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NEUROLOGIC OUTCOME AFTER INTRANEURAL AND PERINEURAL SCIATIC NERVE BLOCK IN PIGS

NEUROLOŠKE POSLJEDICE INTRANEURALNE I PERINEURALNE BLOKADE ISHIJADIČNOG ŽIVCA KOD SVINJA

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Kratak sadržaj - Istraživanja na životinjama su pokazala da intraneuralna primjena lokalnih anestetika može uzrokovati mehaničke ozljede i ishemiju živčanih vlakana. Prethodne studije su, međutim, koristile male životinje i klinički irelevantnu brzinu ubrizgavanja ili opremu. Naša hipoteza je da je intraneuralna injekcija praćena većim injekcionim pritiskom i da dovodi do neurološkog oštećenja kod svinja.

Istraživanje je rađeno na 10 svinja. Nakon opće anestezije,obostrano su prikazani ishijadični nervi (n = 20). Pod izravnom kontrolom, igla promjera 25 gejdža plasirana je ekstraperineuralno (n = 10) ili subperineuralno (n = 10) i aplicirano je 4 ml 2% lidokaina, pomoću automatske infuzione pumpe (15 ml / min). Podaci o injekcionom pritisku su dobiveni pomoću inline manometra povezanog s računalom preko analogno-digitalne kartice. Nakon ubrizgavanja, životinje su probuđene i podvrgnute serijskim neurološkim pregledima, tokom 24 sata nakon intervencije.

Sve, osim dvije perineuralne injekcije rezultirale su injekcionim pritiskom manjim od 20 psija. Nasuprot tome, intraneuralne injekcije su rezultirale značajno većim injekcionim pritiskom. Kod 7 (70%) intraneuralnih injekcija, pritisci su bili veći od 20 psija (20-50 psija). Neurološka funkcija vratila se na početnu u roku od 24 sata kod svih

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ishijadičnih živaca koji su primili perineuralnu injekciju. Nasuprot tome, rezidualno neurološko oštećenje bilo je prisutno kod 7 ishijadičnih živaca nakon intraneuralne injekcije; rezidualno neurološko oštećenje bilo je povezano s injekcionim pritiskom većim od 20 psija.

Rezultati istraživanja sugeriraju da visok injekcioni pritisak tokom intraneuralne injekcije može upućivati na intrafascikularnu injekciju, i nagovijestiti razvoj neurološke ozljede.

Ključne riječi: nervni blok, injekcioni pritisak, neurološka ozljeda, svinje

Abstract - Studies in animals have suggested that intraneural application of local anesthetics may cause mechanical injury and pressure ischemia of nerve fascicles. Previous studies, however, have used small animal models and clinically irrelevant injection speed or equipment. Our hypothesis is that an intraneural injection is heralded by higher injection pressure and leads to neurologic impairment in pigs.

Ten pigs of mixed breed were studied. After general anesthesia, the sciatic nerves (n = 20) were exposed bilaterally. Under direct vision, a 25-gauge insulated nerve block needle was placed either extraperineurally (n = 10) or subperineurially (n = 10), and 4 ml of preservative-free lidocaine 2% was injected using an automated infusion pump (15 ml / min). Injection pressure data were acquired using an in-line manometer coupled to a computer via an analog-to-digital conversion board. After injection, the animals were awakened and subjected to serial neurologic examinations during the 24 post-intervention hours.

All but two perineural injections resulted in injection pressures below 20 psi. In contrast, intraneural injections resulted in significantly higher peak pressures. In 7 (70%) intraneural injections, the injections pressures were over 20 psi (20-50 psi). Neurologic function returned to baseline within 24 hours in all sciatic nerve receiving perineural injections. In contrast, residual neurologic impairment was present in 7 sciatic nerves after intraneural injection; residual neurologic impairment was associated with injection pressures > 20 psi.

The results indicate that high injection pressure during intraneural injection may be indicative of intrafascicular injection and may predict the development of neurologic injury.

