INTRODUCTION. Contraction of the diaphragm increases thoracic volume during spontaneous inspiration by displacing the abdominal contents caudally and by expanding the rib cage (RC). In recent years, the mechanism by which the diaphragm drives cage RC during inspiration has been clarified. Two components of diaphragm action have been identified: 1) an insessional component whereby the directional vector of diaphragm insertions and the mechanics of rib/vertebral articulations result in RC elevation and expansion and 2) an appositional component whereby increases in intra-abdominal pressure (Fap) are transmitted to the inner rib cage wall through the zone of apposition of diaphragm to rib cage* (Fig 1). These diaphragm actions each make use of Fap and thus depend on abdominal muscle contraction.

Note that these mechanisms act directly on the lower rib cage (LRC) only. Upper rib cage (URC) expansion depends on its mechanical coupling to the LRC. This requires intercostal activation. In the absence of abdominal and intercostal muscle use (e.g., in quadriplegia), the chest wall is distorted during inspiration. In a study of quadriplegic patients with complete cervical cord transection*, we found that the URC moved paradoxically (inward) in all 6 patients studied. However, the LRC simultaneously moved outward in 5 of the 6 patients.

We wished to determine whether similar RC distortion occurs as a result of abdominal and intercostal muscle blockade produced by spinal or epidural anesthesia and if the degree of distortion correlates with the level of sensory (and inferred motor) anesthesia.

METHODS. After approval from the human studies committee, we studied 11 ASA I-II adults who were undergoing elective lower extremity orthopedic surgery under spinal or epidural anesthesia. Local anesthetic agents included bupivacaine, lidocaine, and tetracaine for spinal anesthesia; and etidocaine and lidocaine for epidural anesthesia. We measured the separate cross-sectional area changes of the URC, LRC, and abdomen during spontaneous breathing in the supine position by inductance pneumography belts. Changes in lung volume (Vt) were measured by wedge spirometer. Sensory level of anesthesia was determined by pinprick. Data was collected during tidal and deep breathing both immediately before and 30 minutes following administration of the anesthetic.

RESULTS. During tidal inspiration, decreased URC expansion was measured in 7 of the 11 patients, in one patient there was no change, and in three patients there was a small increase. The fractional reduction in URC expansion as a function of Vt correlated with the level of sensory anesthesia (Fig. 2). Spinal or epidural anesthesia resulted in a decrease in the ratio of URC/LRC expansion. This also correlated with sensory level (Fig 2). Epidural and hyperbaric spinal anesthesia appeared to produce greater thoracic distortion than did similar sensory levels of isobaric spinal anesthesia. However, all 11 patients were able to overcome any distortion by recruitment of the accessory muscles of respiration during deep breathing.

DISCUSSION. Intercostal and abdominal paralysis during spinal or epidural anesthesia results in chest wall distortion characterized by decreased URC expansion. The degree of distortion appears to be dependent on the anesthetic technique and the level of anesthesia. With higher sensory level, this distortion was qualitatively similar to that previously measured in the quadriplegic. The choice of local anesthetic and method of administration may influence the degree of intercostal and abdominal muscle blockade produced by regional anesthesia. This information may help to guide selection of anesthetic technique in the patient who already suffers from respiratory compromise.

REFERENCES.