containing 1 mg. per cc. in ampules (Roche; N. N. R.) diluted freshly to a 1:1,000,000 solution with tap water. It seemed to be more toxic to the tadpoles, so that weaker concentrations were necessary. Otherwise, the results agreed entirely with those described above and hence need not be repeated.

Conclusions. 1. Toad tadpoles were found to metamorphose no more rapidly in up to lethal concentrations of dinitrophenol than in the absence of medication, whereas the usual acceleration of metamorphosis was demonstrated in tadpoles on thyroxin. This difference in action agrees with clinical differences in human subjects. 2. Although dinitrophenol can cause increases in metabolism comparable to those of thyroxin, it appears to lack the power of thyroxin to accelerate developmental processes. Apparently, the effect of thyroxin on metamorphosis is not the direct result of increased metabolism, but presumably an independent action. 3. Accordingly, dinitrophenol lacks the hormonal actions of thyroid, and should probably not be used therapeutically to replace the gland in true thyroid deficiencies.

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Effect of Arm Compression on Local Venous Pressure in Patients with Normal and Abnormal Hearts.*

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It is well known that interference with the flow in a vein will cause a rise in venous pressure within that vessel. Villaret, Saint-Girons and Justin-Besançon,[1] and others, have observed that mediastinal tumor or aortic aneurysm may lead to an elevation in venous pressure in one arm if its veins are obstructed. Runge[2] found a higher pressure in the saphenous vein than in the arm veins during pregnancy; a difference which disappeared after delivery. A similar difference was found by Villaret, Saint-Girons and Justin-Besançon[1] in cases of ascites or abdominal tumor. Brams, Katz and Kohn[3] noted a marked rise in pressure in the iliac veins with little or no change in the superior vena cava in experimentally induced abdominal disten-

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tion in animals. Brams and Katz[4] observed that slowly increased compression of a vein in animals produced a gradual rise in venous pressure up to a certain level, after which little or no further change occurred.

In this investigation the effect of experimental compression was studied in man to determine: (a) whether or not the results are the same in persons with normal and failing hearts; and (b) what relation the maximum rise in venous pressure bore to the systolic and diastolic arterial blood pressures.

Six subjects with normal hearts and normal blood pressures were selected as controls. Two patients with mediastinal tumor and 18 with varying degrees of cardiac failure comprised the group of abnormals. Cardiac failure in the latter was due to rheumatic fever, hypertension, cor pulmonale or arteriosclerosis. Venous pressure was measured by the direct method, using a hollow needle inserted into the median basilic vein after the patient rested under basal conditions for at least 15 minutes as described by Eyster. The needle was connected to a suitable mercury manometer since, in preliminary trials, it was found that compression of the vein produced a rise in venous pressure too great to be registered by an ordinary water manometer. The mercury manometer was found to be sufficiently sensitive for the needs of the present research. An ordinary sphygmomanometer cuff was used to compress the arm cephalad to the point where the needle was inserted into the veins. This was gradually inflated in steps and the pressure in the cuff and in the vein noted at each step.

A study of the results in the normal persons showed a progressive rise in venous pressure on increasing compression of the arm, similar to that seen in the previously reported animal experiments.[4] No change in the slope of the rise of venous pressure occurred when the compression force on the arm approached the diastolic arterial pressure. A definite change in the curve of venous pressure occurred in all instances when the compression force approached systolic blood pressure. Further compression, above the systolic pressure, resulted in a fall of venous pressure varying from 2 to 40 mm. of mercury. This last level was maintained practically constant on further compression of the arm.

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[1] Villaret, M., Saint-Girons, F., and Justin-Besançon, L., "La Pression Veineuse Périphérique," Masson and Cie, 1930, Paris.

[2] Runge, H., *Arch. f. Gynak.*, 1924, **122**, 142.

[3] Brams, W. A., Katz, L. N., and Kohn, L., *Am. J. Phys.*, 1933, **104**, 120.

[4] Brams, W. A., and Katz, L. N., PROC. SOC. EXP. BIOL. AND MED., 1932, **30**, 98.

The 2 patients with mediastinal tumor showed similar results. The 18 patients with varying degrees of cardiac failure reacted in the same manner, the fall in venous pressure ranging from 10 to 80 mm. of mercury when the compression force exceeded the systolic arterial pressure level.

The gradual rise in venous pressure with compression by the cuff was due to accumulation of blood in the vein peripheral to the point of obstruction. The abrupt fall in venous pressure when compression reached or slightly exceeded the systolic blood pressure can be explained by assuming that practically all the flow in the arteries beyond the point of compression was stopped. The contents in the distended veins could now regurgitate into their "proximal" capillaries and collapsed arteries since the pressure in the latter had fallen close to zero. Such regurgitation would continue until the pressure became equal in the veins and arteries below the cuff. The constant level of venous pressure after this drop is easily understood since all the vessels of the limb below the cuff are practically isolated from the rest of the body. The fact that no difference was observed in persons with normal and abnormal hearts can be explained by the fact that the previously described changes were due to local factors at the point of compression.

Summary. 1. A series of 26 observations were made in man to study the effect of gradually increased compression on the venous pressure below the point of compression. This group consisted of 6 normal persons serving as controls and an abnormal group of 2 with mediastinal tumor and 18 with cardiac failure. 2. Venous pressure gradually rose as compression was increased, until the latter reached the level of the systolic arterial pressure. At that point or on slightly further compression the venous pressure fell from 2 to 80 mm. of mercury and remained unchanged at that lower level regardless of the degree of further compression. 3. The progressive rise and the drop at the systolic pressure level were found in all the groups of patients regardless of the actual level of the systolic blood pressure or the condition of the heart. No change in the slope of the venous pressure rise was seen when the compression force approximated the diastolic blood pressure.