Key words: nerve block, injection pressure, neurologic injury, pigs

Introduction

Intraneural injections of local anesthetics have long been recognized as a cause of nerve injury after peripheral nerve blocks (1, 2, 3). Studies in animals have suggested that intraneural application of local anesthetics may cause mechanical injury and pressure

ischemia of nerve fascicles (1, 2, 3, 4). Previous studies, however, have used small animal models and clinically irrelevant injection speed or equipment (5). Consequently, the results of those studies remain of questionable relevance to clinical practice. In this study we used equipment and injection methods in common clinical use to study the consequences and pressure dynamics of intraneural injection. Our hypothesis is that an intraneural injection is heralded by higher injection pressure and leads to neurologic impairment in pigs.

Materials and methods

The study was conducted in accordance with the principles of laboratory animal care and was approved by the Laboratory Animal Care and Use Committee. Ten pigs of mixed breed (21-26 kg, 4-6 months old) were studied. After general anesthesia, the sciatic nerves (n = 20) were exposed bilaterally. Under direct vision, a 25-gauge insulated nerve block needle (ProBlock, LifeTech, Stafford, TX) was placed either extraperineurally (n = 10) or subperineurially (n = 10), and 4 ml of preservative-free lidocaine 2% was injected using an automated infusion pump (15 ml / min) (PHD 2000, Harvard Apparatus, Holliston, MA). Injection pressure data were acquired using an in-line manometer (PG 5000, PSI-Tronics Tehnologies Inc., Tulare, CA) coupled to a computer via an analog-to-digital conversion board (DAQ 6023, National Instruments, Austin TX).

After injection the animals were awakened and subjected to serial neurologic examinations during the 24 post-intervention hours. Neurologic examination included assessment for the presence and severity of paresis (0, no paresis; 1, mild paresis; 2, pronounced paresis; 3, flaccid extremity).

Statistical analysis has been executed by using SPSS program, version 11.5. Maximum pressure value during intraneural and perineural injection has been compared using paired t-test. The occurrence of neurological injuries is compared between intraneural and perineural injections using McNemar's test for paired proportions. P value < 0.01 is considered significant.

Results

Pressure data

All but two perineural injections resulted in injection pressures below 20 psi (mean ± SD, 15.8± 6.62 psi), (Fig. 1).

In contrast, intraneural injections resulted in significantly higher peak pressures (mean ± SD, 28.35 ± 11.58 psi), (Fig. 2). In 7 (70%) intraneural injections, the injections pressures were over 20 psi (20-50 psi), (Fig. 2). The intraneural injection group had

significantly higher values of injection pressure compared to the perineural injection group of pigs ($p<0.01$).

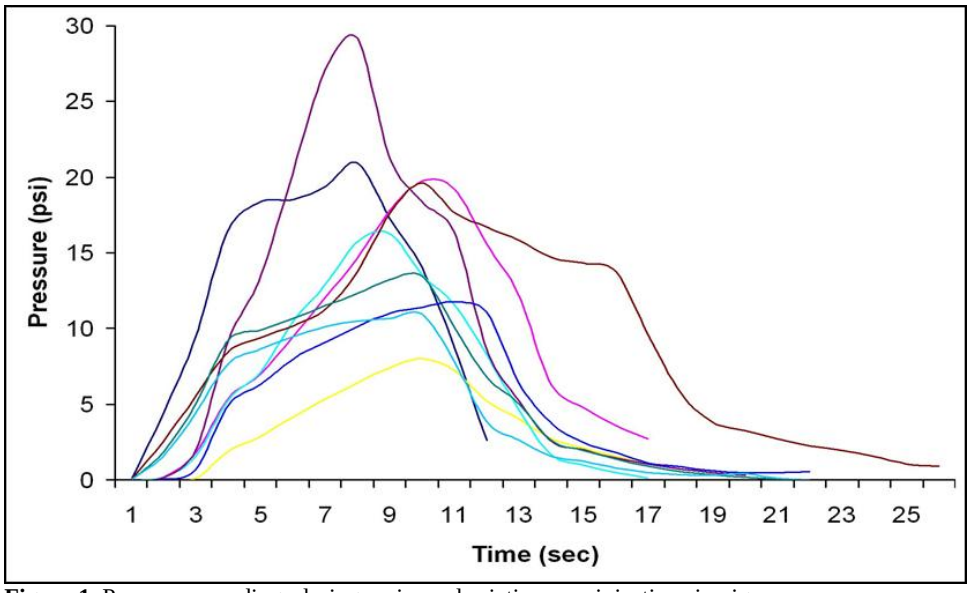

Figure 1. Pressure recordings during perineural sciatic nerve injections in pigs
Slika 1. Promjena pritiska tokom perineuralne injekcije ishijadičnog živca kod svinja

