the ether was extracted with a small amount of 25% HCl. The presence of a red or red violet color in this solution was considered due to porphyrin, and was estimated colorimetrically by comparison with a standard porphyrin solution.

In the present investigation the results of the B.E.S. (Beckh-Ellinger-Spies) test have been compared with the values for coproporphyrin obtained by means of a slight modification of the Fikentscher[2] method (estimation of red fluorescence of coproporphyrin in ultraviolet light, with the Zeiss stufenphotometer). Quantitative determinations were made in 3 of 4 cases of typical alcoholic pellagra.* In the first case but a single 24-hour urine sample was available prior to nicotinic acid therapy; this was examined qualitatively by the usual acetic and ether, followed by 5% HCl, extractions. The amount of porphyrin did not appear to be appreciably increased.

In a series of 5 normal individuals the range of coproporphyrin with the quantitative method employed, was 16-62.8γ per day. In the 3 pellagra cases in whom determinations were made, the following results were obtained:

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1 Beckh, W., Ellinger, P., and Spies, T. D., *Quart. J. Med.*, 1937, **30**, 305.

2 Fikentscher, R., *Biochem. Z.*, 1932, **149**, 257.

* Two of these cases were on the medical service of the Johns Hopkins Hospital, Baltimore, and 2 were on the medical service of the Minneapolis General Hospital. The writer is indebted to Drs. Warfield Longcope and George Fahr, respectively, for access to the clinical data and urines of these cases.