LUMBAR SPINE STIFFNESS IS INCREASED BY ELEVATION OF INTRA-ABDOMINAL PRESSURE

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Introduction
Intra-abdominal pressure (IAP) is elevated during many functional tasks and has been argued to increase stability of the spine (Bartelink, 1957; Grillner et al., 1978; Daggfeldt and Thorstensson, 1997). Although several studies have shown a relationship between IAP and spinal stability (e.g. (Cresswell and Thorstensson, 1994; Cholewicki et al., 1999), it has been impossible to determine whether this augmentation of mechanical support for the spine is due to the increased IAP or the abdominal muscle activity associated with its production. In a recent study we have measured the magnitude of extension torque produced by an increase in IAP, which was produced in the absence of abdominal or back extensor activity (Hodges et al., 2001). This was achieved by electrically evoked contraction of the diaphragm by stimulation of the phrenic nerves in the neck. The aim of the present study was to use a similar method to determine whether elevated IAP increases spinal stiffness, in the absence of concurrent activity of the abdominal and back extensor muscles.

Methods
Three male subjects volunteered for the experiment. Recordings of IAP (gastric pressure (Pga)) and transdiaaphragmatic pressure (Pdi) were made with a pair of thin-film pressure transducers inserted into the oesophagus and stomach via the nose. EMG recordings were made from the erector spinae muscles at L2 and L4, the abdominal muscles and the diaphragm, with surface electrodes placed over the chest wall with electrodes in the 7th and 8th intercostals space in the mid clavicular line.

A sustained increase in IAP was produced by tetanic stimulation of the phrenic nerves either unilaterally or bilaterally at 20 Hz for 5s via percutaneous wire electrodes inserted at the level of the cricoid cartilage after localization of the nerve with external stimulation (Gandevia and McKenzie, 1986). Stimuli were given at a variety of intensities and depended on the tolerance of the subject and accuracy of the placement of the stimulating electrodes. Firm belts were placed around the abdomen and lower rib cage to limit expansion due to diaphragm contraction and thus, maximise the IAP increase. Subjects were positioned with supports under the pelvis and thorax to leave the abdomen unsupported during the trial. Postero-anterior spinal stiffness was measured using a servo-controlled motor that measured the force required to displace an indentor placed over the L4 or L2 spinous process with the subject lying prone. Stiffness was measured as the slope of the regression line fitted to the linear region of the force-displacement curve between 50-110 N.

Results
Tetanic stimulation of the phrenic nerve(s) produced an increase in IAP that ranged between 27-61% of the maximum IAP that could be generated voluntarily in an expulsive effort in the prone position. When IAP was elevated, the posteroanterior stiffness of the spine was increased by ~23% from the stiffness recorded at the end of a quiet expiration without phrenic stimulation (Fig. 1). Postero-anterior stiffness was increased in all subjects.

When the IAP amplitude and stiffness were compared across stimulation intensities there was a positive correlation (mean r=0.73). There was no difference in the amplitude of abdominal or erector spinae electromyographic activity between the trials with and without phrenic stimulation. The affect of IAP was
similar when force was applied to the L2 and L4 levels, although there was a trend for the stiffness increase to be greater at the L2 level.

![Figure 1 A. Representative raw data from a single subject showing force application in a control trial and during phrenic stimulation. During stimulation the gastric and transdiaphragmatic pressures are elevated. There is an additional increase in pressure with force application. B. Force-displacement curves generated from the second to fourth trial for control (filled circles) and stimulation trials (open circles). Same data as panel A. Note the increase in slope of the regression line (i.e. posteroanterior stiffness) with stimulation.]

**Discussion**

The results of this study provide evidence that postero-anterior stiffness of the lumbar spine is increased when IAP is elevated in the absence of abdominal or erector spinae muscle activity. Two findings support this conclusion, first, the slope of the regression line fitted to the force displacement data was increased for all subjects during stimulation of the phrenic nerve(s). This indicates that greater force was required to achieve the same displacement, i.e. the posteroanterior stiffness of the spine was increased. Second, there was a positive correlation between the amplitude of IAP increase and the increase in stiffness, indicating that there was a linear relationship between pressure and stiffness across the range of pressures investigated. The greater change in stiffness at the L2 and L4 levels may be due to the direct attachment of the crural fibres of the diaphragm to the L2 vertebrae.

These results provide the first in vivo evidence of the extent to which IAP can affect spinal stiffness in the absence of abdominal and erector spinae activity. In function IAP is generated by activity of all muscles surrounding the abdominal cavity, including the diaphragm, pelvic floor and abdominal muscles (particularly transversus abdominis) and other muscles of the trunk are likely to be active. Thus, the absolute stiffness of the spine will be the net result of many factors including IAP and muscle activity. While the present study cannot identify the contribution of IAP to spinal stiffness in functional situations, the data have quantified, in vivo, the extent to which IAP may contribute to the overall spinal stiffness. Regardless, the results of the present study confirm that modulation of IAP may provide a significant contribution to the control of the spine during functional tasks.
References