to normal indicates that the prothrombin deficiency was not due to a Vitamin A deficiency. Further work is required to show whether a deficiency of Vitamin A plays any rôle. Vitamin D was used because it has been observed to be of value for the hemorrhagic tendency in jaundiced patients.[3] How Vitamin D acts to decrease the prothrombin time is conjectural. This vitamin has been observed to cause a thrombocytosis and a shortening of the coagulation time in rats.[4] We have not made a study of the thrombocytes. The response to Vitamin K was to be anticipated. From our data it cannot be concluded whether the mineral oil produced the prothrombin deficiency by preventing the absorption of Vitamin K or by producing hepatic injury; but the latter possibility is unlikely. This method of producing a prothrombin deficiency is very convenient and has potentialities for being developed into an assay method for Vitamin K.

Summary. 1. Prothrombin deficiency has been produced in rats by feeding an adequate diet containing 20% by weight of mineral oil. 2. The subcutaneous administration of Vitamin K corrected the prothrombin deficiency; activated ergosterol definitely improved the deficiency; Vitamin A had no effect on the deficiency.

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Changes in Arterial Pressure after Bilateral Complete or Partial Ureteral Occlusion.*

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Recently, we have been investigating the effect of hydronephrosis on the arterial blood pressure. The blood pressure determinations in this study were made with the Hamilton manometer[1] on trained unanesthetized dogs according to a technic previously described by us.[2] In addition the blood non-protein nitrogen was determined before and after operation.

In 7 dogs complete bilateral ureteral occlusion was performed

[3] Gray and Ivy, *Am. J. Digest. Dis.*, 1935, **2**, 368; McNealy, Shapiro and Melnick, *Surg. Gyn. and Obst.*, 1935, **60**, 785.

[4] Phillips, Robertson, Corson and Irwin, *Ann. Int. Med.*, 1931, **4**, 1134; Tocantius, *Medicine*, 1938, **17**, 155.

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[1] Hamilton, W. F., Brewer, J., and Brotman, I., *Am. J. Physiol.*, 1934, **107**, 427.

[2] Katz, L. N., Friedman, M., Rodbard, S., and Weinstein, W., *Am. Heart J.*, 1939, **17**, 334.

under ether anesthesia. A definite elevation in diastolic arterial blood pressure to hypertensive levels resulted in 4, and a mild increase occurred in 2 (V-24, V-26, V-28, W-13, W-19, W-20, Table I). These blood pressure elevations persisted until death in uremia 1 to 5 days following the operation. In one dog (V-87) no change in blood pressure was observed. At necropsy moderate to marked bilateral hydronephrosis was found in all the animals.

In 7 dogs partial occlusion of both ureters was carried out under nembutal anesthesia, by placing an ordinary Goldblatt clamp on a portion of the distal third of both ureters and tightening the screws to produce constriction. Four of these dogs (W-44, W-49, W-53, W-50, Table II) showed an elevation of blood pressure which was maintained for several days, the pressure then tending to return

TABLE I.
Complete Bilateral Ureteral Occlusion.

1. V-24	Syst.	140	150	165	170	170	140
		—	—	—	—	—	— (D)
	Diast.	80	90	90	100	100	80
	Day	(C)	(1 P.O.)	(2 P.O.)	(3 P.O.)	(4 P.O.)	(5 P.O.)
2. V-26	Syst.	150	120				
		—	— (D)				
	Diast.	75	90				
	Day	(C)	(1 P.O.)				
3. V.28	Syst.	160	135				
		—	— (D)				
	Diast.	85	100				
	Day	(C)	(1 P.O.)				
4. V-87	Syst.	140	175				
		—	— (D)				
	Diast.	85	85				
	Day	(C)	(1 P.O.)				
5. W-19	Syst.	150	185	220	215		
		—	—	—	— (D)		
	Diast.	80	105	140	135		
	Day	(C)	(1 P.O.)	(2 P.O.)	(3 P.O.)		
6. W-13	Syst.	175	150	185			
		—	—	— (D)			
	Diast.	80	90	105			
	Day	(C)	(1 P.O.)	(2 P.O.)			
7. W-20	Syst.	115	125	120	175		
		—	—	—	— (D)		
	Diast.	65	65	80	110		
	Day	(C)	(1 P.O.)	(2 P.O.)	(3 P.O.)		

