Summary. The present study concerns itself with a titration of the encephalitogenic activity of mouse brain which induced acute disseminated encephalomyelitis in mice. In this regard the active agent revealed a potency higher than that hitherto described for the guinea pig receiving homologous tissue. The encephalitogenic factor was found not in the supernate but in the sediment after centrifugation at 8000 G; nor was it detected in a dialysate of mouse-brain tissue. Mouse brain was encephalitogenic in a heterologous species of animal and heterologous tissues were similarly encephalitogenic in the mouse; the disorder thus brought about by either was indistinguishable from that induced in mice by means of mouse brain.

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Observations of Neoprene Casts of Vascular Bed of the Kidney. (19156)

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Neoprene is an excellent material for the study of the structure of the renal vascular bed(1). A serious handicap to its use, however, is the fact that, injected *in vivo*, neoprene causes constriction of the vessels and prevents complete filling of the vascular system. Nevertheless, nearly complete casts of the vascular bed of the kidney can be obtained if the renal vessels are not constricted, but are dilated, and are empty of blood. In cases in which casts of the vascular system are obtained under such conditions the only consistently existing vessels which directly communicate between the renal artery and vein are situated in the hilum of the kidney.

We have obtained casts of the entire vascular bed of the renal cortex and a portion of the medulla by injections of neoprene into the aorta or renal artery *in vivo* provided that the renal vessels were rendered incapable of constriction, were dilated, and were emptied of blood by a brief period of ischemia. *In vivo* injections of neoprene or materials of a similar nature caused a marked renal vasoconstriction as indicated grossly by severe blanching of the kidney. Owing to this vasoconstriction, the filling of the vascular bed of the kidney with neoprene was incomplete. However, when the renal artery or the aorta above the origin of the renal arteries was clamped, after a few minutes the arterial bed of the renal cortex would no longer become constricted when neoprene was injected. Neoprene passed through the arterial and venous beds, and flowed out the renal vein. Surface vessels were filled with neoprene. The clamping procedure itself resulted in a transient cortical vasoconstriction which served to empty the cortical vessels of blood. This emptying of vessels was an additional aid in obtaining a more complete cast of the renal vasculature. Thus, although neoprene provides an excellent medium for the study of the structure of the renal vascular bed, it fails to provide accurate information regarding the intrarenal distribution of the circulating blood.

Especially noteworthy were the casts of relatively long vessels near the hilum, which communicated between the large branches of the renal artery and vein and probably were part of the vascular supply to hilar and pelvic tissues (Fig. 1). The exact caliber of the lumen of these vessels was not determined, but they looked small. These vessels had a greater tendency to form plexuses in the dog than in the rat. When the neoprene injected into the renal artery was of a different color than that injected into the vein, the two colors were found to be mixed in these vessels. These were the only direct communications between the renal artery and vein that were found to be constantly present. It was of interest that in several instances neoprene injected *in vivo*

1. Shonyo, E. S. and Mann, F. C., *Arch. Path.*, 1944, v38, 287.

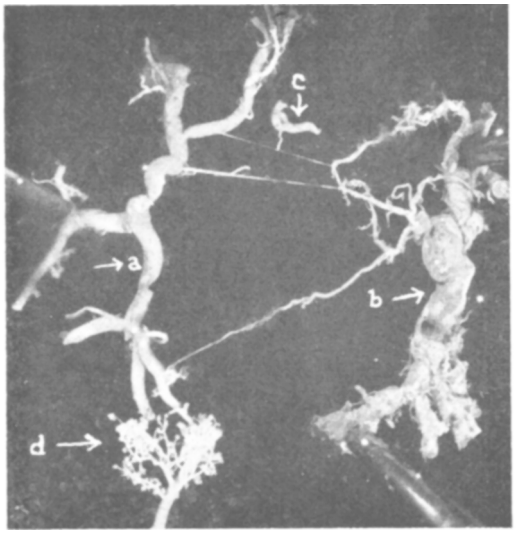

FIG. 1. Communications between arteries and veins in the hilum of the kidney. Injections of white neoprene into the renal artery and of blue neoprene into the renal vein were made into a rat's kidney. A portion of the casts of the hilar vessels were dissected away and mounted separately. One can note the casts of vessels communicating between a large artery, a, and vein, b, in the hilum of the kidney. At c is a short vessel running from one of the communicating vessels to a vessel of blue neoprene, considered to be an interlobar vein. Interlobular arteries with glomeruli from the same kidney are seen at d for comparison of size.

in the rat and dog was found to pass into the renal artery and out the renal vein as seen at the time of the injection, but a cast of only the renal artery and vein and these hilar vessels was obtained. The neoprene had caused a complete constriction of all vessels of the kidney except these hilar vessels, so the neoprene passed directly from the renal artery to the vein through these vessels.

Coiled arterial vessels which communicated between the same or different branches of the renal artery and similar vessels which communicated between the renal vein and its large branches also were found. These vessels were observed by Trueta and associates(2). Spanner(3) reported the finding of arteriovenous shunts in the hilum of one human kidney in which gelatin was injected, but he described short arteriovenous shunts which did not seem to be exactly the same type of vessel as seen in our study.

The results of our study of neoprene casts of the renal vasculature agreed otherwise with observations previously recorded by others indicating that in a normal kidney arteriovenous shunts are extremely rare and are insignificant functionally.

Summary and conclusions. (1) The injection of neoprene into the renal vessels *in vivo* causes vasoconstriction which interferes with the complete filling of the vascular system of the kidney. This vasoconstriction can be prevented by the production of ischemic vasodilatation prior to the use of the injection mass. Satisfactory neoprene casts of the renal vascular bed were obtained by the use of this technic, but the casts do not provide reliable information regarding the intrarenal distribution of the circulating blood. (2) The only consistently existing vessels which directly communicate between the renal artery and vein without an intervening glomerulus are situated in the hilum of the kidney. These vessels are not true arteriovenous shunts but probably are part of the vascular bed of the hilar and pelvic tissues of the kidney.

2. Trueta, Josep, Barclay, A. E., Daniel, P. M., Franklin, K. J., and Prichard, Marjorie M. L., *Studies of the Renal Circulation.* Springfield, Ill., Charles C Thomas, 1947, 187 pp.

3. Spanner, Rudolf, *Anat. Gesellsch. Verhandl.,* 1937, v45, 81.

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