tinuous, fasting, 36-hour secretion; representative studies with the active extract and the control material are shown in Fig. 1 and 2. The hourly volume, free acidity, and output of hydrochloric acid remained essentially unchanged. No untoward effects were observed in these studies.

Conclusion. The intragastric administration of an extract of pregnant mares' urine, Kutrol, in quantities as large as 7.50 g does not decrease the volume, free acidity, or output of hydrochloric acid in the 12-hour nocturnal gastric secretion or in the continuous 36-hour secretion of patients with peptic ulcer.

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Effect of Forced Expiration upon Finger and Pulse Volume.*

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(Introduced by W. S. McCann.)

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Although there is general agreement about the peripheral arteriolar constrictive effect of a deep inspiration, the effect of a deep expiration upon peripheral circulation is not yet clarified. Bolton, Carmichael, and Sturup[1] and Gilliat[2] report no effect on finger volume, whereas Uhlenbruck[3] and de Lalla[4] observed a diminution of finger volume, and the latter reported coincident decrease in skin temperature and arterial rate of flow following a forced expiration. Therefore it seemed of interest to study further the possible changes in finger and pulse volume incident to a deep expiration.

Method. Pulse and finger volumes were recorded in two ways using a finger plethysmograph each time: (1) In 11 cases the plethysmograph changes were picked up by a piezo-electric crystal whose output was fed into an amplifier-galvanometer recording system. The deflections of the mirror galvanometer were recorded on film moving at 25 mm per second; in 4 of these tracings a simultaneous ballistocardiogram was recorded. With this method any desired amplification of the pulse volume could be obtained, but the total finger volume changes could not be recorded since the system would not maintain a change of baseline; (2) In 5 cases the output of the plethysmograph was put directly into a Frank capsule whose mirror deflections were recorded in the same manner as above. Although the pulse impulse waves were not large in amplitude with this technic, the total finger volume changes were accurately recorded. Respirations were recorded simultaneously in all cases by a pneumograph-Frank capsule arrangement.

Six normal adult subjects were used, 4 males and 2 females, and a total of 16 records were obtained. They were instructed in the following manner: "At no time take a deep inspiration; at the end of a normal inspiration expire forcefully and rapidly, as much as you can, through your mouth; do not hold your breath, but continue to breathe normally directly afterwards." This maneuver is described in detail since the manner of performance seems to be important.

Results. In all cases there was an immediate decrease in pulse volume, and in the 5 tracings recorded with the Frank capsule there was a simultaneous fall in total finger

* This work was carried out under a contract between the Office of Naval Research and the University of Rochester, School of Medicine and Dentistry.

1 Bolton, B., Carmichael, E. A., and Sturup, G., *J. Phys.*, 1946, **86**, 83.

2 Gilliatt, R. W., *J. Physiol.*, 1948, **107**, 76.

3 Uhlenbruck, P., *Z. Biol.*, 1924, **80**, 317.

4 de Lalla, V., *Am. J. Physiol.*, 1948, **152**, 122.

