five control groups but not from each other. In the second extinction test, only the SAC-Cue group was significantly different from controls ($P < .05$), and no significant differences were observed between groups ($P > .05$) on the third extinction trial.

Taken together, these data show that electrical stimulation of the ABL, but not CD, can serve as a CS in a bait-shyness paradigm. This conclusion is supported by the following findings. (i) The ABL-Cue group suppressed its drinking in the retention test in a manner similar to that of the SAC-Cue group; (ii) the suppression of drinking was maintained through the first extinction test but returned to baseline on succeeding extinction tests; (iii) the pairing of ABL stimulation with LiCl illness did not affect drinking on the tests with water and no ESB given after the injection; (iv) stimulation of ABL without the LiCl illness (ABL-Stim group) had no significant effect on water intake during retention and extinction tests; (v) electrode implantation (ABL-Impl group) had no significant effect on subsequent water intake, even though this group was injected with LiCl; and (vi) none of the CD groups, including the animals stimulated in the CD prior to the LiCl illness, showed a significant reduction in water intake throughout the experiment.

The locus-specific effect noted is important because it distinguishes the taste-aversion paradigm from other forms of classical conditioning, such as the rabbit's nictitating-membrane response, in which the effectiveness of ESB as a CS is not related to locus of stimulation (*15*). This finding suggests a unique relationship between the amygdala and taste aversion, one that may be related to the importance of this structure in the integration of visceral and gustatory signals (*13, 16*). As such, these data further emphasize the importance of using the CS properties of ESB in the analysis of neural substrates of conditioning.

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Vasectomy Increases the Severity of Diet-Induced Atherosclerosis in *Macaca fascicularis*

Abstract. *Diet-induced atherosclerosis developed more extensively in vasectomized cynomolgus monkeys (*Macaca fascicularis*) than in sham-vasectomized control monkeys fed the same diet. The effect was most pronounced in the abdominal aortas, carotid arteries, distal segments of the coronary arteries, and intracranial cerebral arteries. Antibodies to sperm developed in all vasectomized monkeys, and complement and immunoglobulins were associated with atherosclerotic plaques in some of the vasectomized animals. The immunological response to sperm antigens that often accompanies vasectomy may exacerbate atherosclerosis.*

Atherosclerosis in rabbits can be exacerbated by experimentally inducing both serum sickness (immune complex disease) and hyperlipoproteinemia (*1*). This combination of repeated immunologic injury to arteries and a lipid-rich diet not only increases the extent of rabbit atherosclerosis, but also affects the qualitative characteristics of the plaques so that they more closely resemble atherosclerotic lesions in human beings (*2*). This phenomenon is not limited to rabbits; much more severe atherosclerosis also develops in baboons fed a lipid-rich diet and repeatedly immunized with foreign protein (*3*). Sharma and Geer (*4*) have reported the results of immunologic and morphologic studies of the lesions induced in the aortic intima of rabbits with experimentally induced serum sickness. They concluded that the injury and associated increased permeability of the endothelium were due to immune complex deposition, and that, by way of this mechanism, immunologic injury to arteries plays a role in the atherosclerotic process.

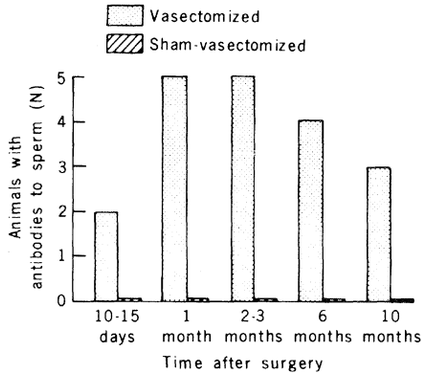

Fig. 1. Correlation between the number of vasectomized and sham-vasectomized monkeys in which antibodies to sperm developed and the time after surgery.

Multiple antigens are present in sperm, both on their surface and internally, and these can elicit production of autoantibodies. After animals are vasectomized, spermatozoa are confined to the epididymis and vas deferens. Presumably because these sperm lack a normal anatomical passage, they degenerate and release antigens that enter the circulation directly or are phagocytosed by macrophages. Sperm agglutination, sperm immobilization, and immunofluorescence provide the means to demonstrate circulating antibodies to sperm in about 50 percent of vasectomized men (*5–8*) and in vasectomized males of several other species (*9–11*).

