

the other was apparently normal. Brain emulsions from these and from one which died soon after hatching were all lethal for mice inoculated intracerebrally. Virus recovered from the latter was carried through young normal chicks and then through mice by intracerebral inoculation. Clinical signs and pathological changes in the mice were typical of encephalitis. Two of the chicks were paralyzed and brain sections from 3 showed characteristic encephalitic changes. Virus was recovered also from one chick which had no clinical or pathological signs of encephalitis.

*Summary.* Three strains of St. Louis encephalitis virus have been cultivated in 4 different media containing living tissue and in developing chick embryos. The virus was recovered also from chicks which hatched after inoculation and from young chicks inoculated intracerebrally.

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#### **Experimental Local Bladder Edema Causing Urine Reflux Into Ureters and Kidneys.**

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There are no experimental studies recorded, as far as we are aware, of the rôle that a local transitory edema of the ureteral valve plays in the ureteral reflux of urine. Such a local edema we produced by infiltrating the vesical site of the ureter with 25% magnesium sulphate or with physiological salt solution.

We used 14 guinea pigs, 21 rabbits and 17 dogs, narcotized by morphine, or sodium barbital with or without magnesium sulphate, or ether. In dogs, ether only was used. The bladder and ureters were exposed through a median incision; pressure was recorded through a flanged cannula inserted in the bladder apex, and a water or mercury manometer. India ink was injected into the bladder as indicator. A few times the urethra was ligated. Infiltration of the ureter in its vesical course was done through a fine hypo needle; the amount varied between 0.2 and 2.0 cc.

In 14 guinea pigs (9♂, 5♀) 10 showed regurgitation (3♂, 7♀). The pressures ranged between 50 and 280 mm. water (4 to 21 mm. Hg.). In 4 non-pregnant females, spontaneous reflux occurred

into both ureters without infiltration under pressures between 20 and 120 mm. water (2 to 9 mm. Hg.).

In 21 rabbits (12♂, 9♀) 19 showed reflux into one or both ureters. The bladder pressures after infiltration ranged between 30 and 120 mm. water (3- to 9 mm. Hg.). The average control pressure giving no reflux before infiltration was 110 mm. water (9 mm. Hg.).

Spontaneous reflux without infiltration was seen in 2 non-pregnant females, one pregnant female and in one male. In the male and in one non-pregnant female biopsy revealed edematous ureteral orifices and a congested bladder mucosa.

In 17 dogs (8♂, 9♀) ureteral reflux was observed in 13 (6♂, 7♀); no reflux was obtained in 2 males and 2 pregnant females. The bladder pressures necessary for reflux after infiltration varied between 3 and 12 mm. Hg. The average control pressure tested with negative results before infiltration was 8 to 40 mm. Hg. Spontaneous regurgitation without infiltration was seen in 2 females, one of them in early pregnancy.

Biopsy of the bladder in the various species showed usually a well marked edema of the entire vesical ureter section and pouting ureteral orifices. The degree of edema produced by infiltration must not be so great that the lumen of the ureter is occluded. The onset of reflux often occurred during infiltration; the extent varied, but in all series filling of the kidney pelvis with ink was verified by biopsy in numbers of cases. The duration varied; usually after a longer or shorter interval both kidneys and ureters emptied themselves of ink. In some experiments, reflux was produced more than 3 times by successive infiltrations.

Contraction of the bladder did not play an important rôle in partial reflux; in dog for example reflux was produced repeatedly by the hydrostatic pressure of the urine alone (40 mm. water, 3 mm. Hg.). Bladder pressure did play a rôle when ureteral peristalsis was active. Even in these cases a pressure of 6 to 7 mm. Hg. was often sufficient to overcome ureteral peristalsis and drive ink into the kidney pelvis.

Copious secretion of urine may prevent the ascent of ink; under this condition ink only then rose when peristalsis had emptied the ureter. Antiperistaltic waves of contraction from bladder to kidney were never seen.

It seems permissible to assume that edema of the neck of the bladder following operative interferences in that region, or accompanying physiological processes such as menstruation and pregnancy, plays a part in the production of urinary reflux and pyelitis.