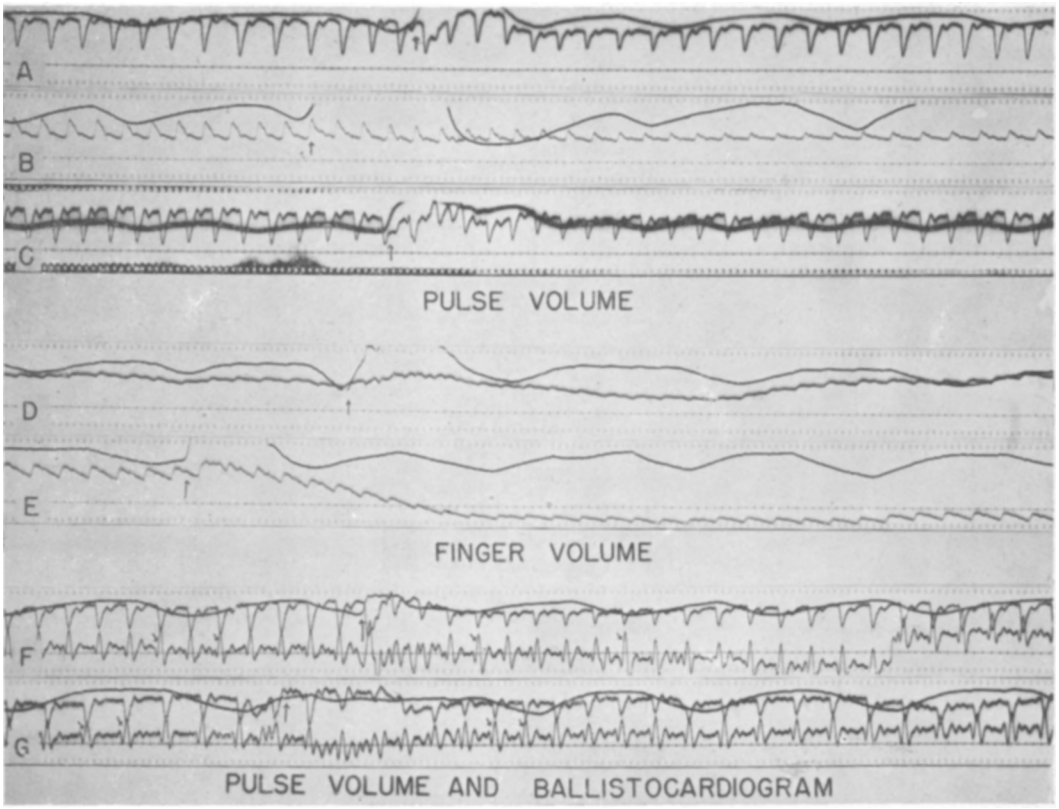

FIG. 1.

Seven records showing the effect of a forced expiration on pulse and finger volume. Respirations are recorded as the top undulating line, with expiration being an upward deflection. The time line is dotted: two-tenths of a second between large dots.

A, B, C are records of finger pulse volume taken with a piezo-electric crystal and do not show baseline changes (see text). Note the immediate decrease in amplitude of the pulse volume stroke in all cases immediately following a deep expiration (arrows). The recovery in B occurred in 16 seconds and is not shown. See Fig. 2 for graphs.

D and E were taken with a Frank capsule, and recorded total finger volume changes as well as individual pulse volumes. Note the fall in total finger volume and the decrease in pulse volume following a deep expiration (arrows). The recovery in E occurred within 30 seconds and is not shown.

F and G are records of finger pulse volume taken as in A with a simultaneous ballistocardiogram recorded below. Note that although the pulse volume diminishes directly after a deep expiration (large arrow), the IJ stroke of the ballistocardiogram (small arrows) does not. See Fig. 3 for graphs and text for interpretation.

volume (Fig. 1, 2). The average duration of effect was 16 seconds for diminished pulse volume and 24 seconds for the diminution of total finger volume. The individual response of pulse volume change can be determined from Fig. 2. The individual duration of total finger volume diminution varied from 10 to 40 seconds.

In the 4 cases where the ballistocardiogram was recorded with the pulse volume, the height of the IJ stroke of the ballistocardiogram in millimeters and the height of the pulse volume stroke in millimeters was averaged for the control period in each case. The deviation from the respective control average of the height of each IJ stroke and pulse volume stroke in millimeters was plotted against the corresponding beat. These correlation graphs are shown in Fig. 3 and representative tracings are given in Fig. 1F and G. In no case was the total stroke volume decreased during or after the deep expiration, whereas the pulse volume had immediately decreased, and remained so even

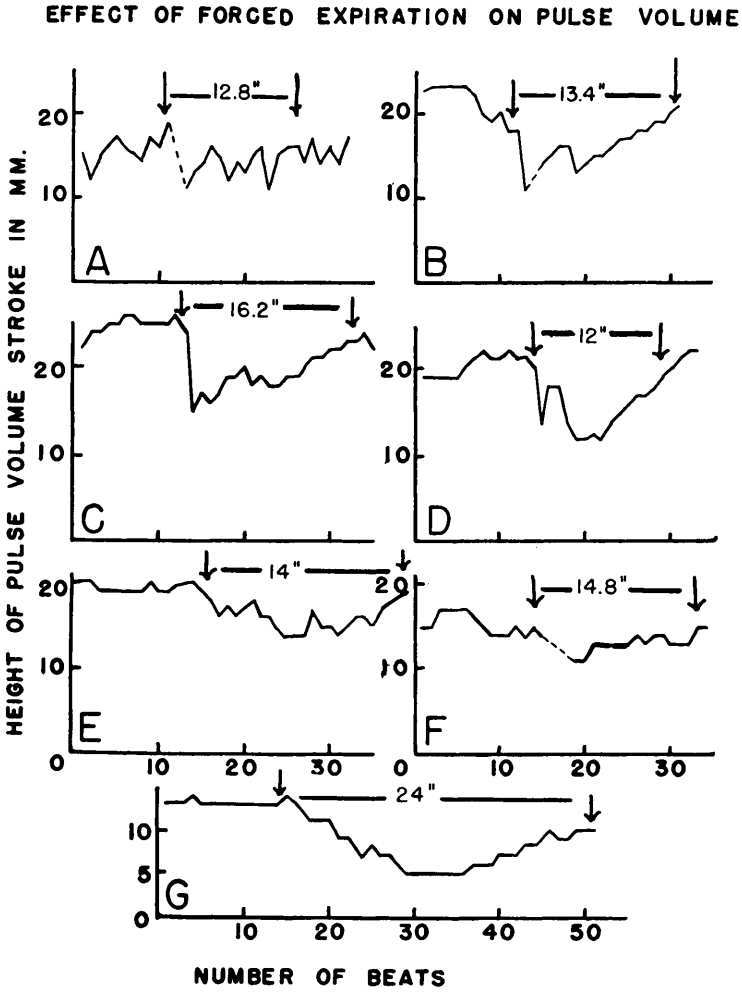

FIG. 2.

