

# Changes in electrical resistivity of swine liver after occlusion and postmortem

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**Abstract**—The resistivity of swine liver tissue was measured *in vivo*, during induced ischaemia and post-mortem, so that associated changes in resistivity could be quantified. Plunge electrodes, the four-terminal method and a computer-automated measurement system were used to acquire resistivities between 10 Hz and 1 MHz. Liver resistivity was measured *in vivo* in three animals at 11 locations. At 10 Hz, resistivity was  $758 \pm 170 \Omega \cdot \text{cm}$ . At 1 MHz, the resistivity was  $250 \pm 40 \Omega \cdot \text{cm}$ . The resistivity time course was measured during the first 10 min after the liver blood supply in one animal had been occluded. Resistivity increased steadily during occlusion. The change in resistivity of an excised tissue sample was measured during the first 12 h after excision in one animal. Resistivity increased during the first 2 h by 53% at 10 Hz and by 32% at 1 MHz. After 2 h, resistivity decreased, probably owing to membrane breakdown. The resistivity data were fitted to a Cole–Cole circle, from which extracellular resistance  $R_e$ , intracellular resistance  $R_i$  and cell membrane capacitance  $C_m$  were estimated.  $R_e$  increased during the first 2 h by 95% and then decreased, suggesting an increase in extracellular volume.  $C_m$  increased during the first 4 h by 40%, possibly owing to closure of membrane channels, and then decreased, suggesting membrane breakdown.  $R_i$  stayed constant during the initial 6 h and then increased.

**Keywords**—Tissue resistivity, Liver resistivity, Radiofrequency, *In vivo* resistivity, *In vitro* resistivity, *Ex vivo* resistivity

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## 1 Introduction

DATA ON tissue resistivity of different mammals, tissue types and frequency ranges have been reported (STOY *et al.*, 1982; GABRIEL *et al.*, 1996; STUCHLY and STUCHLY, 1980; RUSH *et al.*, 1963; STEENDIJK *et al.*, 1993; DUCK, 1990; GEDDES and BAKER, 1967; FAES *et al.*, 1999). However, there are few data on the variation in tissue resistivity after death (ZHENG *et al.*, 1984; SWATLAND, 1980; SUROWIEC *et al.*, 1985) and, especially, on the comparison of *in vivo* with *ex vivo* measurements. There are some very early data (RAJEWSKY, 1938; SCHWAN, 1954) that report a negligible change in resistivity within the first 24 h after death. HEROUX and BOURDAGES (1994) reported changes in extracellular resistance  $R_e$ , intracellular resistance  $R_i$  and cell membrane capacitance  $C_m$  for rat liver, rat cortex and rat gluteus while a toxic drug was administered and up to 10 h after animal

death. KONISHI *et al.* (1995) reported changes in  $R_e$ ,  $R_i$  and  $C_m$  of freshly extracted rat liver tissue at different incubation temperatures, up to 6 h after tissue extraction.

We measured swine liver resistivity *in vivo* in three animals. We continued measurement after excision of a tissue sample in one animal for 12 h to examine the amount of resistivity change. For the first 12 h after excision, we then calculated the parameters  $R_e$ ,  $R_i$  and  $C_m$  of a three-element electric tissue model to examine causes of resistivity change. We measured resistivity during an occlusion experiment in one animal, where hepatic blood supply was stopped to determine changes in resistivity due to ischaemic effects.

## 2 Methods

We used three domestic pigs (30–40 kg) for the experiments. We obtained pre-approval for all animal experiments from the Institutional Animal Care and Use Committee, University of Wisconsin, Madison. All procedures were performed with the animals under general anaesthesia.

Induction of anaesthesia was achieved by using an intramuscular injection of tiletamine hydrochloride, zolazepam

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hydrochloride and xylazine hydrochloride. The animals were then intubated and maintained on inhaled halothane. Once adequate anaesthesia had been achieved, the abdomen was opened. The body temperature of the animals was 39 °C.

We used the four-terminal measurement method, which minimises the effects of polarisation impedance (STEENDIJK *et al.*, 1993) and which has been widely used for resistivity measurements in mammalian tissue (RUSH, 1963; STEENDIJK *et al.*, 1993; BRAGOS *et al.*, 1996). We used four-electrode, silver 0.38 mm diameter plunge electrodes, with electrode spacing of 1.5 mm and depth of 4 mm. We used the same measurement circuit that we have used in previous studies on heart resistivity measurements (TSAI *et al.*, 2000). We calibrated the circuit and electrodes using 0.9% saline solution.

