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DEVELOPMENT AND CHARACTERIZATION OF A NERVE COMPRESSION APPARATUS AND NERVE RESPONSE TO PRESSURE COMPRESSIONS

Hypothesis / aims of study

Repetitive compression of pelvic structures may cause neural injuries associated with urinary stress incontinence (USI)^[1]. A system was developed to investigate electrophysiological response of isolated nerves to repetitive pressure compression. The goal is to validate the devised system in order to investigate the role of neural injury in USI and to develop strategies for mitigating such injuries.

Study design, materials and methods

The system consists of a sealable electrophysiological chamber and a gas compression apparatus. The compression is supplied by an air compressor and precisely controlled through a series of regulators connected to air-piloted directional control valves. A toggle valve allows input pressure to be controlled by the opening and closing of a valve that leads to a sealed nerve recording chamber. The sealable chamber consists of two air fittings that allow an entry point for the compressed air and a pressure transducer. The amount of pressure inside the chamber is adjusted by a precision regulator, which comes equipped with a locking nut to prevent inadvertent pressurization. Once the toggle valve is returned to its normally closed position the chamber returns to atmospheric pressure. The sealable chamber is partially submerged in a water bath and sealed with quick-grip clamps. Nerve compound action potential is recorded in freshly isolated swine phrenic nerves. Standard electrophysiological methods were used for recording and amplification of the nerve signals. Baseline compound action potentials were recorded for their amplitude, conduction velocity, latency and duration. After baseline recordings the nerves were compressed at 100 mmHg for 10 minutes and allowed to recover for additional 10 minutes.

Figure 1 shows a schematic and photo pictures of a neural compression system:



Results

The pressure from the apparatus was measured before (input pressure) and after (chamber pressure) it entered the nerve recording chamber. Both input and chamber pressures were linearly correlated with pressure increases on the settings of the regulator and they were well correlated ($R^2 = 1$, m=1) (Figure 2, Left). The chamber internal pressure can be precisely controlled from 47 mmHg to 450 mmHg. The pressure changes achieved by the opening and closing of the 3-way valve can be completed within 0.01 seconds enabling intermittent-compression on nerve preparations. Pressure changes did not cause signal artifacts to nerve recordings. Figure 2, Right shows a typical recording of an intermittent chamber pressure change.



Figure 2. Characterization of pressure changes. Left: Correlation between chamber and input pressures. Right: Intermittent chamber pressure changes

Four nerve samples have been tested thus far. A total block of compound action potential by compression was seen in one sample. In other two samples compression reduced compound action potential conduction velocity by approximately 74% and increased compound action potential duration by approximately 110%. In the fourth sample compression did not cause significant changes. Changes in compound action potential returned to baseline levels after ten minutes recovery. Figure 3 shows the effects of pressure compression on nerve compound action potentials.



Figure 3. Effects of pressure compression on nerve compound action potentials

Interpretation of results

Our results are in accordance with compound action potential and nerve compression literatures^[2,3]. This apparatus allows the pressure inside the nerve recording chamber can be precisely controlled. The device can produce a wide range of pressure changes and is capable of delivering constant or intermittent nerve compressions. These features should allow us to simulate nerve compression injuries in a variety of pathological conditions.

Concluding message

We have described a nerve compression system that can be used to investigate real-time effects of compression forces on neural physiological responses. These studies will help us to further understand the mechanisms and prevention of nerve injuries in incontinence and other pelvic diseases.

References

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