Whether antibodies to sperm will develop in an individual after vasectomy may depend on genetically determined factors that affect immunologic responsiveness (*12*) or on different rates of sperm production. In vasectomized rhesus monkeys, high and sustained concentrations of antibody against sperm correlated significantly with high sperm counts before vasectomy (*13*); a similar finding has been reported in men (*14*). Another possibility is that antibodies to sperm develop in all vasectomized animals, but that routine methods measure only free antibodies and not

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those complexed with sperm antigens.

In rabbits, the level of circulating antibodies to sperm increases with time after vasectomy, and antibodies to testicular antigens develop in more than half of the bilaterally vasectomized animals. Deposition of the immune complexes occurs around seminiferous tubules, particularly in rabbits with high amounts of circulating antibodies to sperm; immune complexes specific for sperm are also found in the renal glomeruli (*11, 15*).

We now report that vasectomy increases the extent and severity of atherosclerosis induced by diet in *Macaca fascicularis*, a suitable experimental model of atherosclerosis (*16*), in which antibodies to sperm similar to those found in vasectomized men develop (*17*). Ten adult (7 to 8 years of age) male cynomolgus monkeys of Malayan origin (*18*) were individually caged and fed a diet that included 42 percent of its calories from butter and 0.5 mg of cholesterol per kilocalorie (*19*). Total plasma cholesterol was determined at monthly intervals for 6 months before the experimental period by the AutoAnalyzer II method of Rush *et al.* (*20, 21*).

After these baseline cholesterol determinations were made, the animals were divided into two groups of five monkeys each in such a way that the means and variance in plasma cholesterol concentrations of the two groups were equivalent. The mean [± standard error (S.E.)] plasma cholesterol concentrations were 542 ± 91 mg/dl for animals in the sham-vasectomized group and 510 ± 53 mg/dl for animals in the vasectomy group. The animals were fed the atherogenic diet for 6 months before vasectomy so that their diet-induced atherosclerosis might progress to a stage approximating that in young men of ages when vasectomies are most commonly done (*22*). The experimental group was vasectomized by the double ligation technique; the control, sham-vasectomized group was subjected to the same surgical procedure, without ligation or resection of the vas deferens. During the experiment, body weight was determined at monthly intervals, and

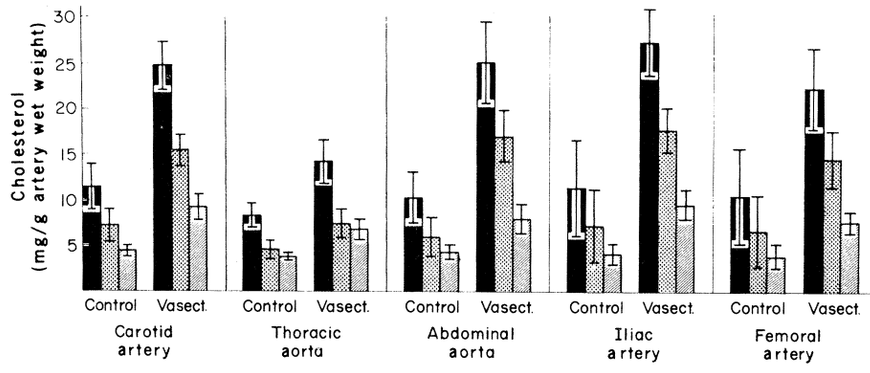

Fig. 2. The total ■, esterified ▨, and free □, cholesterol concentrations of the major arteries of the vasectomized animals are compared with those of the sham-vasectomized controls. The data are presented as means ± S.E.

blood samples were obtained for analyses of plasma cholesterol, triglycerides, and antibodies to sperm. Sperm agglutination, sperm immobilization, and immunofluorescence were used to assess circulating antibody titers against sperm in experimental and control animals (*7, 23, 24*).

No significant difference was found in the mean body weights of the animals in the two groups at any point in the experiment. At the beginning of the experiment, the mean (± S.E.) body weights were 5.1 ± 0.2 kg for the vasectomized group and 5.3 ± 0.3 kg for the sham-vasectomized group. At the time of vasectomy and at the termination of the experiment, the body weights of the vasectomized group were 5.3 ± 0.2 kg and 5.2 ± 0.2 kg, respectively; those of the sham-vasectomized group were 5.5 ± 0.3 kg and 5.7 ± 0.4 kg, respectively. Similarly, no differences were seen in the plasma cholesterol and triglyceride concentrations between the two groups of monkeys. When all of the determinations for the experimental period were considered, the sham-vasectomized animals had a mean (± S.E.) plasma cholesterol concentration of 546 ± 88 mg/dl and plasma triglyceride concentration of 35 ± 9 mg/dl. Among the vasectomized animals, the mean plasma cholesterol concentration was 593 ± 35 mg/dl and the mean plasma triglyceride concentration was 34 ± 3 mg/dl.

