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.

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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.).