

Evaluation of intraabdominal hypertension and genitofemoral nerve motor conduction

Özlem BOYBEYİ^{1*}, Elem İNAL², Yasemin DERE GÜNAL¹, Mustafa Kemal ASLAN¹, Gülümser AYDIN², Tutku SOYER³

¹Department of Pediatric Surgery, Faculty of Medicine, Kırıkkale University, Kırıkkale, Turkey

²Department of Physical Medicine and Rehabilitation, Faculty of Medicine, Kırıkkale University, Kırıkkale, Turkey

³Department of Pediatric Surgery, Faculty of Medicine, Hacettepe University, Ankara, Turkey

Received: 16.11.2014 • Accepted/Published Online: 16.02.2015 • Final Version: 05.01.2016

Background/aim: The aim of this study was to electrophysiologically evaluate the effect of increased intraabdominal pressure (IAP) on genitofemoral nerve (GFN) motor conduction.

Materials and methods: Seven Wistar albino rats were included. After anesthetization, latency and duration of GFN conduction was recorded with a needle-probe at rest. IAP was increased to 15 mmHg by insufflating atmospheric air with a percutaneous intraperitoneal needle. At 30 min of IAP, GFN motor conduction was recorded. Abdominal pressure was then increased to 20 mmHg. At 60 min, GFN motor conduction was recorded again. The consecutive recordings of latency and duration of GFN conduction (rest, 30 min, 60 min) were evaluated statistically.

Results: There was a significant difference between latencies at rest (1.90 ± 0.22 ms), at 30 min (2.3 ± 0.36 ms), and at 60 min (2.74 ± 0.57 ms) (Friedman test, $P = 0.001$). The latency was significantly increased at 60 min compared to rest (post hoc Tukey test, $P = 0.003$). No similar difference was detected between the recordings at 30 and 60 min. The duration of GFN motor conduction showed no difference between consecutive recordings ($P = 0.067$).

Conclusion: Both increased and prolonged IAP causes prolonged latency of GFN conduction, probably due to a compression effect on GFN. Neuropraxial consequences of increased IAP are thought to be related to the compression effect of peripheral nerves.

Key words: Genitofemoral nerve, intraabdominal pressure, electrophysiology

1. Introduction

Since increased intraabdominal pressure (IAP) causes adverse effects on several systems, it is critical to maintain IAP within normal ranges (1–3). An IAP exceeding 12 mmHg is called intraabdominal hypertension (IAH) and clinical findings of abdominal compartment syndrome occur when IAP exceeds 20 mmHg (2). The role of increased IAP on peripheral neuropraxia has only been reported in laparoscopic procedures. Mouton et al. suggested that postoperative pain due to neuropraxia can be attributed to increased IAP (4). Although a causative relation between IAP and neuropraxia has been suggested in laparoscopic procedures, the effect of increased IAP on peripheral nerves and their motor conduction has not been evaluated electrophysiologically.

The genitofemoral nerve (GFN) is a peripheral nerve having genital and femoral branches. The genital branch has motor and sensory fibers; the former supply the cremaster muscle, whereas the latter supply the skin of

the scrotum and inner thigh (5,6). The motor functions of the GFN can be evaluated by the duration and latency of motor conduction in electrophysiological studies (6). The duration of neural motor conduction is the time interval of muscle action, whereas latency is the time to elicit a response in a muscle by stimulating the nerve (6). The prolongation of latency shows possible peripheral neuropathies (5,6). Although alterations in GFN motor functions do not reveal any remarkable clinical findings, the mass effect of inguinal hernia may cause neuropraxia in the GFN (5). It has been suggested that the type of hernia repair may also affect the motor functions. Celebi et al. reported that laparoscopic procedures cause fewer alterations in GFN motor functions when compared to open repairs (7). However, none of these studies evaluated the role of increased IAP on GFN motor functions.

Therefore, an experimental study was performed to evaluate the effect of increased IAP on GFN motor conduction electrophysiologically.

* Correspondence: ozlemboy80@yahoo.com

2. Materials and methods

The experiments were performed in accordance with the recommendations of the Declaration of Helsinki and the local ethics committee on the care and use of laboratory animals.

Seven Wistar albino rats, weighing 200–250 g, were included in the study. The rats were kept at 22 °C room temperature with a 12-h day/night cycle with free access to tap water and standard food.

