Spinal anaesthesia: a century of refinement, and failure is still an option

On August 24, 1898, August Bier and his assistant Hildebrandt undertook ‘experiments on [their] own bodies’ which were part of their historic initial investigations of spinal anaesthesia. Bier’s description of these experiments is notable for the manner in which he documented the lack of sensibility after injection of cocaine into Hildebrandt’s subarachnoid space, which included a burning cigar, a strong blow to the shin with an iron hammer, and strong pressure and traction on the testicles, none of which provoked pain. Absent from this report are descriptions of similar assessments being performed by Hildebrandt on Bier. This was not out of deference to Bier, but rather the Pravaz syringe failed to fit on the needle, and a significant amount of the cocaine intended for Bier’s subarachnoid space was lost, resulting in a failed block.

Although the aetiology of this historic failure is no mystery, a particularly perplexing and frustrating problem with spinal anaesthesia is the occasional failure to achieve adequate sensory block, despite an apparently orthodox injection of an adequate dose of local anaesthetic. Believing certain subjects to be ‘hyper-resistant’ to spinal anaesthesia, Sebrechts coined the term ‘rachi-resistance’ in 1934 to describe this phenomenon. He postulated that this aberration reflected a ‘peculiar idiosyncrasy which renders the nerve roots of certain individuals insensitive or resistant to the action of anaesthetic solutions’. This concept garnished a modicum of support, some proponents proposing that this effect might be due to reduced permeability of the roots. However, most were critical, suggesting such failures more likely reflect inactive solution, or a variety of anatomic variations or abnormalities such as arachnoid adhesions, unusual arrangements of the dentate ligament, or a dilated lower end of the thecal sac. Ultimately, the term ‘rachi-resistance’ disappeared from the anaesthesia literature.

A review article and two clinical studies in this issue of the British Journal of Anaesthesia attempt to shed light on this elusive subject. In their systematic review, Fettes and colleagues dissect the spinal technique into its sequential components, providing a framework to understand the mechanisms that may account for failure. Such information is a prerequisite for maximizing success rate, and for rational clinical management of a failed block. In the first of two related research papers, Ruppen and colleagues explore the cerebrospinal fluid (CSF) concentrations of anaesthetic associated with the development of adequate spinal anaesthesia after administration of plain bupivacaine 0.5%. Their principal finding was the enormous range of concentrations (26–781 μg ml⁻¹), which did not correlate with the level of block. Further, the variability in samples obtained at randomized time-points between 5 and 45 min post-injection was so large as to prohibit any meaningful pharmacokinetic assessment. An even larger variability (3.36–10² μg ml⁻¹) was seen in the second study where samples were obtained from patients who had received the same anaesthetic but had inadequate anaesthesia. The range of concentrations from these two studies overlapped, with only four of the 20 failed spinals having a CSF concentration below any associated with successful spinal anaesthesia.

Although these findings may appear incomprehensible, they can be understood by appreciating the limitations of single-site assessments of CSF anaesthetic concentrations, a point well appreciated and considered by the authors of these two research papers. Because local anaesthetic will often distribute unevenly within the subarachnoid space, the concentration at a single point cannot be used to determine the true volume of distribution, nor can concentrations found below the conus reliably predict those at more rostral locations servicing clinically relevant dermatomes. Consequently, faced with a relatively low concentration, clinical correlation or imaging is needed to permit any distinction between a poor injection, a large volume of CSF below the conus, extensive spread, or an anomalous sample. With the exception of technical failure, similar considerations apply to interpretation of high concentrations.
Despite these limitations, there are some interesting insights that can be gleaned from the data. For example, the highest concentration in any sample came from a patient with inadequate anaesthesia, supporting the concept of maldistribution as an aetiology of failure, a point highlighted by the authors. A similar observation was made in a very early study of hyperbaric lidocaine administered through continuous spinal catheters. Of 16 patients receiving an initial injection of lidocaine 150–200 mg, there was only one failure requiring general anaesthesia. The anaesthetic concentration in the CSF sample from this patient was roughly 2 SD above the mean, and an X-ray demonstrated the catheter to be curled with the end resting in the cul-de-sac at the level of the second sacral vertebra. Such findings are also consistent with data obtained from in vitro investigations modelling subarachnoid anaesthetic distribution.

That failure from maldistribution cannot be readily distinguished clinically from technical failure has important implications for management of a failed spinal. In such cases, repetitive injection can distribute in the same pattern, reinforcing the already high concentrations, and potentially resulting in neurotoxic injury. Review of the closed claims database and case reports appear to confirm these concerns. On the basis of these considerations, we previously made suggestions for management of a failed spinal, which included evaluation of the likelihood of technical error and adjustment of dosage for the second injection. However, these recommendations impart significant delay, as one must allow sufficient time for achievement of near-maximal block before assessment of sensory anaesthesia. A more efficient—and probably much safer—alternative is to simply limit the combined anaesthetic dosage to the maximum reasonable to administer as a single intrathecal injection.

In four of the failed spinals, there was no detectable sensory anaesthesia, despite CSF concentrations that exceeded the lowest value associated with a completely successful block. And, in one case, the concentration was above the 5th percentile for successful spinal anaesthesia, the cut-off (albeit somewhat arbitrary) that the authors thought should be adequate for successful spinal anaesthesia. Although it is possible that this reflects the limitations of sampling and errant concentrations obtained in the study of successful spinals, it is intriguing to consider the possibility that these patients were actually resistant to the anaesthetic. Such consideration is more than a theoretical possibility, as there are well-described mutations of the voltage-gated sodium channel that can profoundly affect function, including altered sensitivity to anaesthetic.

Further advances in pharmacogenetics might thus identify genetic diversity as a factor in spinal anaesthetic failure, justifying a reintroduction of the term ‘rachi-resistance’ into the language of anaesthesia. Who knows, Sebrechts belief that there are familial tendencies in susceptibility, and that the ‘the Anglo-Saxon has resistance to larger doses than the Italian’ might just turn out to be correct.

K. Drasner

University of California
San Francisco
CA 94110
USA
E-mail: kdrasner@anesthesia.ucsf.edu

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