The outputs of the differential amplifier and of the current-to-voltage converter were connected to an HP54600B digital oscilloscope. The input signal was generated using an HP33120A function generator. Both the oscilloscope and the function generator featured an RS232 serial port. We used an IBM PC-compatible computer for controlling the signal generator and automatically applying sinusoidal signals of frequencies from 100 Hz to 1 MHz using the RS232 interface. The oscilloscope reported the RMS values of the differential amplifier output and the current-to-voltage converter output to the computer.

The program running on the PC for controlling the devices was written in Microsoft Visual Basic and entered the measured data together with the calculated resistivity values into a Microsoft Excel sheet for further processing. One measurement cycle incorporating the gathering of data at seven different frequencies took 25 s.

All resistivity measurements described below were performed at 10 Hz, 100 Hz, 1 kHz, 10 kHz, 100 kHz, 500 kHz and 1 MHz. We measured three animals, at 11 locations in all liver lobes, at about 1 h after start of the anaesthetic preparation.

In one of the animals, we performed vascular inflow occlusion of the hepatic artery and the portal vein, while continuing resistivity measurements at a single location in the right upper lobe for 10 min. In another animal, we resected an 8 × 9 × 2 cm piece of the left upper lobe before sacrificing the animal.

We performed resistivity measurements on the extracted liver tissue at 0.5, 2, 4, 8 and 12 h after excision. At each time point, we acquired four data sets that were then averaged. The temperature of the tissue sample was recorded at the time points when data were acquired and during the *in vivo* measurements. Between the measurements at different time points, the probe was removed from the tissue and cleaned. Both the occlusion experiment and the post-mortem experiment were performed after the initial *in vivo* measurements had been completed. All measurement locations were selected so that the tissue was thicker than 2 cm, to reduce any errors from tissue samples that were too thin.

Biological tissue can be described by a three-element circuit model (see Fig. 1) consisting of extracellular resistance  $R_e$ , intracellular resistance  $R_i$  and cell membrane capacitance  $C_m$ . We determined a least-square circle approximation (i.e. approx-

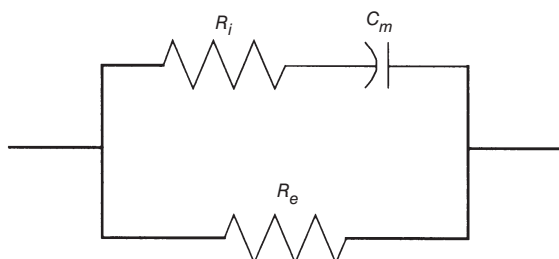


Fig. 1 Tissue equivalent circuit model

imation to the Cole–Cole half-circle) of the resistivity data we acquired from the measurements on extracted liver tissue. From the circular approximation, we determined  $R_e$ ,  $R_i$  and  $C_m$  as previously done by KONISHI *et al.* (1995). We determined  $R_0$  (DC) and  $R_\infty$  ( $f \rightarrow \infty$ ), where the half-circle crossed the real axis, and  $\omega_{max}$ , where the half-circle had its maximum imaginary value (see Fig. 2). We then estimated  $R_e$ ,  $R_i$  and  $C_m$  according to

$$R_e = R_0$$

$$R_i = \frac{R_0 \cdot R_\infty}{R_0 - R_\infty}$$

$$C_m = \frac{1}{\omega_{max} \cdot (R_e + R_i)}$$

We determined these parameters for *in vivo* resistance (baseline value) and for all time points where we measured *ex vivo* resistivity on the extracted liver sample.

### 3 Results

First, we measured resistivity at 11 locations *in vivo* in three animals. Then, we performed the occlusion experiment on one animal and measured the resistivity time course of an extracted tissue sample of the second animal. Fig. 3 shows the mean and standard deviation of 11 *in vivo* measurements in three animals. Fig. 4 shows the time course of resistivity during the occlusion experiment. Each data point is taken from only a single measurement, owing to time constraints.

Fig. 5 shows the time course of resistivity of the extracted tissue sample for the first 12 h after extraction. Each data point represents the average of four measurements. The initial value at  $t = 0$  is from an *in vivo* measurement at the location of tissue extraction. Fig. 6 shows the time course of temperature of the extracted tissue sample. Fig. 7 shows the time course of fitted parameters  $R_e$ ,  $R_i$  and  $C_m$  of the extracted tissue sample in relation to baseline values (*in vivo*, i.e.  $t = 0$ ).