Antibodies to sperm developed in all the vasectomized monkeys but not in any of the sham-vasectomized animals. Antibodies developed in two of the five vasectomized animals as early as 15 days and were detected in all of the animals by 1 month after vasectomy. Antibodies to sperm could still be detected in four of the five experimental animals after 6 months and in three of five after 10 months (Fig. 1).

The experiment was terminated, and necropsies were done 10 months after vasectomy or sham vasectomy (16 months after the initiation of the atherogenic diet). Tissue for immunologic studies was taken from the testis, efferent ducts, epididymis, kidney, and aorta. To locate possible immune complexes in frozen sections, we used fluoresceinisothiocyanate-labeled antibodies to immunoglobulins of the G, M, and A classes (IgG, IgM, IgA) and to the third component of complement (C3) (*25*). No immunoglobulin deposition was detected around the seminiferous tubules; thus, the response of *M. fascicularis* to vasectomy is similar to that of *M. mulatta* (*26*) and man (*27*) rather than that of the rabbit (*11, 15*). Deposition of C3 occurred in one of the five vasectomized monkeys in the basal lamina of the efferent ducts, but was not found in the caput and cauda epididymis. These results confirm find-

Table 1. Extent of atherosclerosis among groups of sham-vasectomized and vasectomized monkeys.

Group	Carotid artery*	Carotid bifurcation†	Thoracic aorta*	Abdominal aorta*	Iliaco-femoral artery*	Cerebral arteries‡	Coronary arteries§
Sham-vasectomy	22 ± 11	2.4 ± 0.4	20 ± 5	28 ± 8	35 ± 16	1.4 ± 0.6	37 ± 13
Vasectomy	77 ± 10	3.8 ± 0.2	54 ± 14	92 ± 3	82 ± 10	4.4 ± 0.7	68 ± 2.7
	$P < .01$	$P < .02$	$P < .10$	$P < .001$	$P < .05$	$P < .02$	$P < .05$

*Percentage of intimal surface affected with raised atherosclerotic lesions, mean ± S.E. †Extent of atherosclerosis based on a scale of 0 to 4, mean ± S.E. ‡Number of grossly visible plaques in vertebral, basilar, and posterior communicating arteries. §Mean percentage ± S.E. of the apparent lumen occupied by lesions.

ings from studies on vasectomized rhesus monkeys in which deposits were found in the efferent ducts and kidney glomeruli (*26*).

Sections of thoracic and abdominal aortas were also examined for localization of immunoglobulins or complement by direct immunofluorescence. Both immunoglobulin and C3 deposition were demonstrable in the atherosclerotic plaques in two of the five vasectomized animals but in none of the controls. Deposition of immunoglobulin was found in the renal glomeruli of all the vasectomized monkeys, compared to two of the five control animals. The animals exhibiting C3 deposition in the atherosclerotic plaques exhibited IgM, IgG, and C3 deposition in sections of renal glomeruli in a finely granular pattern.

To evaluate the effect of vasectomy on the extent and severity of atherosclerosis, we used both angiochemical and pathological methods. At the time of necropsy, the following arteries were removed, cleaned, weighed, measured, and frozen for subsequent determinations of free and esterified cholesterol: the left common carotid, the left half of the thoracic aorta, the left half of the abdominal aorta, the left iliac artery, and the left femoral artery. For subsequent pathological evaluations, the right carotid artery, the right half of the thoracic aorta, the right half of the abdominal aorta, the right iliac artery, and the right femoral artery were opened longitudinally, flattened on cardboard, and fixed in 10 percent neutral buffered formalin. Hearts and brains were also fixed in 10 percent neutral buffered formalin.

For chemical analysis, arterial samples were homogenized in chloroform and methanol (2 : 1, by volume); the lipids were extracted (*28*), and separated by thin layer chromatography on silica gel G with a solvent system of Skelly-solve B, ethyl ether, and acetic acid (146 : 50 : 4). After chromatography, both the nonesterified and esterified cholesterol fractions were identified by iodine staining and eluted from the silica gel.

Arteries from the vasectomized animals contained more total and esterified cholesterol than did those from the sham-vasectomized animals (Fig. 2). Differences between the groups were the same when the data were expressed on the basis of wet weight or surface area (millimeters squared). All differences were significant ($P < .05$) by the Student's t-test except for the thoracic aorta and femoral artery.

