gestion of food by rats in the early stages of dimethylaminoazobenzene feeding and of rats receiving a protective supplement is approximately 10 g per day per rat. Rats supplied the unsupplemented carcinogenic diet eat between 4 and 6 g per day when they have been on this diet for 100 days or more. Another contributing factor probably is that late in the experiment less aminophenol or other phenolic compounds are available for conjugation with glucuronic acid due to some metabolic change associated with the damage to the liver cells.

Conclusions. 1. The ability of rat liver slices to conjugate borneol with glucuronic acid *in vitro* is not impaired, even when marked cirrhosis and malignant degeneration of the liver are produced, by the feeding of dimethylaminoazobenzene. 2. The glucuronide excretion of rats fed dimethylaminoazobenzene is initially somewhat higher than in the control animals. However, after 100 days of dimethylaminoazobenzene feeding, the rate is less than 25% of that seen initially.

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Experimental Hypertension from Section of Moderator Nerves: Relationship to Presence of Kidney Tissue.*

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Hering,[1] Koch and Mies,[2] Heymans, Bouckaert and Regniers[3] and others have shown that section of both carotid sinus nerves and both aortic depressor nerves in experimental animals results in an immediate rise in blood pressure. Section of these 4 moderator nerves in dogs results in persistent hypertension which is essentially neurogenic in origin. Following total sympathectomy, this form of hypertension disappears, according to Heymans and Bouckaert.[4]

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1 Hering, H. E., *Die Karotissinusreflexe auf Herz und Gefasse,* Dresden, Th. Steinkopff, 1927.

2 Koch, E., and Mies, H., *Krankheitsforschung,* 1929, **7**, 241.

3 Heymans, C., Bouckaert, J. J., and Regniers, P., *Le Sinus carotidien,* Paris, 1933.

4 Heymans, C., and Bouckaert, J. J., *Bull. de l'Acad. Royale de Med. de Belg.,* 1936, **6**, 42.

Grimson, Bouckaert and Heymans[5] produced hypertension in dogs completely sympathectomized save for the origin and distribution of the splanchnic nerve fibers to kidneys and adrenals by cutting the moderator nerves. Following denervation of the kidneys at the pedicle in 2 such animals, blood pressure fell to normal for 90 and 40 days of observation. They therefore postulated that the sustained neurogenic hypertension thus obtained had been of renal origin.

Heymans and Bouckaert[6] showed that the blood of dogs made hypertensive by moderator nerve section possessed a higher degree of vasopressor activity than the blood of normal dogs. This they attributed to an increased adrenalin content of the blood. Hermann, Jourdan, and Delrieu[7] demonstrated that in the dog made hypertensive by moderator nerve section, adrenalin is not the only substance present in the circulating blood responsible for the increase in peripheral vascular resistance. Page[8] has found that the blood of similar hypertensive animals showed vasoconstrictive effects when perfused through the rabbit's ear, which he attributes to increased amounts of angiotonin.

In view of the recent interest in the etiological relationship of the pressor substance angiotonin, isolated from kidney tissue, to other forms of experimental hypertension, and since the above mentioned reports bring up the possibility that angiotonin may play a rôle in the hypertension resulting from moderator nerve section as well, a series of acute experiments was carried out comparing the effect of moderator nerve section in normal animals and in animals in which both kidneys had been removed.

The 4 moderator nerves were sectioned in 19 dogs, anesthetized lightly either with ether or with chloralose. Ten were normal animals with kidneys intact. Nine had had both kidneys removed under ether anesthesia. Bilateral nephrectomy had been carried out 24 to 48 hours before the acute experiment in 7 instances; these animals were lively and in good condition. In one dog the kidneys were removed 4 days before, and in another dog immediately before section of the moderator nerves. Blood pressure was recorded directly from the femoral artery by means of a mercury manometer.

[5] Grimson, K. S., Bouckaert, J. J., and Heymans, C., PROC. SOC. EXP. BIOL. AND MED., 1939, **42**, 225.

[6] Heymans, C., and Bouckaert, J. J., PROC. SOC. EXP. BIOL. AND MED., 1938, **39**, 94.

[7] Hermann, H., Jourdan, F., and Delrieu, A., *Arch. des Mal du Coeur*, 1939, **39**, 545.

[8] Page, Irvine, personal communication.

