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Unmyelinated Sensory Fibers.

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Unmyelinated sensory fibers are present in large numbers in the spinal nerves. They take origin from the small cells of the spinal ganglia and divide like the myelinated fibers into central and peripheral branches.[1] The central branches enter the tract of Lissauer through the lateral division of the dorsal roots and probably end in the *substantia gelatinosa* Rolandi.[2] The peripheral branches run in the spinal nerves and are distributed chiefly in the cutaneous branches; very few are found in the motor nerves.[3] Ingvar has been able to confirm most of these observations on human material.[4]

In order to exclude the possibility of confusion due to the presence of sympathetic unmyelinated fibers the question has been reinvestigated on sympathectomized cats. The right abdominal sympathetic chain was removed from the diaphragm to the pelvis and 5 weeks or more allowed for the degeneration of the sympathetic fibers in the femoral nerve. Pyridine-silver preparations were made of the saphenous nerve and of the nerve to the *vastus medialis*. Great numbers of unmyelinated fibers were found in the saphenous nerve; but very few in the nerve to the *vastus medialis*. Other pieces of the same nerves from the same sympathectomized cats were treated with osmic acid. Counts showed that as many myelinated fibers could be seen in the pyridine silver as in the osmic acid preparations. In one fascicle of a saphenous nerve 1294 myelinated fibers were counted in the osmic acid preparation and 1258 in the pyridine silver preparation. It is obvious that when the unmyelinated fibers are taken into consideration the number of axons present in the sympathectomized saphenous nerve far exceed the number of myelin sheaths.

The unmyelinated axons stain much darker than the myelinated axons in pyridine silver preparations. They are closely grouped together in bundles. They must be sensory since there are no sympathetic fibers in these preparations, and very few if any unmyelinated fibers leave the spinal cord in the ventral roots.

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[1] Ranson, S. W., *J. Comp. Neurol.*, 1912, **22**, 159.

[2] Ranson, S. W., *J. Comp. Neurol.*, 1913, **23**, 259.

[3] Ranson, S. W., *Brain*, 1915, **38**, 381.

[4] Ingvar, S., *Acta Medica Scand.*, 1926-27, **65**, 645.

Unmyelinated fibers having exactly the same appearance are found in large numbers in the vagus[5] and splanchnic nerves.[6]

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Gall Bladder Visualization and Jaundice.

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In this study we were interested in 2 questions: (1) Does tetraiodophenolphthalein when present in the gall bladder disappear following obstruction of the common bile duct on the ingestion of meals containing fat? (2) What is the effect of existing obstructive jaundice on the visualization of the gall bladder? To answer the first question the gall bladders of 5 dogs were visualized with tetraiodophenolphthalein, the common bile duct was ligated and meals containing fat were given daily. This is a repetition of the experiment of Copher.[1] It was found that the shadow becomes more dense for 2 or 3 days following the ligation of the common bile duct and this density is maintained as long as 2 weeks after the ligation. We did not follow any of the dogs for a longer period. These findings confirm those of Copher.

To answer the second question the common bile ducts of 5 dogs were ligated, and from 60 to 96 hours later the tetraiodophenolphthalein was injected. It was found that in 2 dogs the gall bladder was faintly perceptible in 14 hours, in one dog in 22 hours, and in the 2 others in 50 hours. At later periods up to 114 hours, the shadow slowly became more visible, but "normal density" was not obtained in any of the dogs. In each of these dogs the gall bladder was visualized and evacuated with egg yolk prior to common duct ligation. The presence of jaundice did not increase the toxicity of the dye.

It has been maintained by some writers[2] that the bile which enters the gall bladder is normally resorbed *in toto*. If this were true, it would be possible to claim that the disappearance of the gall bladder

[5] Chase, M. R., and Ranson, S. W., *J. Comp. Neurol.*, 1914, **24**, 31.

[6] Ranson, S. W., and Billingsley, P. R., *J. Comp. Neurol.*, 1918, **29**, 405.

[1] Copher, G. H., *J. Am. Med. Assn.*, 1925, **84**, 1563.

[2] Halpert, B., and Hanke, M. T., *Am. J. Physiol.*, 1929, **88**, 351.