The rats were anesthetized with ketamine hydrochloride (40 mg/kg, Ketalar, Pfizer Warner Lambert). They were placed in a supine position, breathing 100% oxygen spontaneously during the experiment. A size 6 French orogastric tube was inserted into the stomach in order to measure the IAP with a manual manometer. A 20-G, 32-mm needle was inserted percutaneously into the peritoneal cavity. In order to prevent respiratory complications of the hypercapnia effect of CO₂ insufflations in spontaneously breathing animals, atmospheric air was used to increase the IAP in all animals (Figure 1). IAP was first increased up to 15 mmHg and held for 30 min, and then it was increased up to 20 mmHg and held for another 30 min.

The latency and duration of GFN motor conduction were recorded at rest, at 30 min of IAH, and at 60 min of IAH with the electrophysiological method, similar to that performed by Ertekin et al. (6).

The GFN was stimulated at the medial site of the anterior superior iliac spine with bipolar surface electrodes (Medelec Small, ref. 16894T; Oxford Medical Instruments, UK) with 0.2 ms in duration and varying intensities (50 Hz to 5 kHz band-pass filter, 200 mV per division amplifier display gain and 200 ms of sweep time).

For recording, a disposable concentric needle electrode (diameter: 0.46 mm; recording area: 0.07 mm²) was inserted into the cremaster muscle (CM) at the level of the

pubic bone after palpating the intracanalicular structures. A ground electrode was placed on the skin (Figure 1).

The latency and duration of consecutive recordings at rest and at 30 and 60 min of insufflations were evaluated statistically with a Friedman test and a post hoc Tukey test by using SPSS 15.0. $P < 0.05$ was considered to be significant.

3. Results

The examples of electrophysiological responses obtained at rest and at 30 and 60 min of IAH are given in Figure 2. The latencies and durations of GFN motor conduction were measured separately and estimated in milliseconds. The measured latencies of GFN motor conduction and the comparison of the measurements are given in the Table.

There was a significant difference between latencies in rest (1.90 ± 0.22 ms), at 30 min (2.3 ± 0.36 ms), and at 60 min (2.74 ± 0.57 ms) (Friedman test, $P = 0.001$). The latency was significantly prolonged at 60 min of insufflations when compared to rest (post hoc Tukey test, $P = 0.003$). However, no similar difference was detected between the recordings at 30 and 60 min or between 30 min and at rest (Figure 3).

The durations of GFN motor conduction were 7.25 ± 0.89 ms at rest, 8.28 ± 2.22 ms at 30 min, and 8.88 ± 1.83 ms at 60 min. The durations showed no significant difference between consecutive recordings ($P = 0.067$).

4. Discussion

IAP exceeding normal limits causes dysfunction in several organs and systems (3). Increased IAP to maintain adequate space for laparoscopic procedures may cause neuropraxia in peripheral nerves and lead to postoperative pain in patients (4). Although the effect of IAH on the