The 4 moderator nerves were cut in succession in varying order. The entire vago-sympathetic-depressor nerve trunk was severed on each side. The carotid sinus bifurcation was often removed at the time of carotid sinus nerve section.

Upon section of a single moderator nerve, arterial pressure usually rose abruptly for a minute or two, reaching a peak which was sustained for one to 3 minutes. Pressure then fell more gradually, generally attaining a new baseline somewhat above the original one in 5 or 10 minutes. The arterial blood pressure just preceding section of the first moderator nerve was compared in each experiment with the pressure at the peak of the response to section of the fourth moderator nerve. The total net rise (or fall) in arterial blood pressure from section of the 4 moderator nerves was thus observed. A net rise of 10% or more of the initial arterial pressure was observed in 9 of the 10 normal animals and in 6 of the 9 totally nephrectomized dogs. (Table I.) The mean initial pressure of these 9 normal dogs was 130 mm of mercury, while that of the 6 nephrectomized dogs was 98 mm of mercury. The mean rise in arterial pressure was 51 mm of mercury, or 38% of the initial pressure, in normal dogs, and 87 mm of mercury, or 87%, in nephrectomized dogs.

One normal animal showed a drop of 56 mm of mercury, or 34% of the original pressure of 188 mm of mercury. Two nephrectomized dogs also had a drop of pressure from initial pressures of 106 and 135 mm of mercury, amounting to 72 and 51 mm of mercury respectively, or to 68 and 38% of the initial pressure. All 3 of these experiments were technically faulty. In 2, well marked cyanosis denoting definite anoxemia was present and in the third, the arterial pressure fell spontaneously throughout the experiment, possibly from too large a dose of chloralose for that animal.

The single dog which was studied 4 days after nephrectomy seemed lethargic and weak before anesthetization, and the arterial pressure ranged from 62 to 67 mm of mercury throughout the experiment.

TABLE I.

		Initial arterial pressure in mm Hg			Rise in arterial pressure in mm Hg			Rise in arterial pressure in %		
	No.	Range	Mean	Median	Range	Mean	Median	Range	Mean	Median
Normal dogs	9	74-197	130	120	14-100	51	42	13-62	38	42
Dog immediately after nephrectomy	1	—	100	100	—	82	82	—	82	82
Dogs 24-48 hr after nephrectomy	5	64-123	98	103	20-137	87	105	34-140	87	100

Section of the moderator nerves at no time produced any pressor or depressor response in this animal.

Summary. From the foregoing results it seems clear that in the dog, removal of both kidneys does not prevent the immediate appearance of a pressor response to moderator nerve section. The reflex response to such a procedure is at least equal to that seen in normal dogs and if anything, seems to be heightened. Chloralose and ether were each used as anesthetic agents in both groups of experiments. It so happened that one of the most marked pressor responses occurred after the use of each kind of anesthetic agent in both the normal and nephrectomized group of dogs. In view of the slightly different initial blood pressure levels in the two groups, as well as the difficulty in ascertaining the exact depth of anesthesia in each experiment, close quantitative comparison of the pressor responses in the two groups is not justified. It would seem, however, that circulating pressor substances arising from the kidney (*e. g.*, angiotonin) are not demonstrably important in the production of acute neurogenic hypertension.

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Comparative Study of Effect of Sulfadiazine and Sulfathiazole on *Staphylococcus aureus.*

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(Introduced by Chester S. Keefer.)

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Sulfadiazine has been found to be as effective as sulfapyridine and sulfanilamide in the treatment of experimental pneumococcal and streptococcal infections,[1, 2] and its administration to man causes relatively few toxic symptoms.[3, 4, 5] In staphylococcal infections

[1] Feinstone, W. H., Williams, R. D., Wolff, R. T., Huntington, E., and Crossley, M. L., *Bull. Johns Hopkins Hosp.*, 1940, **67**, 427.

[2] Robbin, R. O., Jr., and Winnek, P. S., *J. Am. Chem. Soc.*, 1940, **62**, 1999.

[3] Peterson, O. L., Strauss, E., Taylor, F. H. L., and Finland, M., *Am. J. Med. Sci.*, 1941, **201**, 351.

[4] Plummer, N., and Ensworth, H. K., PROC. SOC. EXP. BIOL. AND MED., 1940, **45**, 734.

[5] Reinhold, J. G., Flippin, H. F., Schwartz, L., and Domm, A. H., *Am. J. Med. Sci.*, 1941, **201**, 106.