### 4 Discussion

Fig. 3 shows the mean and standard deviation of 11 *in vivo* liver resistivity measurements, performed on three different animals. The standard deviation is higher at low frequencies. The liver is a very heterogeneous organ, particularly with regard to the distribution of blood vessels. Both number and size vary greatly depending upon location, which results in differences in local blood circulation.

Blood has a resistivity of 100–200  $\Omega \cdot \text{cm}$  (GABRIEL *et al.*, 1996; DUCK, 1990; GEDDES and BAKER, 1967) within the frequency range of 100 Hz–1 MHz. At frequencies below

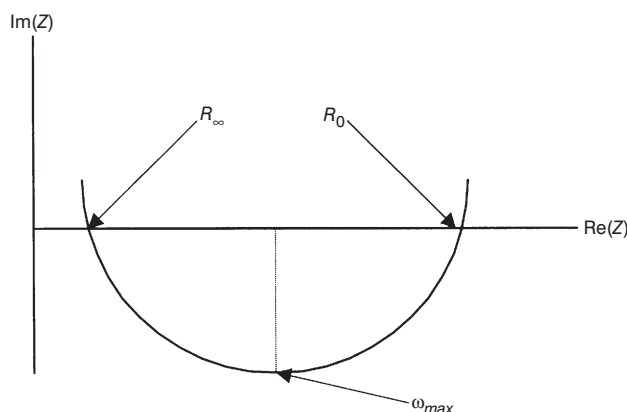
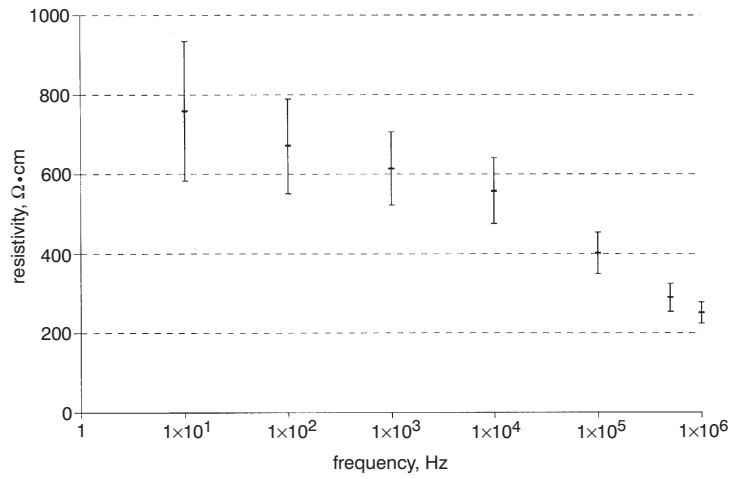
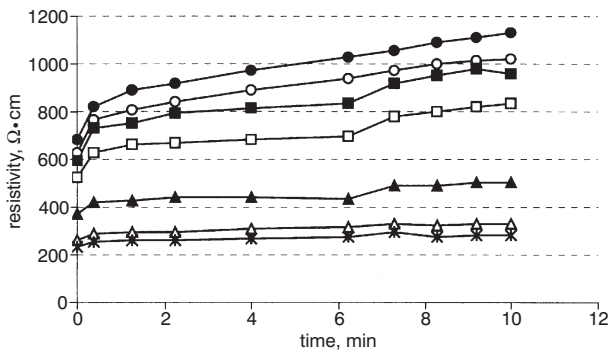