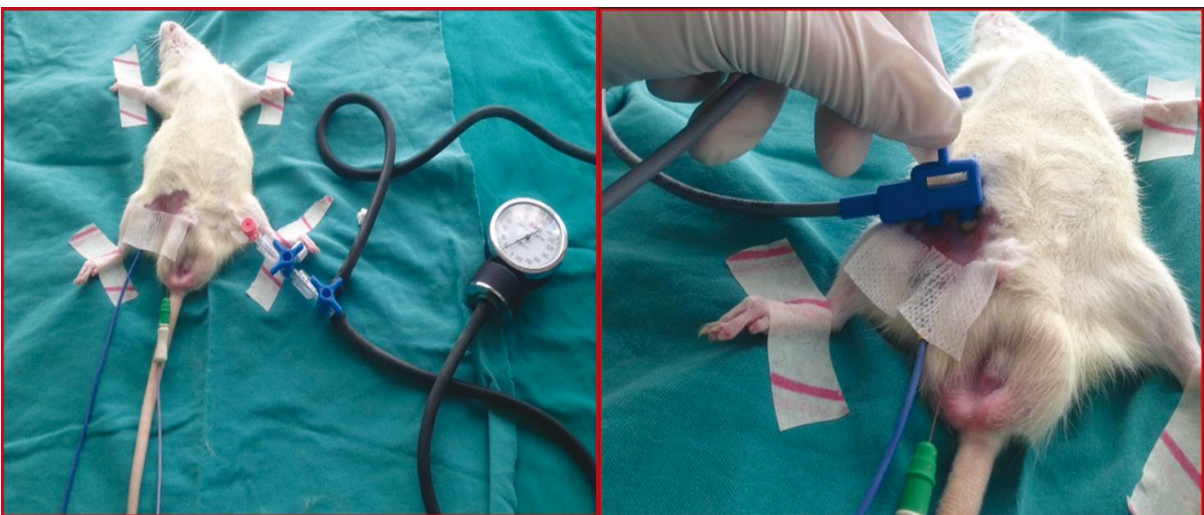


Figure 1. The experimental design: IAP increase (left), electrophysiological examination (right).

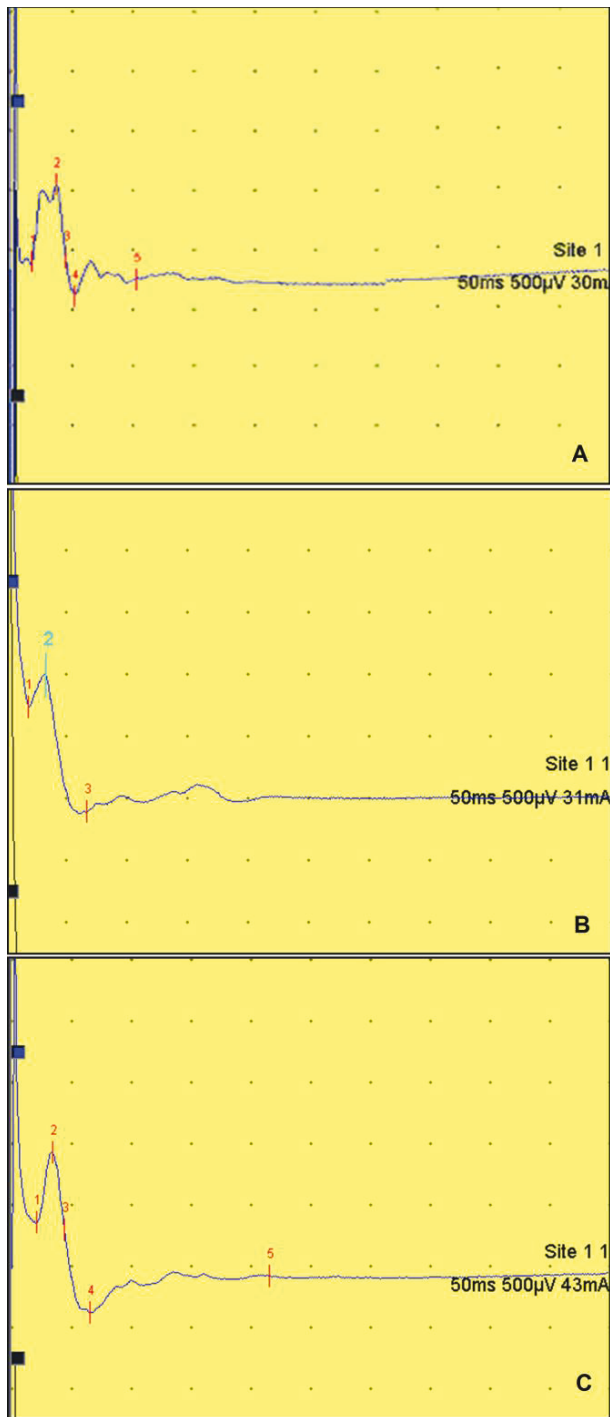


Figure 2. An example of EMG graphics of GFN motor conduction; latency is the period from baseline to first sign, duration is the total period of action (A- at rest, B- at 30 min, C- at 60 min).

phrenic nerve has been evaluated, the effect of increased IAP on the GFN has not been evaluated previously.

The GFN, arising from the first and second lumbar vertebral roots, has genital and femoral branches. The

GFN has several important functions, such as innervations of the cremaster muscle, sensory nerve supply of scrotal and inner thigh skin, thermoregulation of testes, and the second phase of testicular descent (5–6,8).

