46 (384)

An experimental study of the influence of kidney extracts and of the serum of animals with renal lesions upon the blood pressure.

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1. Extracts of the rabbit's kidney injected into the rabbit cause a slight increase in blood pressure which is barely more than that due to the mechanical effect of the injection.

2. Extracts of the dog's kidney injected into the dog cause a decided fall in pressure; an equal fall may be caused by the dog's urine. A series of control experiments indicates that the fall caused by the kidney extract may be due to the urinary salts which it contains.

3. Extracts of cat's kidney cause a rise in pressure; as the cat's urine causes a fall, this rise in pressure indicates the possibility of a kidney extract containing a pressor substance which cannot be influenced by the depressor substance of the urine.

4. Rabbit's kidney which in the rabbit produces a slight rise when injected into the dog causes a drop comparable to that caused by the dog's kidney itself. Similarly the dog's kidney, which injected into the dog causes a drop, produces in the rabbit a rise analogous to that produced by rabbit's kidney. It is evident therefore that these pressor and depressor substances of the kidneys in question do not have a constant effect on all animals as do the extracts of the adrenal gland.

5. Extracts of kidneys which are the seat of various forms of nephritis cause the same effect as extracts of normal kidneys.

6. The serum of dogs with considerable reduction of kidney substance causes a slight fall in pressure; the serum of dogs with spontaneous nephritis gives divergent results, as does also the serum of rabbits with various forms of acute nephritis. The serum of dogs with chromate nephritis causes a slight rise, while that of dogs with uranium nephritis produces a sharp and decided fall in pressure. Although there is no uniformity in these results, their

general character, and especially the experience with uranium and chromate sera of the dog, suggests that pressure-disturbing substances are present in the serum as the result of the kidney lesion. The very slight evidence of the constant presence of a pressor substance, however, offers little support to the theory that such a substance is furnished by the diseased kidney or is due to disturbances of metabolism caused by disease of the kidney.

47 (385)

Further observations on the effect of asphyxia and curare on the reducing power of the blood after section of the hepatic nerves in dogs.

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In a previous communication on this subject (Macleod, *American Jour. of Phys.*, 1909, XXIII, 278) it was concluded that section of the hepatic nerves does not prevent the establishment of hyperglycæmia as a result of asphyxia and curare poisoning (Table III, p. 293, *loc. cit.*). The conclusion was based on the results of three experiments in which asphyxia was practiced, and in two of which marked hyperglycemia was observed; and on one in which curare was injected. Subsequent experiments of the same nature have yielded results which do not corroborate the above conclusion for asphyxiated animals, but do so for those which are curarized.

The following table gives the results of these experiments:

No. and nature of experiments.	*Per cent. of reducing substance in blood.*	
	Before.	*After.*
102 asphyxia	0.104	0.121 (30 min.). 0.147 (90 min.).
104 asphyxia	0.244 0.244	0.262 (60 min.).
105 asphyxia	0.200 0.214	0.176 (45 mm.).
107 asphyxia	0.113	0.155 (45 min.). 0.138 (75 min.).
108 curare	0.265	0.304 (30 min.). 0.363 (45 min.). 0.354 (75 min.).
109 curare	0.178	0.334 (40 min.).
110 curare	0.146	0.225 (60 min.). 0.272 (90 min.).