Syst.—Systolic pressure mm Hg.
Dias.—Diastolic pressure mm Hg.
(C)—Control blood pressure.
(D)—Dead.
(No. P.O.)—Day post-operative.
(S.L.)—Still living.

towards the normal level. One dog still living (W-78), continues to show fluctuations between normal and moderately hypertensive blood pressure levels. Six of these animals died from intercurrent infection or were sacrificed 3 to 26 days after the establishment of the partial obstruction. In 1 of the 4 dogs (W-44), a marked increase in the blood pressure was noted after the operation, and this dog died in uremia 3 days later. At necropsy one ureter of this dog was found to be completely occluded while the other permitted the easy passage of fluid in either direction. In addition the bladder was moderately distended with urine indicating that this animal was not anuric. In the 6 other animals the blood non-protein nitrogen also rose after the operation, indicating a transient relative renal excretory insufficiency, but within a few days the non-protein nitrogen tended to return toward the normal level.

Our observations indicate that an increase in blood pressure may follow an increase in intrarenal pressure such as is produced by complete or partial ligation of both ureters. Although this rise in blood pressure is sometimes coexistent with a relative renal excretory insufficiency, it has been demonstrated previously that the two conditions are independent since the factors leading to renal excretory insufficiency are not identical with those which result in hypertension.[3]

Increased intrarenal pressure has been shown to cause a diminution in blood flow through the kidney.[4, 5] Hinman and Morison[6] noted that changes occurred in the smaller renal arteries subsequent to progressive hydronephrosis which could be the basis for a decrease in the renal blood flow, and which might therefore operate in producing chronic renal ischemia with subsequent chronic hypertension. However, we have not as yet succeeded in producing chronic arterial hypertension by this method. Our experiments suggest the possibility that hydronephrosis may be associated with hypertension on a purely mechanical basis.

Summary. 1. Complete bilateral ureteral occlusion was followed by a rise in the arterial blood pressure to hypertensive levels in 6 out of 7 dogs. Moderate to severe hydronephrosis was observed at necropsy. 2. Partial bilateral ureteral occlusion was followed by a transient rise in arterial blood pressure to hypertensive levels in 5 out of 7 dogs. Mild to moderate hydronephrosis was observed at necropsy.†

3 Friedman, M., and Katz, L. N., *J. Exp. Med.*, 1938, **68**, 485.

4 Levy, S. E., Mason, M. F., Harrison, T. R., and Blalock, A., *Surgery*, 1937, **1**, 238.

5 Enger, R., Gerstner, H., and Sarre, H., *Zentralblatt f. Med.*, 1937, **58**, 865.

6 Hinman, F., and Morison, D. M., *Surg. Gyn. and Obs.*, 1926, **42**, 209.

† The blood NPN determinations were made by the chemical department.

TABLE II.
Bilateral Partial Ureteral Occlusion.

1. W-47	Syst.	135	150	170	115 (D)						
	Diast.	85	85	95	65						
	Day	(C)	(1 P.O.)	(3 P.O.)	(5 P.O.)						
2. W-44	Syst.	170	175	205	190 (D)						
	Diast.	100	120	140	135						
	Day	(C)	(1 P.O.)	(2 P.O.)	(3 P.O.)						
3. W-49	Syst.	155	165	190	170	140	150	135	125 (D)		
	Diast.	85	110	105	90	105	105	65	75		
	Day	(C)	(1 P.O.)	(3 P.O.)	(5 P.O.)	(7 P.O.)	(8 P.O.)	(20 P.O.)	(26 P.O.)		
4. W-51	Syst.	150	140 (D)								
	Diast.	80	75								
	Day	(C)	(1 P.O.)								
5. W-53	Syst.	170	145	175	160	150 (D)					
	Diast.	75	85	110	95	95					
	Day	(C)	(1 P.O.)	(3 P.O.)	(8 P.O.)	(9 P.O.)					
6. W-50	Syst.	170	150	160	160	180	145	150	160	135	75 (D)
	Diast.	85	100	100	100	120	90	85	120	85	25
	Day	(C)	(1 P.O.)	(2 P.O.)	(4 P.O.)	(5 P.O.)	(8 P.O.)	(9 P.O.)	(10 P.O.)	(12 P.O.)	(15 P.O.)
7. W-78	Syst.	140	150	175	175	170	200	170	175 (S.L.)		
	Diast.	75	85	110	90	85	100	85	100		
	Day	(C)	(1 P.O.)	(2 P.O.)	(3 P.O.)	(6 P.O.)	(14 P.O.)	(24 P.O.)	(30 P.O.)		

Conventions as in Table I.