The large arteries were stained overnight in a supersaturated solution of Sudan IV in 35 percent isopropanol to evaluate the extent of grossly visible atherosclerosis. They were then washed in water and stored in 10 percent neutral buffered formalin. Each artery was studied separately, and the percentage of intimal surface affected with raised atherosclerotic plaques was recorded (*29*). The extent of atherosclerosis in the carotid artery, thoracic and abdominal aortas, and iliaco-femoral artery from the sham-vasectomized and vasectomized groups was recorded (Table 1). Atherosclerosis was more extensive in the arteries of the vasectomized animals; the difference between the groups was statistically significant for all tissues but the thoracic aorta. The atherosclerotic involvement of the carotid bifurcation was considered separately, and severity was expressed on a scale of 0 to 4. The vasectomized animals were more severely affected than the sham-vasectomized controls (Table 1).

The vertebral, basilar, anterior, middle, and posterior cerebral arteries were examined to determine the extent of atherosclerosis in the intracranial cerebral arteries. Cerebral artery atherosclerosis was recorded on a scale in which 0 represented no plaques present and each subsequent number represented the number of plaques seen (Table 1). Although intracranial cerebral atherosclerosis is infrequent among cynomolgus monkeys fed an atherogenic diet, extensive involvement was seen in the vasectomized animals (Fig. 3).

The extent of coronary artery atherosclerosis was determined histologically. Thirteen tissue blocks, each approximately 3-mm thick, were cut perpendicularly to the long axis of the arteries. Blocks were taken from the following sites: one from the midregion of the left main coronary artery, three from the right coronary artery, three from the left circumflex, and six from the left anterior descending (three of which were selected from the proximal portion of the artery adjacent to the bifurcation of the left

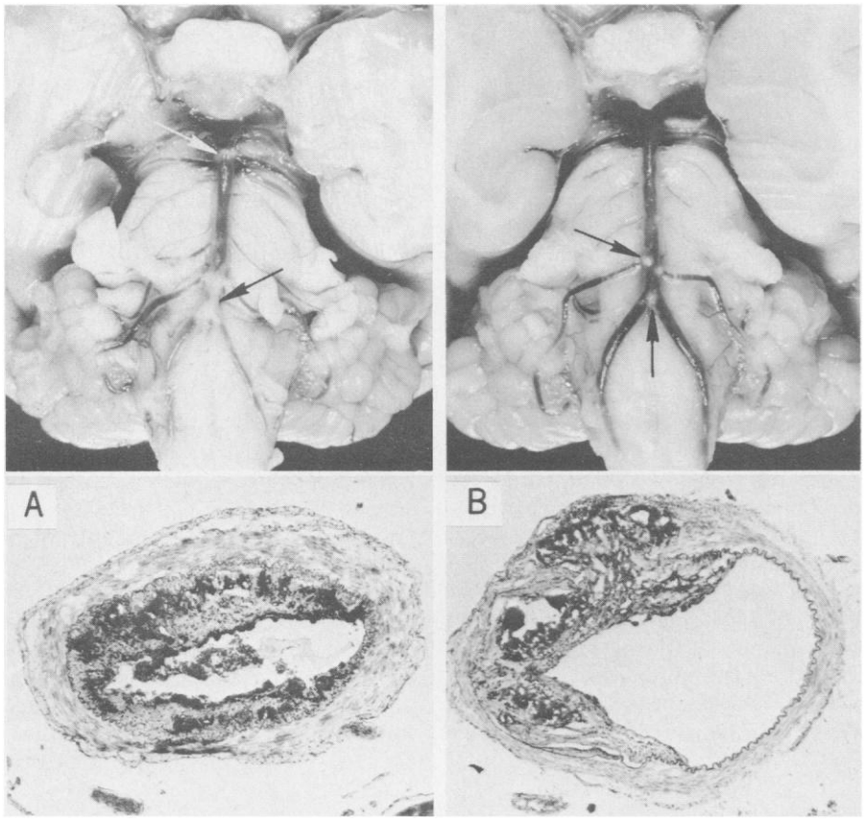

Fig. 3. The cerebral arteries from two of the vasectomized monkeys. (A) The upper photograph (×2) shows atherosclerotic plaques of the basilar artery. Several plaques are present within the anastomosis of the vertebral arteries and extend into the basilar artery at the origins of the posterior inferior cerebellar arteries (dark arrow). A large plaque is also present at the origin of the posterior cerebral arteries (white arrow). Microscopically, the lower photograph (×100) shows that the plaque from the proximal basilar artery is abundant in sudanophilic material (Sudan IV–hematoxylin stain). (B) The upper photograph (×2) contains atherosclerotic plaques at the vertebral anastomoses and at the origin of the posterior inferior cerebellar arteries (dark arrows). Microscopically, the lower photograph (×100) shows an eccentric plaque containing abundant lipid with disruption of the internal elastic lamina and underlying tunica media (Sudan IV–hematoxylin stain).