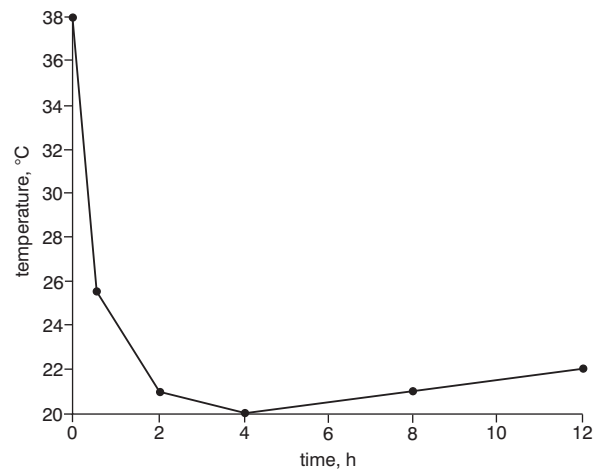
Fig. 2 Cole–Cole circle



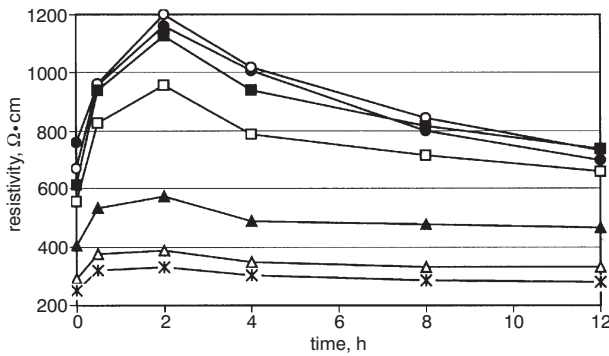
**Fig. 3** Resistivity decreases with frequency. Mean and standard deviation of 11 measurements in three animals



**Fig. 4** Time course of resistivity during first 10 min after blood vessels supplying liver have been obstructed. (●) 10 Hz; (○) 100 Hz; (■) 1 kHz; (□) 10 kHz; (▲) 100 kHz; (△) 500 kHz; (\*-) 1 MHz



**Fig. 6** Temperature of excised tissue sample during first 12 h after excision

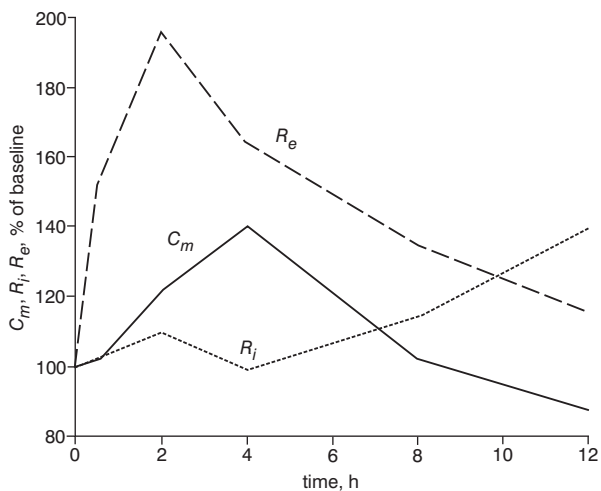


**Fig. 5** Time development of resistivity during first 12 h after excision of liver. Leftmost value is resistivity measured *in vivo*. (●) 10 Hz; (○) 100 Hz; (■) 1 kHz; (□) 10 kHz; (▲) 100 kHz; (△) 500 kHz; (\*-) 1 MHz

10 kHz, the average measured values for liver resistivity are 700–800  $\Omega \cdot \text{cm}$ . The difference, compared with the resistivity of blood, is much higher than at high frequencies. Therefore the amount of blood flow at the measurement site has a higher impact at low frequencies than at high frequencies and results in greater variation in the acquired values.

Fig. 5 shows that resistivity increases for all frequencies from 0–2 h after removal of the liver. The difference is lowest at radio frequencies (e.g. +33% at 500 kHz) and increases for low frequencies (+184% at 1 kHz).

One contribution to the rise in resistivity after excision is due to the temperature dependence of liver tissue. The conductivity temperature coefficient is highest at low frequencies, where it ranges between 1%  $^{\circ}\text{C}^{-1}$  and 2%  $^{\circ}\text{C}^{-1}$  (GABRIEL *et al.*, 1996),



**Fig. 7** Changes in (---) extracellular resistance  $R_e$ , (----) intracellular resistance  $R_i$  and (—) membrane capacitance  $C_m$  during first 12 h of excised tissue sample

and lies at about 1%  $^{\circ}\text{C}^{-1}$  below temperatures of 50  $^{\circ}\text{C}$  at radio frequencies (DADD *et al.*, 1996). Assuming a resistivity temperature coefficient of 1.5%  $^{\circ}\text{C}^{-1}$  for frequencies above 1 kHz, we can estimate the change in resistivity caused by temperature, according to Fig. 6, to be around 18% at the 0.5 h time point, and around 26% at the 2 h and later time points, compared with *in vivo* values. The change in resistivity seen at the 0.5 h time

point (e.g. 85% at 1 kHz, and 30% at 500 kHz) is therefore partially a result of the lower temperature. However, the rise in resistivity within the first 2 h is much higher than can be explained by a change in temperature alone. Later than 2 h after excision, the temperature stays between 20 °C and 22 °C, and so all changes seen after that time stem from different effects.

