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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.

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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