6376

Studies in Venous Pressure. III. Effect of Gradually Increased Compression of Neck on Curve of Venous Pressure in Occluded Veins.*

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Eyster,[1, 4] Villaret, Girons, Bosveil,[2] Clark,[3] Eyster and Middleton,[5] and Lemierre and Bernard[6] among others have noted the elevation of venous pressure in congestive heart failure, which they report is of serious prognostic significance when it persists. Eyster[1, 4] concluded that an elevated venous pressure may point to impending cardiac decompensation, and that a sustained rise of general venous pressure is met only in this condition. More recent work has shown that other factors affect the level of venous pressure in man. Brandt[7] points out that the arteriolar pressure, the tonus of the veins, the visceral pressure in the abdomen and, most important of all, the circulating blood volume, also affect venous pressure. He found occasional coexistence of congestive heart failure and normal venous pressure, particularly in patients with auricular fibrillation or emphysema. Kroetz[8] believes that venous pressure may be affected by capillary and arterial pressure and by the venopressor mechanism described by Henderson.[9] Fleisch[10] suggests the possibility of a venomotor center in the brain. Brandt and Katz[11] found venous and arterial hypertension to coexist in the same patient, apparently due to an increased venous tonus. Arnoldi,[12] and Rotky and

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1 Eyster, J. A. E., "The Clinical Aspects of Venous Pressure," Macmillan and Company, 1929, New York.

2 Villaret, M., Saint Girons, F., and Grellety-Bosveil, P., *Presse Med.*, 1923, **31**, 318.

3 Clark, A. H., *Arch. Int. Med.*, 1915, **16**, 587.

4 Eyster, J. A. E., *J. Am. Med. Assn.*, 1927, **89**, 428.

5 Eyster, J. A. E., and Middleton, W. S., *Am. J. Med. Sci.*, 1927, **174**, 486.

6 Lemierre, A., and Bernard, E., *Presse Med.*, 1926, **34**, 705.

7 Brandt, F., *Ztschr. f. Klin. Med.*, 1931, **116**, 398.

8 Kroetz, C., *Deut. Arch. f. Klin. Med.*, 1922, **139**, 325.

9 Henderson, Y., *J. Am. Med. Assn.*, 1931, **97**, 1265.

10 Fleisch, A., *Arch. f. d. ges Physiol.*, 1930, **225**, 26.

11 Brandt, F., and Katz, G., *Z. f. d. ges Exp. Med.*, 1931, **76**, 158.

12 Arnoldi, W., *Deut. Med. Wchnschr.*, 1920, **46**, 4.

Klein[13] found many cases of essential hypertension without evidence of cardiac failure with elevated venous pressure. Connet[14] found that merely slowing the normal heart may elevate venous pressure. Von Gonczy and Kiss[15] frequently found the venous pressure elevated during the menopause.

This study was undertaken to investigate further the correlation of the various factors controlling venous pressure. Dogs were used, under morphine and sodium barbital anesthesia. A tracheotomy tube was inserted just above the sternum; the external jugular vein and both carotids were exposed. Heparin was injected intravenously to prevent clotting. The external jugular vein was then severed high up in the neck and the horizontal arms of a short T tube inserted in each end. The perpendicular arm of the T tube was connected to an inflating rubber bulb and to a mercury manometer. A rubber cuff was then placed loosely between the T tube in the jugular vein and the tracheotomy tube, enclosing all the structures in the neck. Care was taken not to compress the jugular vein at this stage (checked by the venous pressure cephalad to the cuff).

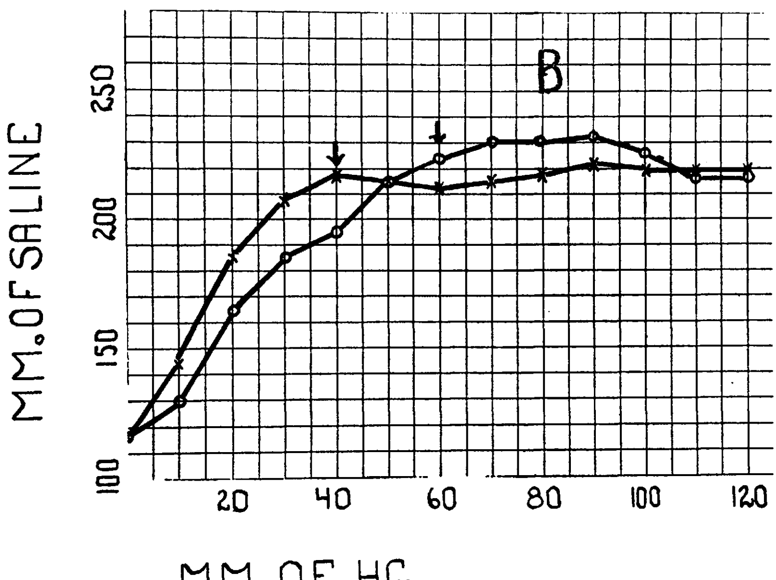

FIG. 1.