Graphs of the effect of a deep expiration on finger pulse volume in 7 cases. The height of the pulse volume stroke (recorded as in Fig. 1A) measured in millimeters is plotted against the corresponding beat. Note the immediate drop in pulse volume in all cases following a deep expiration (first arrow). The recovery time in seconds is indicated between the first and second arrows, in each case. The dotted lines in A, B, and F represent strokes which were not measurable because of artifacts caused by the motion of the subject.

though the corresponding ballistocardiogram beat was not smaller than a comparable control beat.

Discussion. A deep, forced expiration performed in the manner described produced a decrease in both total finger volume and purse volume, in the 6 subjects studied. This confirms the reports of Uhlenbruck and de Lalla.

By using the height of the IJ stroke as a measure of relative stroke volume[5,6] the simultaneous ballistocardiogram may be interpreted as confirmatory evidence for reflex vasoconstriction since the only other probable cause for the observed diminution of pulse and total finger volume (*i.e.* diminished left ven-

[5] Starr, I., Rawson, A., Schroeder, H., Joseph, N., *Am. J. Physiol.*, 1939, **127**, 1.

[6] Brown, H. R., Jr., and de Lalla, V., Jr., Unpublished data.

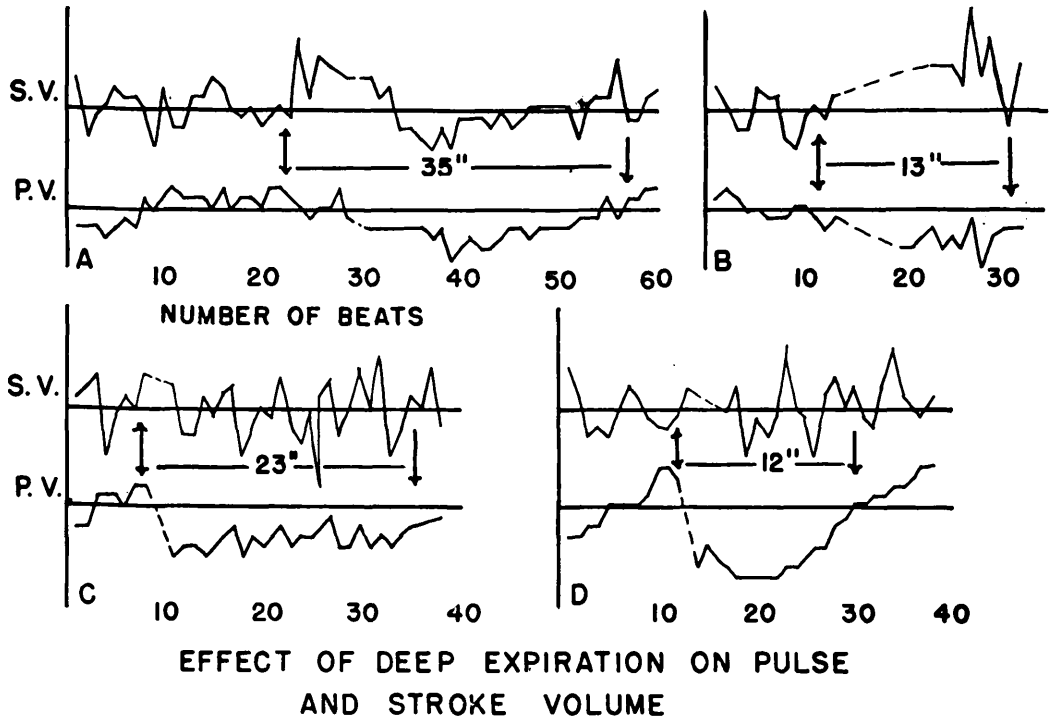

FIG. 3.