A large contribution to the initial resistivity increase most probably stems from ischaemic effects, which was demonstrated by the occlusion experiment. The liver is more susceptible to ischaemia than any other organ except the brain. Functional abnormality occurs 15 min after complete stoppage of blood flow into the liver (BASSI and BERNELLI-ZAZZERA, 1964; DELMAS-BEAUVIEUX *et al.*, 1992).

Fig. 4 shows that resistivity rises significantly within the first minutes after blood supply has been stopped (e.g. +62% at 1 kHz and +24% at 500 kHz). BRAGOS *et al.* (1996) obtained similar results during resistivity measurement on the heart while a coronary artery was obstructed.

It is well known that metabolism and ATP concentration decline rapidly when the blood supply is cut off. This results in reduced activity of the ion pumps, which leads to changes in ion distribution between inter- and extra-cellular spaces. More specifically, cessation of sodium-potassium pump activity ( $\text{Na}^+\text{-K}^+\text{-ATPase}$ ) leads to depolarisation of the cell. Eventually, the result is inflow of water and sodium, which causes cell swelling (LAMBOTTE, 1986). Furthermore, influx of  $\text{Ca}^{2+}$  leads to swelling and rupture of mitochondria and to damage of the other organelles. Lysosomal enzymes are released within the cell, finally resulting in cell death (FARBER *et al.*, 1981). Another contribution to the rise in resistivity may come from drainage of blood, as blood has a much lower resistivity than liver tissue has, as discussed earlier.

We see the same initial rise in resistivity in the measurements of extracted liver tissue. Fig. 5 shows that this initial rise persisted until 2 h post-mortem. Considering the electric tissue model from Fig. 1, this rise is mainly due to an increase in extracellular resistance, as seen in Fig. 7. The rise in extracellular resistance is the result of a decline in extracellular fluid volume.

The membrane capacity  $C_m$  also rises initially and reaches its peak 4 h post-mortem. This membrane capacitance rises due to the increase in membrane surface resulting from cell swelling. Other contributions could come from the closure of membrane ion channels due to ATP depletion and depolarisation, and from closure of gap junctions. Gap junctions represent a path between neighbouring cells that allows the passage of molecules smaller than 1000 Da, particularly ions (KUMAR and GILULA, 1996). In normal liver parenchyma, each cell has an average of six neighbouring cells. Gap junctions provide intercellular connections between all neighbouring cells (MEYER *et al.*, 1981). SCHELLENS *et al.* (1987) found initial changes in the structural arrangement of connexin 32, the protein that forms gap junctions in the liver, 30 min after induced ischaemia *in vivo*. MEYER *et al.* (1981) observed that the intercellular resistivity  $\rho_i$  increases from  $\sim 3000 \Omega \cdot \text{cm}$  in normal liver to  $\sim 30\,000 \Omega \cdot \text{cm}$  in regenerating liver, where only a few gap junctions are present. Closure of gap junctions could contribute to the steady rise in resistivity within the first 2 h.

After 2 h post-mortem, resistivity decreases. Several factors may contribute to this decrease. HEFFRON and HEGARTY (1974) found an increase in extracellular fluid, due to loss of membrane integrity post-mortem, that allows continuity between intracellular and extracellular fluid compartments. As discussed earlier, lysosomal agents are released within the cell, contributing to cell membrane damage.

Fig. 7 shows that the resistivity decrease is accompanied by a decrease in both extracellular resistance  $R_e$  and membrane

capacitance  $C_m$ . The decrease in  $R_e$  is in agreement with the increase in extracellular fluid volume. After tissue suffers extensive post-mortem damage, cell membranes begin to break down (SWATLAND, 1980). The loss of membrane integrity results in a steady decrease in membrane capacitance.

ASTBURY *et al.* (1988) observed a decrease in the low-frequency resistivity of canine spleen within 10 h after the death of the animal. These changes were accompanied by membrane breakdown, which they confirmed histologically. Membrane breakdown preferentially decreases low-frequency resistivity, where the magnitude of resistivity is mainly governed by the existence of cell membranes that force the current (i.e. ions) to flow in extracellular fluid.

For the parameters  $R_e$ ,  $R_i$  and  $C_m$ , we acquired results similar to those of KONISHI *et al.* (1995). During their *ex vivo* measurements at 20 °C, they observed initial rises in  $R_e$  and  $C_m$ , with peaks 1.5 h post-mortem, followed by steady decreases.

Our results show that there is a significant change in tissue resistivity after removal of the organ and that accurate results require *in vivo* measurements with intact circulation.

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