Curves of venous pressure of jugular vein following compression of neck. O—with carotid arteries exteriorized; X—with carotid arteries under compressing band.

[13] Rotky, H., and Klein, O., *Med. Klin.*, 1923, **19**, 1542.

[14] Connet, H., *Am. J. Physiol.*, 1920, **54**, 96.

[15] von Gonczy, V. I., and Kiss, J., *Z. f. d. ges Exp. Med.*, 1931, **78**, 398.

The control level of venous pressure in the jugular vein having been determined, the pressure in the rubber cuff was then increased at intervals of 10 mm. of mercury at a time. The venous pressure was read off as millimeters of saline, after the level had become stabilized. The same animals were then used in similar experiments but with both common carotids exteriorized (the section of cuff under these vessels was clamped off in order to prevent the possibility of kinking or deforming the carotids during its inflation). In a few experiments both vertebral arteries were ligated.

The pressure noted in the graph (Fig. 1), while measuring the pressure within the rubber cuff, probably does not represent the degree of actual compression of the neck. The cuff was comparatively narrow and the overlapping ends were not parallel to each other, to avoid the protruding tracheotomy tube. These factors interfered with the full force of compression as registered by the mercury manometer. Nevertheless, the arrangement did provide a means of producing gradually increasing pressure around the neck.

A study of the curves so obtained shows that the venous pressure rises at an almost constant rate with increasing compression of the neck until a certain point is reached. Further compression of the neck results in little or no further rise, *viz.,* B of Fig. 1.

The point at which the curve changed from an oblique to a nearly horizontal line did not occur at the same stage of compression in different animals. In the entire series of 17, the change in slope usually occurred when the pressure within the constricting collar registered between 30 and 60 mm. of mercury, but there were a number of exceptions. In one dog the change occurred at 180 mm. of mercury. The proximity of the external jugular vein to the constricting cuff was the chief cause of this variation. Only 30 mm. of mercury pressure was necessary when the jugular vein was widely exposed and in actual contact with the cuff.

The level of venous pressure at which the pressure curve changed its slope also varied in different animals. In 9 animals the point was reached when the venous pressure was between 125 and 155 mm. of saline; in 6 at 200 and 250 mm. of saline; and in 2 at 370 and 515 mm. of saline.

Similar curves of venous pressure were obtained on gradually increasing the compression of the neck when both carotids were exteriorized, and when both vertebrals were ligated.

The results show that gradually increasing compression of the neck, under the experimental conditions described, produced a uniform type of change in venous pressure. The curve at first showed a rise of almost constant slope and after a certain point rapidly became less steep—practically horizontal—during further compression. Neither the degree of compression as registered in these experiments nor the height of the resulting venous pressure formed definite criteria for the level at which the curve changed in direction; nor was the degree of patency of the carotids and vertebrals an important factor. The one important variable was the accessibility of the vein.

The pressure within the external jugular vein is normally due to a number of factors, as outlined in the introduction. These factors may be divided into 3 groups: (a) the force of the heart beat as transmitted through the capillaries, (b) the tonus of the veins and, (c) the volume of blood within the veins studied. During gradually increasing compression the veins are probably the first to become closed, since the venous pressure is much lower than either capillary or arterial. When the veins are completely obliterated the inflow through the capillaries is still maintained, though possibly to a reduced degree, with the result that the veins continue to be filled. As the venous pressure rises, however, more and more resistance is offered to filling until a point is reached where the venous pressure practically equals the pressure in the capillaries, so that little, if any, additional filling can occur. The pressure rise thereafter will be less steep. This corresponds to B of Fig. 1. The eventual occlusion of the capillaries, probably soon after, acts in the same way.

No studies were made on the effect of venous stagnation in the head during these experiments on the CO_2 content of the blood. But an accumulation of CO_2 in the stagnant blood might be partly responsible for the contour of the curve, by acting on the brain or locally on the external jugular vein.

Summary. 1. A series of experiments was performed in dogs to determine the effect of gradually increased compression of the neck on the venous pressure of the occluded veins. 2. When the compressing pressure is plotted against the resulting rise in venous pressure the curve shows, at first, a steep slope up to a certain point. Further compression produces an abrupt change in slope and the assumption of a more or less horizontal level regardless of the further degree of compression.

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