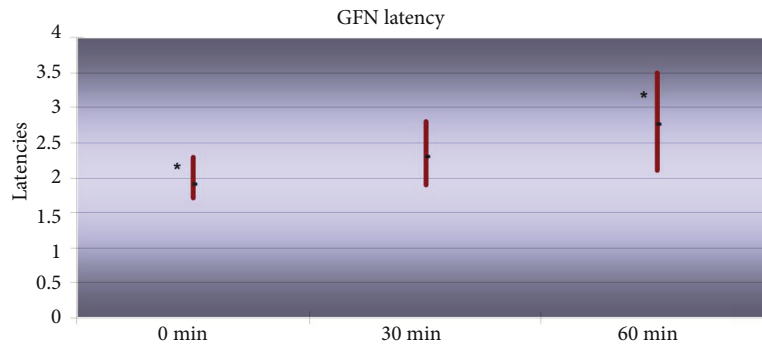
Electrophysiological evaluation of the GFN as described by Ertekin et al. provides us with information about its motor function (6). Compression is one of the most common causes of peripheral nerve injuries. It is known that the degree and duration of pressure are the main determinants of nerve injury. As the degree and/or duration of pressure increases initially demyelination occurs, and it is observed as focal slowing, conduction block, or both in electrophysiological studies. Coexistent axonal loss may or may not be present (6,9–11). Thus, prolonged latency is an indicator of a pathological condition involving the myelin sheath of the peripheral nerve and the significant prolongation of latency determined only in evaluations at 60 min in our study is consistent with these data.

The GFN motor conduction studies were performed in different inguinoscrotal pathologies, such as undescended or retractile testes and inguinal hernia (5,9,12,13). It was shown in previous studies that GFN latency is prolonged in inguinal hernia, which is thought to be due to chronic compression by the hernia sac (9). It was also stated that the GFN could be entrapped by mesh grafts, sutures, staples, or scar tissue after inguinal hernia repair (12). In a recent study, the GFN latency prolongation was found to continue in the early postoperative period, probably due to the continued mass effect or operative manipulation of the nerve (7). In the same study, GFN motor conduction was shown to improve 3 months after hernia repair, revealing that the mass effect has transient consequences on GFN function. Laparoscopic procedures have been recommended as having fewer alterations of GFN motor conduction (7). However, it is not clear whether increased IAP in laparoscopic repair or the continued mass effect results in neuropraxia of the GFN. Therefore, an experimental study evaluating the role of increased IAP on GFN motor functions was performed.

In the present study, we performed IAH by insufflating atmospheric gas and examined the GFN motor conduction electrophysiologically. The prolonged latency of the GFN was only significant between the measurements at rest and 60 min. It can be suggested that not only increased IAP but also prolonged pressures cause deterioration in GFN motor functions. These results showed that increased pressure results in delayed responses in GFN motor fibers. Since the duration of the GFN did not alter in increased pressures, we suggest that increased IAP reflected throughout the abdomen causes compression in the cremaster muscle instead of neuropraxia in the GFN. No clear clinical implication can be attributed to GFN motor dysfunction.

Table. The genitofemoral nerve latency measurements and comparison of the measurements.

At rest	30 min of IAP, 15 mmHg	60 min of IAP, 20 mmHg
1.90 ± 0.22 ms	2.3 ± 0.36 ms	2.74 ± 0.57 ms
Friedman test, P = 0.001		
Post hoc Tukey test		
Rest vs. 30 min		P = 0.191
Rest vs. 60 min		P = 0.003
30 min vs. 60 min		P = 0.137

**Figure 3.** The latencies of GFN motor conduction (*P < 0.05).

We suggest that both increased and prolonged IAP may cause postoperative pain. Although only motor functions of the GFN were evaluated in the current study, sensory fibers may be also affected. The clinical implications of neuropraxia in sensory fibers may have some clinical implications, such as pain or sensory loss after laparoscopy. While we only aimed to evaluate the role of increased IAP on GFN motor functions, affected sensory fibers may cause postoperative pain after laparoscopic procedures. We did not perform CO₂ insufflations since the pH of the medium may also interfere with electrophysiological findings, as reported in the literature (14).

