ng/ml. These results are contrary to our own experience, which suggests that much higher doses (at least 10–20 times the dose used in this study) and plasma concentrations are necessary to completely abolish somatic pain responses in all dogs.

However, the difference may be related to the methods that we and Arndt *et al.* use to evaluate pain responses in the dog. Quasha *et al.*[2] suggest that a supramaximal stimulus is obtained in a dog by clamping the tail or using a subcutaneous electrical current (50 V at 50 Hz for 10 ms). When a tail clamp is used, they state that the hemostat must be "full length," applied close to the base of the tail, and clamped to "full ratchet lock." Furthermore, they define a positive response to be "a gross purposeful muscular movement, usually of the head or extremities." Of course, these criteria were established for determining the MAC of an inhalation anesthetic. Unfortunately Arndt *et al.* do not state what kind of a hemostat was employed nor whether it was clamped at the base of the tail and to "full ratchet." They do indicate that the clamp was "applied for a few seconds."

Although time of clamp applications and method of application (over 10–20 s *versus* immediate clamp to full ratchet) were never addressed by Quasha *et al.*,[2] we believe they are also important, especially when evaluating the analgesic/anesthetic effects of opioids or other intravenous anesthetics in the dog. We believe that dogs given large doses of opioids often may not respond to a tail clamp slowly applied (over 10–20 s) or maintained clamped for only a few seconds. These same animals will respond when the clamp is immediately applied to full ratchet and allowed to remain in place for 30 s, especially if the tail is moved continuously with the hemostat for the duration of stimulation. Thus, slowly applying a hemostat and maintaining it at full ratchet for only a few seconds (even if it is applied at the base of the tail, over the bone) *may not be a supramaximal stimulus,* at least with opioids and intravenous anesthetics in the dog. Eger and colleagues[3] believe that the same may be true when evaluating the minimal alveolar anesthetic concentration of inhalation anesthetics. Indeed, they also recommend that the hemostat be large (10 inches) and applied to the base of the tail for at least 30–40 s, and that the tail be moved continuously with the hemostat for the duration of the stimulation. They also cautioned that a negative response at 10 s of stimulation might become positive if the stimulus were maintained for 30 s.

We are anxious about the impact of the report of Arndt *et al.*[1] Their data suggest that 167 μg/kg of fentanyl is "anesthetic" in the dog. Furthermore, a cursory reading of the paper might lead to a conclusion that fentanyl produces about the same responses in the dog as it does in humans. Indeed, the report seems to clearly suggest the opposite, although it is not emphasized. The dogs' rspiratory and circulatory changes and responses to pain all returned to control values 30 min after the last injection of fentanyl. More impressively, Pa_{CO_2} never increased as high as 50 mmHg. These are certainly markedly different responses to 167 μg/kg of fentanyl than would occur in humans and, in our opinion, need to be emphasized.

In view of all of the above, we have difficulty accepting the authors' conclusion that fentanyl exerts its full analgesic, respiratory, and circulatory actions in an identical range of plasma concentrations and that these occur at a similar range of concentrations in dogs and humans. We are afraid that one result of this article could be that inexperienced investigators will begin using 100–200 μg/kg of fentanyl as an anesthetic (in dogs) and cause undue pain and suffering in this most ubiquitous experimental animal.

THEODORE H. STANLEY, M.D.
Professor of Anesthesiology/Surgery

J. DAVID PORT, B.S.
Technical Research Supervisor

Department of Anesthesiology
The University of Utah School of Medicine
50 North Medical Drive
Salt Lake City, Utah 84132

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In reply:—Dr. Stanley and Mr. Port have expressed their anxiety about the impact our recent article[1] might have on the "inexperienced investigator." Obviously, one could draw unjustifiable conclusions by unduely extrapolating from dogs to humans or by confusing analgesia with anesthesia. Therefore, I appreciate the opportunity to reply to these most important questions.

To be clear: We did not claim to have applied a

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"supramaximal painful stimulus" as stated in the letter. Nor did we say that fentanyl produces "about the same responses" in humans and dogs. Nor did we ignore the rapid decay of the action of fentanyl.

In our unanesthetized dogs (pets), a hemostat (15 cm in length) was clamped quickly and full length to the skin at the base of the tail for 1–2 s in the awake dogs, but full ratchet for up to 10 s under the action of fentanyl. This evoked in the awake animals predictable increases in heart rate and blood pressure together with purposeful movements. Whether we stimulated supramaximally we do not know, but in agreement with others,[2] we found the evoked autonomic responses to be useful and quantifiable "pain" indices. As we have shown, the responses so provoked were suppressed by fentanyl in a concentration-related manner and were completely abolished at and around plasma concentrations of 30 ng/ml where the cardiorespiratory side effects also had reached plateaus. Liu *et al.*[3] certainly used enormous doses of fentanyl (0.05–2 mg/kg) in dogs, but they only looked at the cardiovascular not the analgesic effects.

Our conclusion that "in dogs plasma concentrations in the order of 30 ng/ml are sufficient to reach the full action of fentanyl" is therefore supported by the data presented. It is also true that humans are rendered unconscious and unresponsive to noxious stimulation (absence of heart rate and blood pressure increases during tracheal intubation) at similar plasma concentrations.[4] Thus, dogs and humans do seem to require about the same fentanyl plasma concentrations for abolishing the autonomic responses to noxious stimulation, even though there are obvious differences in the kinetics of fentanyl. Clearly, the fentanyl plasma concentrations decay (for unknown reasons) much more rapidly in dogs than in humans, which would necessitate more frequent reinjections or higher rates of infusion to maintain a certain plasma level in dogs.

Finally, I'd like to point out an essential semantic aspect of this dispute. Stanley and Port repeatedly use the term "anesthetic," which we strictly avoided in our article because opiates and general anesthetics are not the same. The former exert their highly specific actions via receptors confined to the nervous system, whereas the latter impair rather unspecifically cell functions in general and precipitate a comatose state. Therefore, when anesthesia is required, we recommend anesthetics alone or together with opiates but not opiates alone.

I realize that our observations hardly can be reconciled with Dr. Stanley's belief in the use of a large single dose of the highly lipophilic fentanyl without nitrous oxide for the purpose of "stress-free" anesthesia.

JOACHIM O. ARNDT, M.D.
Professor of Experimental Anesthesiology and Physiology
Department of Experimental Anesthesia at the Center of Anesthesia at the University of Düsseldorf
Düsseldorf, West Germany

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Lightwand-guided Nasotracheal Intubation Is an Effective Technique

To the Editor:—"A Complication of Lightwand-guided Nasotracheal Intubation" reported by Stone *et al.*[1] in the December issue discloses a disadvantage of this technique, but it seems to miss a major point. That is, a very difficult technical feat was successfully accomplished by the transillumination technique of intubation.

We have used this method of intubation with a Flexilum 10″ Surgical Light® over the past several years on 78 of our most difficult intubations and have not yet failed. This technique only takes a few seconds for intubation and is not difficult to master. To minimize the possibility of disconnection of the bulb, we are careful not to allow the light source tip to protrude past the end of the endotracheal tube.

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