OBSERVATIONS ON THE OUTPUT OF THE HEART AND THE PRESSURE IN THE VEINS OF PREGNANT WOMEN.

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Many patients with heart disease pass through a stage when heart disease is clearly present, but during which it produces no symptoms. To prolong this asymptomatic stage and thus to prevent the onset of cardiac failure is an important part of the physician's task in managing heart disease. When one observes the factors which influence the progression of heart disease, it is clear that the activity of the causative disease process (e.g., acute rheumatic fever) is important. It is equally clear that this underlying disease process is not the only important influence on progression. There is a group of conditions which do not by themselves cause heart disease, but which often act to accelerate its course or to precipitate the onset of heart failure. These conditions include: physical or emotional strain, acute infections, obesity, anemia and pregnancy.

Pregnancy is frequent and temporary, and its termination is abrupt. It offers a favorable opportunity to study the effect of one of these secondary influences upon the circulation. This report deals with some of the alterations in the circulation which occur during the gravid state. The observations were made with the aid of three of my colleagues: Dr. W. David Strayhorn, Jr., Dr. Marvin B. Corlette and Dr. Don Flickinger.

The observations may be presented in three groups:

I. Normal young women in the early months of pregnancy were selected; their coöperation was enlisted; and they were trained as subjects. Observations were begun in the third and fourth months of pregnancy and were repeated at intervals of two to six weeks during pregnancy and also after delivery. During the months of pregnancy these patients exhibited certain physical signs: an increase in the heart rate; a forcible apex beat; loud heart sounds and systolic murmurs; a loud *bruit* was audible over the uterus; and the heart was displaced upward. The leg veins were distended, the neck veins

also appeared more prominent than normal. The abdomen was distended, but its wall was not tense. After delivery the signs of an overactive heart disappeared, the heart rate diminished, the heart assumed its usual position, and the veins, their usual degree of distention. During this same period observations were made on the output of heart by the acetylene method of Grollman.[1] This method was applied under the usual standard "basal" conditions. Four women were thus observed. Figure 1 summarizes the data concerning oxygen consumption, cardiac output and pulse rate in one patient. It is seen that the maximum increase in oxygen consumption is about 20%. At the same time the output of the heart increases by more than 50%; i.e., the output of the heart per minute increases out of proportion to the increase in the consumption of oxygen. This disproportion, of course, implies a fall in the arterio-venous difference; this difference in normal young people is usually from 50 to 60 c.c.; in this woman it falls to 35-50 c.c.

The four observations during the last month of pregnancy show a less marked disproportion. Post-partum studies show essentially normal figures. Two other patients exhibited changes very similar to these. In a fourth patient the increase in output was less, but the

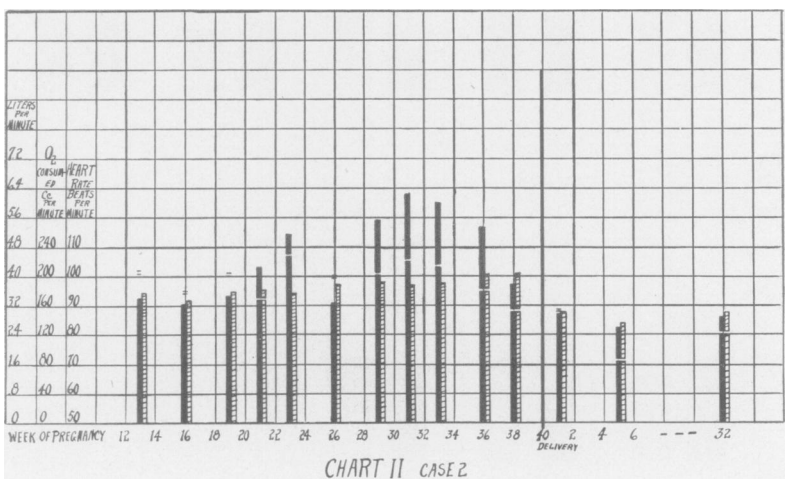

FIGURE 1.

