only 20-30 percent of our patients. Did you find this in all patients investigated?

Dr. Wagner: Yes, we have found this phenomenon in all of the studies that we have done which include normal
volunteers, dogs (on respirators) and sick patients who did not have adult respiratory distress syndrome. We found that the more normal the lung to begin with the less the change post-100 percent FlO₂.

Noninvasive Measurement of Intrathoracic Fluids*

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The seriousness of impending pulmonary insufficiency following stress—hemorrhage, burns, operation, trauma, left ventricular failure, sepsis, and shock—has long been recognized by clinicians. However, its detection and quantitation frequently elude currently used diagnostic techniques until irreversible pulmonary insufficiency or cardiac failure occurs. Recent applications of the electrical impedance method for acquiring physiologic data show promise of providing a safe, noninvasive, and continuous measurement for early detection, localization, and quantitation of intrathoracic fluid accumulations.², ³

In a series of studies of four groups of healthy anesthetized dogs, the basal thoracic impedance (Z₀) was evaluated as a means of detecting, localizing, and quantitating thoracic fluid accumulations. The Kubicek circumsferential band electrode lead system⁴ and two variable “spot” electrode arrangements were used with a constant sinusoidal current applied to one pair of electrodes and the voltage reflecting thoracic impedance measured across the remaining pair of electrodes (Fig 1). In groups 1 and 2, pleural and pericardial effusion were simulated by the infusion and withdrawal of normal saline (37°C) from each hemithorax or pericardial space, respectively. Pulmonary edema was induced in group 3 either by alloxan or sucrose, or simulated by pulmonary lavage with saline. A fourth group was subjected to multiple penetrating thoracic wounds inflicted by a 13-gauge needle.

These studies indicate that transthoracic electrical impedance provides a sensitive index of thoracic fluid accumulation. Although the band and both spot electrode systems were sensitive to thoracic fluid changes, only the latter systems were useful in fluid localization. This is readily apparent from Figure 2 in which the percentage of thoracic impedance change associated with the infusion of saline into the right hemithorax of a dog is shown. A linear correlation (r>0.9; P<0.01) between Z₀ and various thoracic fluid accumulations is characteristic of data for individual animals in all groups (Fig 3); the differences in conductivity of body fluids are reflected as differences in slopes (blood is less conductive than plasma or saline). Pleural and pericardial effusion typically produce an average total impedance change of 2.0 and 1.5 ohms per 100 ml of saline infused, respectively.

Alloxan- or sucrose-induced pulmonary edema produces a typical impedance change of 2 to 5 ohms for the band electrode array, with 4 ohms for the tetrapolar spot electrode array. This edema is accompanied by marked ventilatory obstruction and early deaths, attributable to the copious amounts of fluid and foam in the tracheobronchial tree. This method of producing edema does not permit simple quantification of fluid accumulations; however, by using unilateral pulmonary lavage, it is possible to maintain the animal while measuring im-

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![Diagram](https://example.com/diagram.png)

Figure 1. Four terminal systems for the measurement of thoracic impedance. A. Flexible metal electrodes encircling neck and thorax. 50 kHz constant sinusoidal current (1 mA rms) was applied to electrodes 1, 4. Voltage changes reflecting thoracic impedance changes were picked up from electrodes 2, 3. B. Stainless steel “spot” electrodes (1 cm diameter) sutured to chest wall at level of sixth intercostal space. Twenty kHz constant sinusoidal current (1 mA rms) was applied to electrodes 2, 4, and impedance changes between other pairs of electrodes were recorded. C. Standard disposable ECG type electrodes were applied to thorax as shown. Twenty kHz constant sinusoidal current (1 mA rms) was applied to electrodes 1, 6, and impedance changes between electrodes 2, 3, or 4, 5 were recorded.
pedance changes produced by infusion of 50 ml increments of 0.9 percent saline or plasma. Figure 4 shows typical results of this procedure.

The impedance method shows promise of providing a sensitive, noninvasive method of detecting thoracic fluid accumulations. The band electrode system provides detection of generalized thoracic impedance changes which may be localized by a tetrapolar spot electrode array.

REFERENCES

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DISCUSSION

Dr. Branscomb: In the measurements of impedance, geometry is a crucial factor and it must be noted that the geometry of the dog is very much different than the geometry of the thoracic cage of man. Have you studied humans in this manner? Secondly, have you studied dogs with a nonconductive fluid?

Dr. Baker: I agree with Dr. Branscomb that the thoracic geometry of the dog is different from that of man. We have, of course, not been able to study human subjects in this manner. Neither have we studied dogs using a nonconductive fluid; however, since air is a very nonconductive material, there is a marked increase in impedance when a pneumothorax is present.

Dr. Branscomb: Injection of a nonconductive fluid into the thorax would result in a change of the geometry only, and thus could tell you how much your change in impedance was due to physical movement of the lung and how much due to change in impedance produced by extra fluid itself.

Dr. Klein: Does a change in lung volume alter the impedance? If it does, it will be difficult to know what to make of a change of impedance in a situation such as the respiratory distress syndrome where both lung volume and intrathoracic water may change.

Dr. Baker: This is a problem because baseline impedance has been shown to be a function of FRC. There are several clinicians who have used this method in clinical trials. They feel that this method gives them a good early warning of impending problems. When the patient is in the same position for each measurement, the FRC doesn’t seem to be a terribly important factor.

Dr. Brigham: This doesn’t tell you if the fluid is extravascular or intravascular. What sort of changes do you get if the increase in fluid is entirely intravascular?

Dr. Baker: We have attempted to keep the blood volume constant. In one series of studies we withdrew whole blood from the dog, infused only the red cells intravenously, and then infused the plasma into the thorax. We can detect fluid distribution easily, and, presumably, there was no change in intravascular volume.

Dr. Brigham: Yes, but could this method be influenced by congestion of the vascular bed?

Dr. Baker: Yes, I am sure that we would pick up congestion of the vascular bed as a change in impedance.

Dr. Rogers: One of the big problems in patients with ARDS who are on respirators is that a simple pneumothorax will be rapidly converted into a very dangerous tension pneumothorax. Is this method specific enough and sensitive enough to be an early indication of pneumothorax in these types of patients?

Dr. Baker: I would think that this would be easily picked up by this method.

Dr. Lowry: Powers has shown that the FRC decreases progressively with increasing severity of ARDS, and thus, that the thoracic gas volume falls. Both of these are corrected with PEEP. Therefore, this decrease in thoracic gas volume in ARDS would be important in changing the impedance. I would like to direct this question to Dr. Pontoppidan and Dr. Petty. Do they think that this would be helpful in the intensive care unit? Now we so frequently have to monitor arterial blood gases and use the change in the P02 as our major criteria for changing FIO2. Would a method such as this for measuring extra- or intravascular lung water be helpful?

Dr. Pontoppidan: I see as a major drawback with this present technique the need for reproducible position in the patient. It is very bad to keep the patient in the same position during the length of time needed to make serial studies in severely ill patients and it is very difficult to return the patient to the exact position that he was in previously. In the late phases of the adult respiratory distress syndrome the FRC, VD/VT, extravascular lung water, and compliance seem to be better prognostic indicators than arterial blood gases.

Dr. Petty: I agree entirely with Dr. Pontoppidan. Bedside measurements of impedance are not very reproducible in the clinical setting. I feel that bedside physiologic measurements including functional residual capacity, arterial-alveolar O2 gradients, and compliance are more useful at the present time.