

Unitary Explanation for Differential Nerve Block Remains Elusive

To the Editor:—The interesting observations by Gissen, Covino, and Gregus¹ on differential nerve fiber block by local anesthetics do not explain why in clinical peripheral nerve blocks, pain (small fibers) is frequently lost before pressure or vibration (large fibers). The clinical differential sequence is apparently the reverse of what they observed *in vitro*. In the cervical vagus nerve of the rabbit the large myelinated fibers to the larynx are segregated in a segment at the surface of the nerve.² Any similar tendency to non-random distribution in the rabbit sciatic nerve might go some way toward explaining the sequence observed in these latest laboratory observations.

The comment that decrease in oxygen partial pressure of the perfusing medium causes large-diameter fibers to fail before small-diameter fibers, probably does not fully represent the complexity of the situation. There is good evidence that (in cat splanchnic nerve) anoxia blocks different groups in the order: B, slow A, fast A, and C.³ Thus, the

vulnerability of nerve fibers does not always scale according to size, and a unitary explanation for the relative vulnerabilities of different fiber groups, such as proposed by Gissen *et al.*, apparently does not yet account for all the relevant facts.

B. RAYMOND FINK, M.D.
Professor of Anesthesiology
University of Washington
Seattle, Washington 98195

REFERENCES

1. Gissen AJ, Covino BG, Gregus J: Differential sensitivities of mammalian nerve fibers to local anesthetic agents. *ANESTHESIOLOGY* 53:467-474, 1980
2. Evans DHR, Murray JG: Histological and functional studies on the fiber composition of the rabbit vagus nerve. *J Anat* 88:320-327, 1954
3. Lundberg A: On the mechanism of differential sensitivity of mammalian nerve fibers to depolarizing agents. *Acta Physiol Scand* 26:156-173, 1952

(Accepted for publication January 26, 1981.)

Intranasal Nitroglycerin (NTG) during Infrarenal Aortic Cross-Clamp

To the Editor:—Hill *et al.*,¹ reported that intranasally administered NTG (0.8 mg, two tablets dissolved in 1 ml of 0.9 per cent NS) reached its peak level in central venous blood at 1 min and remained above therapeutic levels for 8 min. The authors confirmed that when the oral mucosa is not a predictable route of absorption for sublingual NTG because of the presence of an endotracheal tube, airway, secretions, and sometimes dryness (*i.e.*, under general anesthesia), nasal mucosa is a convenient alternative. NTG relieves angina pain if the patient is not under anesthesia, probably because it decreases myocardial wall tension [primary venodilation results in decreased pulmonary capillary wedge pressure (PCWP)] and redistributes perfusion favorably to ischemic endocardium.

Infrarenal aortic cross-clamp during abdominal aortic aneurysmectomy or aortofemoral bypass procedure increases systolic blood pressure (SBP) and PCWP, produces little change in heart rate, and de-

creases cardiac output, all due to the sudden increase in after-load. The maximum rate of these hemodynamic changes occurs within 1 to 3 min after clamp.^{2,3} Because intranasal NTG works within 2 min and relieves the cardiovascular stress associated with aortic cross-clamp by decreasing arterial BP and PCWP, this technique has been used in our institution. Data are now being accumulated to document our experience.

Although nasal NTG can be given after SBP is maximally raised, the preferred way is to give it as soon as SBP begins to rise, to match the time course. Usually, 0.4 mg NTG (*i.e.*, 0.5 ml of the dissolved solution squirted into the free nostril while the other is occupied by a nasogastric tube) lowers SBP to near preclamp level, with little change in the heart rate. If the blood pressure is still high after 2 to 3 min, compared to preclamp level, another 0.5 ml is given. The effect seems to last 10 to 15 min. Because it changes the myocardial oxygen demand-supply ratio

favorably, NTG is likely to be especially useful in patients who are taking sublingual NTG preoperatively, or who have little functional reserve of the left ventricle.

With the potential decrease of preload with NTG, questions have been raised about the change of cardiac output. When cardiac output was measured in patients in whom a Swan Ganz catheter was used, it was near or above the preclamp level. The only precaution to be taken in using NTG is that the patient should be kept at the upper normal limit of preload by monitoring CVP or PCWP so that blood pressure and cardiac output do not fall below the preclamp level. If one compares nasal NTG with the iv infusion technique in this particular situation, it has several advantages. It can be prepared freshly in a matter of seconds, it does not need to be sterilized, it is inexpensive, and it eliminates the time required to find the optimal infusion rate. Intranasal NTG can be used in many other situations during anesthesia, such as at the end of a coronary bypass operation. If the patient's BP happens to rise just before or during transfer from the operating room to the special care unit (which is an awkward time to infuse a

vasodilator via tangled noodles of iv tubings) a small squirt in the nose solves the problem.

G. JAE MOON, M.D.
Senior Staff Anesthesiologist

JIN G. KIM, M.D.
Senior Staff Anesthesiologist

FRANKLIN T. COOKINHAM, M.D.
Chairman

*Department of Anesthesiology
Henry Ford Hospital
Detroit, Michigan 48202*

REFERENCES

1. Hill AB, Bowley CJ, Nahrwold ML, et al: Intranasal administration of nitroglycerin. *ANESTHESIOLOGY* 51: S67, 1979
2. Meloche R, Potecher T, Audet O, et al: Haemodynamic changes due to clamping of the abdominal aorta. *Can Anaesth Soc J* 24:20-34, 1977
3. Silverstein PR, Caldera DL, Cullen DJ, et al: Avoiding the hemodynamic consequences of aortic cross-clamping and unclamping. *ANESTHESIOLOGY* 50:462-466, 1979

(Accepted for publication January 28, 1981.)