main coronary artery and three from the distal midventricular portion of the artery). Two 5-μm sections were cut from each block and stained with either hematoxylin and eosin or Verhoeff–Van Gieson stains. For each block, the percentage of the apparent lumen occupied by atherosclerotic lesion (stenosis) was recorded (Table 1). Consistent with the presence of atherosclerosis in all of the arteries except the thoracic aorta was the more extensive stenosis in the coronary artery of the vasectomized monkeys compared with the sham-vasectomized controls. From the gross appearance of the hearts, it seemed that the coronary artery atherosclerosis was more distally extended among the vasectomized animals. For this reason, both proximal and distal segments of the left anterior descending artery were examined. Both gross and microscopic observations showed a greater difference in the distal segment. The mean stenosis of the proximal segment of the left anterior descending coronary artery of the vasectomized animals was 69.8 ± 4.3 compared with 42.1 ± 16.2 for the sham-vasectomized group. This difference was not statistically significant ($P < .20$). However, the mean stenosis of the distal segment of the same artery in the vasectomized animals was 67.0 ± 4.9, whereas in the sham-vasectomized group it was only 25.6 ± 11.4, and this difference was statistically significant ($P < .02$).

In our experiments, the monkeys were fed a diet containing about twice as much cholesterol as that consumed by the average North American and the plasma cholesterol concentrations in these monkeys were generally around 500 to 600 mg/dl, in contrast to the usual concentrations of 220 to 250 mg/dl for North American adult human males. Although the findings are statistically significant, the data are based on two groups of only five animals each.

We suggest that the basis of the observed effects after vasectomy may be immunologic injury to the vascular endothelium, resulting in a rapid progression of the lesions. The data collected under the conditions of our study showed that vasectomy was associated with a marked exacerbation of diet-induced atherosclerosis in *M. fascicularis*.

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29. The following measurements were determined for each artery: total surface area, surface area containing fatty streaks, fatty plaques, fibrous plaques, and diffuse intimal thickening. The presence and extent of ulcerated plaques, hemorrhage into plaques, and thrombosis were recorded.
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Proctodeal Feeding by Termitophilous Staphylinidae Associated with *Reticulitermes virginicus* (Banks)

Abstract. Trichopsenius depressus *Le Conte,* Xenistusa hexagonalis *Seevers, and* Philotermes howardi *Kistner and Gut solicit and receive proctodeal and stomodeal fluids from their host, as well as engage in allogrooming with them. No other trophic behaviors were observed, suggesting that the beetles are completely integrated into the termite's trophic system.*

Behavioral and biochemical studies of trophic relationships between termitophiles and their hosts are scarce. A few authors (*1*) have reported that integrated termitophiles (*2*) solicit and receive saliva or regurgitated crop contents (stomodeal food) (or both) from their host termites. But no one has reported the solicitation and receiving by a termitophile of the liquid hindgut contents of its host (proctodeal food). I report here the first observation of such proctodeal feeding by a termitophile, and also describe two other trophic relationships between the subterranean termite *Reticulitermes virginicus* (Banks) and its three termitophiles, *Trichopsenius depressus* Le Conte, *Xenistusa hexagonalis* Seevers, and *Philotermes howardi* Kistner and Gut.

Portions of three colonies of *R. virginicus* and their associated termitophiles were collected in early April 1976 from fallen logs in the De Soto National Forest approximately 30 km north of Gulfport, Mississippi. Colony 1 yielded 29 *P. howardi*, 9 *X. hexagonalis*, and 30 *T. depressus*. Colony 2 yielded 4 *P. howardi*, 2 *T. depressus*, and 1 *X. hexagonalis*. Colony 3 yielded 18 *P. howardi*, no *X. hexagonalis*, and 6 *T. depressus*. The beetles from each source colony were placed with a representative mixture of about 200 of their host termites in petri dishes (14 cm) containing 13.5 g of moistened synthetic termite diet (*3*). Twenty-four hours were allowed for gallery establishment before the beginning of behavioral observations. A portion of a fourth colony of *R. virginicus* from the same area was collected during April 1977 and was used as a source of termitophiles for the dissection experiments. Behavioral observations (*4*) were made

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