

## In Response

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How resourceful of the authors to segue effortlessly from one discussion of the implications of transforaminal techniques used for benign radicular pain (1,2) to the novel and somewhat heroic use of transforaminal phenol injections in a patient suffering from unremitting terminal cancer pain (3)! While we perhaps naively presumed that the obvious ramifications of neuraxial neurolysis would not be conjoined with glucocorticoid treatment, we must acknowledge our imprudent miscalculation. A few comments are in order.

In our case report cited, 5% phenol in glycerin was used for transforaminal neurolysis (3). Studies have demonstrated that at these concentrations, destruction of nociceptive fibers prevails with minimal likelihood of causing axonal abnormalities, nerve root damage, spinal cord infarcts, arachnoiditis or meningitis (4,5). Indeed, for peripheral neurolysis, 5% phenol is equipotent to 40% alcohol (6), a concentration rarely used for neuraxial neurolysis due to its relative lack of efficacy (7). A higher affinity of phenol for vascular tissue than for neuronal tissue has been suggested (5). However, Racz and associates (8) studied the morphologic changes that occurred following epidural and subarachnoid phenol injection. They found that massive tissue destruction was present following subarachnoid injection as compared to epidural injection despite intact vasculature in areas of spinal cord destruction (8). These findings support a direct neurotoxic effect of phenol rather than an effect secondary to vascular destruction (4,9).

The authors cite a prior case report of paraplegia following intercostal injection of aqueous phenol solution as being "probably" the result of a transforaminal phenol neurolysis wherein vascular penetration

and injection occurred (10). What the authors fail to reconcile are at least 2 salient points; 1) That the concentration employed was 7.5% aqueous phenol (up to 50% more toxic than non-aqueous) (10), which we above-acknowledge to be inherently more dangerous than the 5% in glycerin used in our report (3), and 2) In the case report cited, there was an actual measurement of CSF concentrations of phenol, much more consistently approximating an intrathecal absorption of this drug than what would have occurred with an intravascular injection (7,10). Their statement that "Accordingly, we have to conclude that in a previous, although inadvertent, transforaminal phenol neurolysis, a sudden anterior spinal syndrome ensued" is not supported by the case report itself, nor by the known, published pharmacological toxicology of aqueous phenol preparations.

Furthermore, their reliance upon a letter to the editor (11) postulating a vascular-induced mechanism of spinal cord injury occurring in the Katz, et al study of nonhuman primates exposed to epidural aqueous phenol (12), once again is somewhat misleading, as it is merely an untested hypothesis being suggested by those authors, wherein they referenced articles concerned with subarachnoid and not epidural phenol administration (13). Indeed, if one investigates the literature used in support of Wilson and Kaplan's hypothesis (11), it is duly noted that the subarachnoid phenol concentrations studied in this cat model (13) were 7.5% (aqueous and in glycerol) and 15% (aqueous and in glycerol). Baxter and Schacherl state; "...an 0.5% aqueous solution of phenol is more potent in terms of nerve blocking and damaging potential than a 7.5% or 10% solution of phenol in glycerol or Myodil" (13).

Additionally, the present authors misrepresented the findings of Brown and Rorie (14) in stating that "... phenol produces sustained contractile responses compared to norepinephrine-induced controls". In fact, what Brown and Rorie did find was that an 8%, 9% or 12% concentration of aqueous phenol was required to produce sustained contractile responses compared to norepinephrine-induced control contractile responses (14). Brown and Rorie (14) stated: "The magnitude of phenol-induced contractile response was directly related to concentration; 1%, 3%, and 6% phenol caused a small, transient contractile response." Once again, we humbly suggest that the authors re-read our case report, wherein we were clearly apprised that phenol exerts a dose-dependent and concentration-dependent effect and we utilized this information when carefully and thoughtfully considering whether or not to proceed with this approach of transforaminal injection of 5% phenol in glycerin.

In summary, we believe the authors have performed a potential disservice to advanced interventional pain physicians by misrepresenting the preponderance of scientific literature that clearly supports

the judicious and extremely selective use of neurolysis in managing pain associated with both non-malignant conditions (15) as well as that encountered in end-of-life scenarios. While we do not expect everyone to embrace our heroic approaches to managing severe, unremitting cancer pain, we do however expect fair and balanced assessments of new and novel strategies aimed at doing so.

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