

If the genus *Aerobacillus* Donker (1926) is to be adopted, the following species are recognized for the present:

1. *Aerobacillus polymyxa* (Prazmowski) Donker 1926, syn:
  - Clostridium polymyxa* Prazmowski 1880
  - Granulobacter polymyxa* Beijerinck 1893
  - Bacillus polymyxa* Beijerinck and Den Dooren De Jong 1923
  - Astasia asterospora* Meyer 1892
  - Bacillus asterosporus* (Meyer) Migula 1900
  - Bacillus mycoides* var. *ovoathylicus* Wagner 1916
  - Bacillus aerosporus* Greer 1928.
2. *Aerobacillus macerans* (Schardinger) Donker 1926, syn:
  - Bacillus macerans* Schardinger 1905
  - Bacillus acetoathylicum* Northrop 1919
  - Aerobacillus acetoathylicus* (Northrop) Donker 1926.

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### Ligation of Carotid and Vertebral Arteries in Monkeys.

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The mortality resulting from ligation of the common carotid artery in man varies from 7 to 90%, depending on the condition for which the ligation was done, and the age of the patient.<sup>1</sup> The value of pre-ligation compression of the carotid artery to establish collateral circulation, is not definitely established. This work was primarily undertaken to determine the effect of pre-ligation compression of the common carotid in monkeys. Dogs and rabbits seldom develop symptoms after ligation of a common carotid artery,<sup>2</sup> whereas we hoped that monkeys, whose cranial blood supply is quite similar to that of man,<sup>3</sup> would react in a way comparable to man. However, we were unable to cause paralysis or death in monkeys by ligation of a common carotid artery, or by simultaneous unilateral ligation of vertebral, internal, external and common carotid arteries. The ligations were continued in an effort to determine

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<sup>1</sup> Freeman, L., *Ann. Surg.*, 1921, **74**, 316.

<sup>2</sup> Sussi, L., *Ann. ital di chir.*, 1932, **11**, 311.

<sup>3</sup> Hartman and Straus, *Anatomy of the Rhesus Monkey*, Chapter XII, p. 251.

TABLE I.

Exp. Procedure Ligation of	Symptoms and Results	Gross and microscopic pathology. (Positive findings only)
1—A. Rt. Com. Carotid	No change 6 days	
B. Rt. Int. Carotid	No change 6 days	
C. Rt. Thyrocervical axis	No change 24 days	
D. Rt. Vertebral	Rt. foreleg paralyzed (1) Death in 6 days hemorrhage stump of Vertebral	(2) Perivascular cellular infiltration, bilateral.
2—A. Rt. Vertebral Int. Ext. and Com. Carotids	No change in 11 months	
B. Lt. Com. Carotid	Animal comatose, no paralysis. Death in 2 days.	Cerebellum showed glial proliferation, absence of Purkinje cells and of normal markings. Patchy areas of early necrosis in cerebrum. (3)
3—A. Rt. Vertebral, Thyrocervical axis, Int. Ext. and Com. Carotids	Death from pneumonia in 2 days. No symptoms of cerebral ischemia.	Hemorrhage bilateral in area of Int. capsule. Sections cerebellum normal. Cerebrum showed congestion of blood vessels, perivascular hemorrhage and cellular infiltration.
4—A. Rt. Vertebral Int. Ext. and Com. Carotids	No change in one year.	
B. Lt. Com. Carotid (4)	Slight ataxia and weakness right foreleg. No paralysis. Stupor in 2 hr. Death in 4 hr.	No change. (5)
5—A. Rt. Vertebral, Int. Ext. and Com. Carotid	Transient vertigo and ataxia; otherwise no change in 11 months	
B. Lt. Com. Carotid	No change in ½ hr. Killed by inject. ether	Microscopic shows (7,8) blanching of tissues about vessels.
6—A. Lt. Vertebral Thyrocervical axis, Int. Ext. and Com. Carotids	Ataxia. Animal fell to right. Rt. side weak and Rt. extremities moved poorly. Symptoms decreased gradually. Death 6th post-operative day. Definite pneumonia.	Some softening Ft. frontal lobes. (9) Tissue edematous and vacuolated. Mid and ant. portions of cerebrum took a homogeneous stain. Ganglion and glial cells pale staining and nuclei indistinct. Many cavities. Phagocytic cells about larger vessels on Rt. Some perivascular hemorrhage.

(1) Due to traction on brachial plexus?

(2) Injection of ink into Lt. Common Carotid. Vessels of both sides filled equally well.

(3) Rt. frontal lobes least involved.

(4) Local anesthesia.

(5) Definite intimal thickening of arteriosclerosis.

(6) Lt. Common Carotid first digitally compressed for one hour. No change except for periods of excitement, hyperpnea and nystagmus.

(7) Barium in formaldehyde allowed to flow into the cannulized Lt. Common Carotid. Vessels of both sides equally well filled as shown by X-ray and post-mortem.

(8) Due to ether injection? Similar in amount on both sides.

(9) Postmortem had been delayed 24 hours.

how much alteration of cranial blood supply the monkey would tolerate, and what changes in the brain were produced.

Table I summarizes the procedures and results obtained.

1. The rhesus monkey is unaffected by ligation of a common carotid artery, and in more than one-half of the procedures, will be unaffected by simultaneous ligation of the common, internal and external carotid and vertebral arteries. In no instance did hemiplegia result; in one there was weakness of the opposite side; and in 2, transient ataxia

2. If, in addition to the ligation of the main cervical vessels on one side, the common carotid artery on the other side is ligated, the animals will die, even though the second ligation is done 9 months to a year after the first. The survival of the animals following the first ligation is due to the adequate blood supply obtained from the vessels of the opposite side, as demonstrated by the injection of ink, or of barium solution into the common carotid of one side, and its equal distribution in the superficial vessels of both hemispheres. Little collateral develops on the side undergoing primary ligation of the main vessels, as death occurs when the remaining common carotid is ligated.

3. Of the 6 animals undergoing ligation of the main arteries of one side, only one showed gross brain changes, (Exp. 6) and these were probably post-mortem, as necropsy had been delayed. On the same basis the microscopic changes here cannot be accepted as due to cerebral ischemia alone. The pathology in the cerebellum in Experiment 2 represents an older change than would be expected to occur in the 2 days intervening between the second ligation and death, and is, therefore, probably the result of the primary ligations. The areas of early necrosis in the cerebrum in this instance are undoubtedly the response to the ligation of the left common carotid. There were no gross or microscopic changes in the brain of the other animal dying from the ligation of the remaining carotid. (Exp. 4). This may be due to the short period (4 hours) between the ligation and death. We may conclude that animals may die of cerebral ischemia, yet have no gross or microscopic change in the brain.

4. The monkey, like the dog and rabbit, does not react to ligation of a common carotid artery in a way comparable to man, and is not a satisfactory animal for pre-ligation compression experiments.