Four graphs of the effect of a deep expiration on the height of the finger pulse volume stroke and on the height of the IJ stroke of the ballistocardiogram (relative total stroke volume). The deviation in millimeters from the control average height of pulse volume and IJ stroke in millimeters is plotted against the corresponding beat in each case. Note that although the pulse volume curve (P.V.) drops after a deep expiration (first arrow), the relative stroke volume curve (S.V.) does not change significantly. The pulse volume recovery time is indicated in seconds between the first and second arrows in each case. The large swings of the S.V. curve reflect the respiratory variation of the total stroke volume. See Fig. 1F and G for representative tracings. The dotted lines represent strokes which were not measurable because of artifacts caused by the motion of the subject.

tricular output) does not seem to have occurred. (See Fig. 1F, G and 3). Since the IJ stroke reflects total stroke volume, and since the right ventricular stroke volume is known to decrease in expiration[6-8], then one might expect to find smaller IJ strokes during or after the forced expiration than in comparable control beats, if the left ventricular stroke volume had diminished or remained the same. That this was not observed indicates that the left heart stroke volume was increased during the forced expiration at the same time that the pulse volume was observed to have decreased. This deduction is sound in view of the report of Shuler *et al.*, which states that the left ventricular stroke volume increases with expiration.[7] These findings suggest that a reflex vasoconstriction is the cause of the diminution of pulse and total finger volume incident to a forced expiration.

One of us had already postulated that stretch receptors in the large extrathoracic veins may be the origin of this reflex.[4] However, it is not necessary that these receptors be located in the large veins themselves in order to respond to increased abdominal venous pressure. Gammon and Bronk[9] have demonstrated Pacinian corpuscles in the

[7] Shuler, R., Ensor, C., Gunning, R., and Moss, W., *Am. J. Physiol.*, 1942, **137**, 602.

[8] Lauson, H. D., Bloomfield, R. A., and Cournand, A., *Am. J. Med.*, 1946, **1**, 315.

[9] Gammon, G. D., and Bronk, D. W., PROC. SOC. EXP. BIOL. AND MED., 1934, **31**, 788.

mesentery which respond to engorgement of the neighboring tissue by raising the systemic blood pressure (*i.e.* arterial vasoconstriction). They also showed that these receptors would respond in the same manner to increased pressure in the abdominal vena cava. It is possible that these receptors are the initiators of the observed reflex.

Should this be true, then it is understandable why the expiration may not produce diminution of pulse and finger volume if not forced. It is likely that a critical intrathoracic pressure must be reached before the abdominal venous pressure is raised enough to stimulate the receptors. In that event, a deep expiration that does not do this will not elicit the reflex.

Summary. 1. Twelve records of the effect of a deep, forced expiration on finger pulse volume were obtained from six subjects. In four of these records a ballistocardiogram was recorded simultaneously. In 5 other tracings total finger volume changes were recorded.

2. In all cases the pulse volume and total finger volume (when recorded) decreased immediately following the deep forced expiration. Average duration of diminution was 16 seconds for pulse volume and 24 seconds for total finger volume.

3. The absence of a simultaneous decrease in corresponding ballistocardiogram beats suggests that the observed finger changes were due to reflex arteriolar constriction, possibly initiated by abdominal presso-receptors in the great veins or mesentery.

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Location of Communications between Cognate Bed of Descending Ramus of Left Coronary and Adjacent Collateral Vascular Beds.*

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More than 40 years ago, Hirsch and Spalteholz[1] noticed that after ligation of a coronary artery, the area of infarction was much smaller than the myocardium apparently supplied by that artery (cognate bed). Two years later Miller and Mathews[2] made the same observations, and suggested that this was due to the presence of fine epicardial arterial anastomoses. Since that time, numerous additional reports have appeared describing the variability in the occurrence of and in the size of infarcts produced by coronary artery ligation.[3-8] Sutton and Davis[9] suggested tying the superficial anastomotic vessels in order to get a large infarct.

No actual studies, however, appear to have been done in order to demonstrate whether or not the blood supplied by the superficial

* Supported by a grant from the Life Insurance Medical Research Fund.

1 Hirsch, C., and Spalteholz, W., quoted by L. Gross, 1921, Hoeber, N. Y.

2 Miller, J. L., and Mathews, S. A., *Arch. Int. Med.*, 1909, **3**, 476.

3 Smith, F. M., *Arch. Int. Med.*, 1918, **22**, 8.

4 Mendlowitz, M., Schauer, G., and Gross, L., *Am. Heart J.*, 1937, **13**, 663.

5 Karsner, H. T., and Dwyer, J. F., *J. Med. Research*, 1916, **34**, 21.

6 Smith, F. M., *Am. J. Med. Sc.*, 1918, **156**, 706.

7 Gold, H., Travell, J., and Modell, W., *Am. Heart J.*, 1937, **14**, 284.

8 O'Neill, J. F., and Thomas, W. C., Personal communications, 1947.

9 Sutton, D. C., and Davis, M. D., *Arch. Int. Med.*, 1931, **48**, 1118.