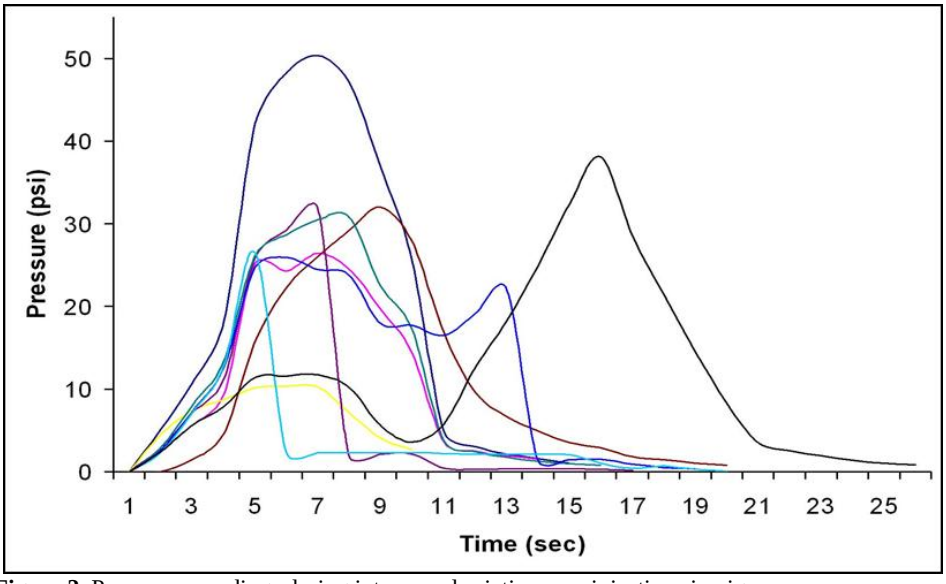

Figure 2. Pressure recordings during intraneural sciatic nerve injections in pigs
Slika 2. Promjena pritiska tokom intraneuralne injekcije ishijadičnog živca kod svinja

Most injections were characterized by slightly higher pressure at the beginning of the injection resulting in pressure peak followed by lower pressure throughout the remainder of the injection.

Neurologic data

On awakening from general anesthesia, all pigs exhibited motor signs of sciatic nerve blockade during the neurological examination. However, the recovery of the neurologic function varied between the groups. Using paresis as the sentinel measure of injury we saw that neurologic function returned to baseline within 24 hours in all sciatic nerve receiving perineural injections (Fig. 3). In contrast, residual neurologic impairment was present in 7 sciatic nerves after intraneural injection; residual neurologic impairment was associated with injection pressures above 20 psi (Fig. 3). The animals showed a complete paralysis, which turned into a pronounced paresis at the end of the observation. More specifically, all sciatic nerves for which high injection pressure was recorded during intraneural injection, exhibited impairment of neurologic function persisting beyond 24 h after the injection. In contrast, sciatic nerves receiving intraneural injections for which low injection pressure was recorded, recovered neurologic function within 24 h after the injection.