The limitations of our study include the lack of clinical relevance of our experimental results. Our model does not simulate a laparoscopic procedure, and we could not make any conclusions about the GFN motor function in laparoscopic procedures. However, we suggest that

regardless of the cause of increased IAP, GFN motor functions, and especially the latency of elicited response, are significantly prolonged with higher IAP. Further studies are needed to evaluate whether alterations in GFN motor functions are reversible or not.

According to our results, we can conclude that clinical conditions associated with increased IAP, including laparoscopic procedures, result in a transient alteration in GFN motor conduction. We suggest that altered latency of GFN motor conduction not only occurred in increased pressures but was also detected in prolonged IAP.

Acknowledgements

This study was presented at the 14th European Congress of Pediatric Surgery in 2013, Leipzig, Germany, and was supported by the Kırıkkale University Scientific Research Council (2013/01).

References

1. Tihan DN, Erbil Y, Seven R, Arkaya S, Türkoğlu U, Hepgül G, Borucu I. The effect of glutamine on oxidative damage in an experimental abdominal compartment syndrome model in rats. *Turk J Trauma Emerg Surg* 2011; 17: 1–8.
2. Meier C, Contaldo C, Schramm R, Holstein JH, Hamacher J, Amon M, Wanner G, Trentz O, Menger MD. A new model for the study of the abdominal compartment syndrome in rats. *J Surg Res* 2007; 139: 209–216.

3. Meier C, Schramm R, Holstein JH, Seifert B, Trentz O, Menger MD. Measurement of compartment pressure of the rectus sheath during intra-abdominal hypertension in rats. *Intens Care Med* 2006; 32: 1644–1648.
4. Mouton WG, Bessell JR, Otten KT, Maddern GJ. Pain after laparoscopy. *Surg Endosc* 1999; 13: 445–448.
5. Soyer T, Tosun A, Aydın G, Kaya M, Arslan A, Orkun S, Çakmak M. Evaluation of genitofemoral nerve motor conduction in inguinoscrotal pathologies. *J Pediatr Surg* 2008; 43: 1540–1542.
6. Ertekin C, Bademkiran F, Yıldız N, Özdedeli K, Altay B, Aydogdu I, Uludag B. Central and peripheral motor conduction to cremasteric muscle. *Muscle Nerve* 2005; 31: 349–354.
7. Celebi S, Aksoy D, Cevik B, Yıldız A, Kurt S, Dokucu AI. An electrophysiologic evaluation of whether open and laparoscopic techniques used in pediatric inguinal hernia repairs affect the genitofemoral nerve. *J Pediatr Surg* 2013; 48: 2160–2163.
8. Lie G, Hutson JM. The role of cremaster muscle in testicular descent in humans and animal models. *Pediatr Surg Int* 2011; 27: 1255–1265.
9. Soyer T, Tosun A, Keleş I, Inal E, Cesur O, Çakmak M. Electrophysiologic evaluation of genitofemoral nerve in children with inguinal hernia repair. *J Pediatr Surg* 2008; 43: 1865–1868.
10. Oh SJ. General concepts of electrophysiological studies in neuromuscular disease. In: Oh SJ, editor. *Clinical Electromyography. Nerve Conduction Studies*. 3rd ed. Philadelphia, PA, USA: Lippincott Williams & Wilkins; 2003. pp. 14–24.
11. Preston DC, Shapiro BE. Clinical-electrophysiologic correlations: overview and common patterns. In: Preston DC, Shapiro BE, editors. *Electromyography and Neuromuscular Disorders*. 2nd ed. Philadelphia, PA, USA: Elsevier; 2005. pp. 233–251.
12. Bademkiran F, Tataroğlu C, Özdedeli K, Altay B, Aydogdu I, Uludag B, Ertekin C. Electrophysiological evaluation of the genitofemoral nerve in patients with inguinal hernia. *Muscle Nerve* 2005; 32: 600–604.
13. Bingöl-Koloğlu M, Demirci M, Büyükpamukçu N, Tanyel FC. Cremasteric reflex of boys with descended, retractile or undescended testes: an electrophysiological evaluation. *J Pediatr Surg* 1999; 34: 430–434.
14. Soyer T, Aydos TR, Aktuna Z, Korkut O, Osmanoğlu G, Hañcerlioğulları Ö, Çakmak M. The effect of experimentally induced carbon dioxide pneumoperitoneum on intra-abdominal and intra-esophageal pH. *Turk J Med Sci* 2009; 39: 709–712.