The solid columns indicate the cardiac output in liters per minute; the barred columns, the total oxygen consumption in c.c. per minute.

sequence of changes was similar. This patient had no unusual symptoms or signs.

II. The second group of observations deals with venous pressure in the arms and legs of pregnant women and extends the early observations of Runge.[3]

The venous pressure was determined by the direct method of Moritz and Tabora[2] in the ante-cubital and femoral veins. These studies were made a few weeks before term and again some weeks after delivery. Figure 2 summarizes the results obtained on nine patients. The arm venous pressures are occasionally above the normal limits; the pressure in the femoral vein is invariably higher than normal, and also higher than the corresponding arm pressure. After delivery, both arm and leg pressures are restored essentially to normal.

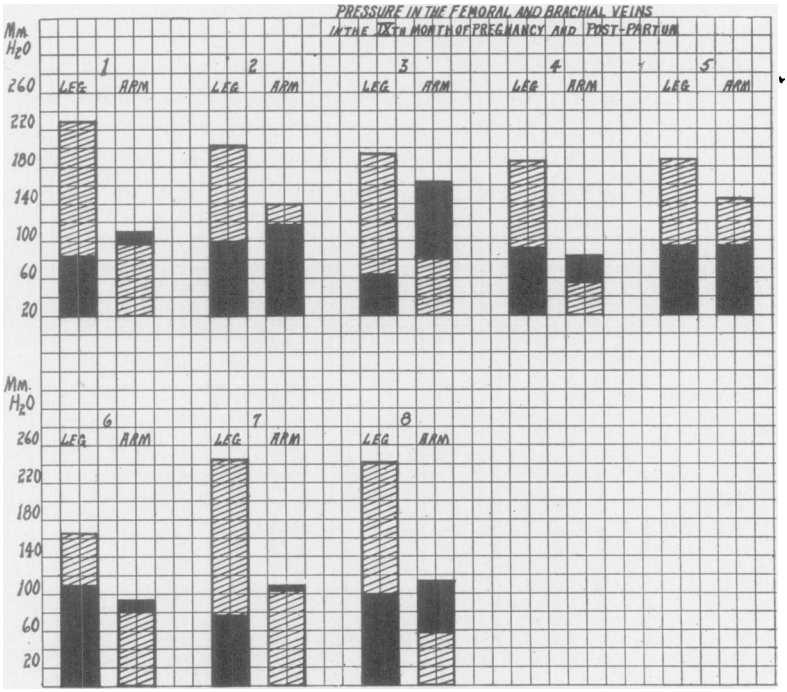

FIGURE 2.

The solid portions of the columns indicate post-partum values; the barred portions, the values during pregnancy. All values in millimeters of water.

In two women, uterine contractions occurred while the pressure was being measured, and the femoral pressure rose about 100 millimeters of water.

III. Stander, Duncan and Sisson[4] have shown that pregnant bitches may exhibit a high cardiac output. The third group of observations was carried out on such animals shortly before term. The following observations were made:

1. The pressure in the femoral vein was ± 100 millimeters higher than that in the jugular vein.

2. The pressure in the uterine veins was higher than that in the femoral vein, usually as high as 250-300 millimeters of water.

3. When the uterus was lifted from its position and held so that it no longer pressed upon venous channels, the femoral venous pressure diminished; but even after such a maneuver was still consistently above the jugular pressure.

4. The blood in the uterine veins contained more oxygen than the blood in the right side of the heart.

These three sets of observations seem to justify the following tentative conclusions:

1. A measurable increase in the output per minute of the heart occurs during pregnancy. In patients with heart disease such an increase in work may be of importance in precipitating failure.

2. The leg venous pressure is high in pregnant women. Obviously this may be a factor in the occurrence of edema and varicosities during pregnancy.

3. It appears that a large volume of blood passes through the uterine sinusoids and enters the uterine veins at a relatively high pressure. This arterio-venous shunt or arterio-venous fistula is one of the factors leading to the increase in cardiac output.

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