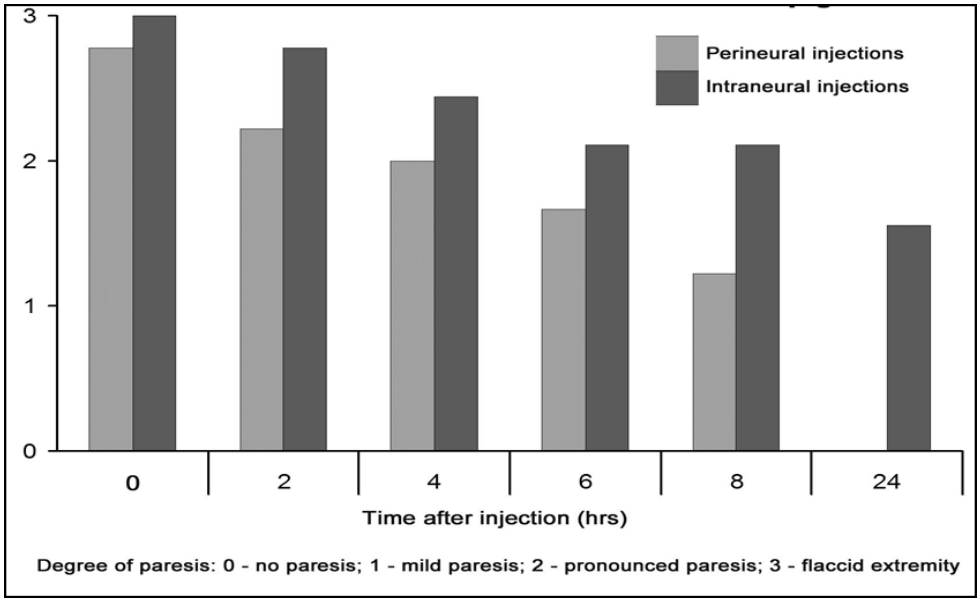

Figure 3. Duration and degree of paresis after perineural and intraneural sciatic nerve injections
Slika 3. Trajanje i stepen pareze poslije perineuralne i intraneuralne injekcije ishijadičnog živca

Discussion

In the last few decades there has been a great development of regional anesthesia; all the postulates are defined and all the techniques of usage are perfected. The world trend of favoring various techniques of regional anesthesia is a result of the advantages that the regional anesthesia comes with, especially in comparison with the general anesthesia like avoiding hemodynamic instability and lung complications and enabling faster mobilization and earlier release of the patients to their homes (6). In this healthcare environment, continual assessment of the safety and efficacy of clinical practice is critical. Neurologic complications of regional anesthesia can result in disability and are feared by patients and clinicians. Our study shows that detection of pressure during peripheral nerve blocks is unique as a nerve localizing technique in terms of being able to avoid needle-nerve contact and potentially prevent direct trauma to nerves.

Unintended intraneural injections of local anesthetics and other theraputic agents has long been established as a mechanism of nerve injury (1). Intraneural injection of local anesthetic in sciatic nerve block model in pigs is associated with high injection pressure (> 20 psi) and delayed neurologic recovery. These findings support the concept of poor compliance of nerve fascicles and suggests that injection pressure > 20 psi can be used as a marker of intrafascicular injection. If these results are applicable to clinical practice, avoiding excessive injection pressure during nerve block administration may help to reduce the risk of neurologic injury.

Neurological injury after peripheral nerve blocks is uncommon. Our data suggest that neurological injury does not always develop, even after intraneural injection (1, 4, 7). Our data suggest that such blocks may be the results of the intraneural but extrafascicular deposition of local anesthetic. Fascicular injury with the neurologic deficit appears to occur only when an intraneural injection results in a high injection pressure, suggesting that a high injection pressure may serve as a marker of intraneural injection (1, 4).

In our study all limbs recovered within 24 hours after perineural injections following a model of a normal clinical nerve block injections. However, neurologic deficits persisting after 24 hours after injections were noted in all intraneural injections that resulted in high injection pressure (> 20 psi, seven of 10 injections).

The mechanism of injury with intrafascicular injection includes a combination of direct mechanical injury, changes in the permeability of the blood – nerve barrier, associated oedemea, pressure ischemia, epinephrine – mediated vasoconstriction and increased endoneural fluid pressure, all of which may contribute to the nerve injury (8, 9, 10).

Conclusion

The pig model of intraneural injection suggests that intraneural injection of local anesthetic does not always lead to nerve injury. High injection pressure during intraneural injection may be indicative of intrafascicular injection and may predict the development of neurologic injury. If these results are applicable to clinical practice, the avoidance of excessive injection pressure during peripheral nerve blocks may reduce the risk of neurologic complications.

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Conflict of Interest: Non Declared
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