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Rise of Blood Pressure During Ischemia of the Gravid Uterus.*

ERIC OGDEN, G. J. HILDEBRAND AND ERNEST W. PAGE. (Introduced by N. W. Shock.)

From the Division of Physiology, University of California Medical School.

To test the concept that diminished uterine blood supply might play some part in the hypertension of eclampsia[1] the carotid blood pressure was recorded in acute experiments while the aorta was partially occluded below the renal arteries.

Ten animals anesthetized with chloretone, with morphine and pentobarbital sodium, or by decerebration under ether were used. Six were pregnant (5 dogs, 1 cat); 4 were non-pregnant controls.

The carotid and femoral blood pressures were recorded kymographically and a long-handled screw clamp was adjusted around the aorta just below the renal arteries. The incision was closed with the clamp handle protruding and the animal was left undisturbed until the blood pressure was stable. The clamp was then tightened until the femoral pressure fell to about half its previous value and the carotid pressure was followed (Table I).

In the non-pregnant control animals aortic compression was not followed by any change in carotid pressure other than the immediate adjustments discussed by Brotchner.[2] Rytand,[3] Brotchner,[2] and Goldblatt, Kahn, and Hanzal[4] have all pointed out that there is no progressive increase of arterial pressure after constriction of the aorta below the renal arteries.

In 4 of the 6 pregnant animals aortic compression was followed by a definite gradual rise of blood pressure; in the other 2 the rises were small (16 mm in 2 hours and 24 mm in one hour respectively). The rises were so gradual that one could not usually say exactly when they began. The rises under chloretone were clear-cut and amounted to 10-58 mm but no strictly hypertensive levels were reached, perhaps because of the very low initial values. In the animal under morphine and nembutal the blood pressure was more normal but the rise was small. The 2 decerebrate animals showed considerable (34 and 48 mm) rises of blood pressure, reaching in one case the definitely hypertensive level of 196 mm Hg.

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1 Page, Ernest W., and Ogden, Eric, *Am. J. Obst. and Gyn.*, 1939, **38**, 230.

2 Brotchner, R. J., PROC. SOC. EXP. BIOL. AND MED., 1939, **40**, 264.

3 Rytand, D. A., *J. Clin. Invest.*, 1938, **17**, 391.

4 Goldblatt, H., Kahn, J. R., and Hanzal, R. F., *J. Exp. Med.*, 1939, **69**, 649.

TABLE I.
Blood Pressure Rise After Aortic Compression.

B.P. Mm Hg					
Before clamping	After clamping	Immediate rise	Subsequent rise	Duration of clamping (min)	
			Pregnant Animals		
148	196	20	28	42	Dog, decerebrate. Early pregnancy.
68	120	?	?	30?	Cat, decerebrate. First observations after accidental
60	104	10	24	34	occlusion of aorta. Late pregnancy.
86	104	8	10	225	
56	70	6	8	36	Dog, chloretone. Late pregnancy.
68	130	4	58	138	
64	76	4	8	87	Dog, morphine and nembutal. Late pregnancy. Very slight reductions in femoral pressure.
134	158	4	28	72	Dog, morphine and nembutal. Late pregnancy. First
164	110	8	—62	400	aortic compression extreme. Fetuses probably killed. Dog in bad condition at end of experiment.
54	80	10	16	36	
54	96	8	32	147	Dog, chloretone. Late pregnancy
62	98	10	26	130	
58	66	8	0	65	Same dog after removal of both uterine horns.
			Non-pregnant Animals.		
100	100	6	— 6	150	Dog, morphine and nembutal; non-pregnant female.
42	46	4	0	122	Dog, chloretone; male.
150	154	16	—12	26	Dog, nembutal; non-pregnant female.
144	148	14	—10	47	Dog, decerebrate; non-pregnant female.
98	116	20	— 2	93	

Figures in columns 1 and 2 measured immediately before compression and decompression respectively.
Column 3 represents highest point attained within 3 minutes of compression.
Figures in column 4 were obtained by subtraction to indicate the slow changes in blood pressure after the immediate adjustments had been made.

Release of the aortic clamp was followed by a return to the previous blood pressure, sometimes immediately, sometimes gradually during 20 minutes.

These rises of blood pressure could be produced repeatedly in the same animal except after a long period of unduly severe constriction, when it may be supposed that prolonged anoxemia had caused irreversible changes in the uterus or its contents.

From one dog, after several repetitions of the rise in blood pressure, the uterus and its contents were removed after clamping the mesosalpinx. It was then no longer possible to reproduce the slow rise of blood pressure by tightening the aortic clamp.

Since our 4 control animals exactly confirm Brotchner's finding that compression of the aorta below the renal artery produces no such prolonged rise as we have here described, we are forced to conclude that the products of conception (*i. e.*, fetus, placenta, or gravid uterus) are fundamentally responsible for these slow blood pressure rises. This conclusion is confirmed by the animal in which the blood pressure failed to respond to aortic constriction after the uterus and its contents had been removed. The fact that these characteristic rises in pressure began to appear and disappear only with tightening and loosening of the aortic clamp makes it hard to escape the conclusion that compression of the aorta is the factor in these experiments which determined the changes. It is possible that some nerve excitation originating from the constriction or manipulation might produce this effect in the gravid and not in the non-gravid animal, but on the grounds already discussed it seems to us very much more likely that the immediate determining factor is limitation of blood pressure or blood flow to the gravid uterus.

The attempt to show this effect in chronic experiments has been so far unsatisfactory in both dogs and rabbits because if the blood supply is not reduced beyond a point which would endanger the fetus or cause abortion, there is so much anastomotic circulation established within 24 hours that the femoral pressure is nearly equal to the carotid. Further steps are being taken to effect a more permanent diminution of blood supply to the gravid uterus.

If these experiments signify that circulatory insufficiency to the gravid uterus may produce a rise in systemic blood pressure in abnormal pregnancy, it may well be asked whether a balance between the actual uterine blood supply and the effects of temporarily inadequate blood supply may not play a part in the regulation of the circulation during normal pregnancy.