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●*Original Contribution*

HUMAN *IN SITU* DOSIMETRY: DIFFERENTIAL INSERTION LOSS DURING PASSAGE THROUGH ABDOMINAL WALL AND MYOMETRIUM

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Abstract—We constructed a specialized *in vivo* exposimetry system and determined selected ultrasonic field quantities. We examined two groups of non-pregnant women (nulliparas = 14, multiparas = 9) under conditions of full and empty bladder. A calibrated 7-element linear array hydrophone was placed in the anterior fornix of the vagina in each subject. In the full bladder condition, the sound beam traversed the anterior abdominal wall and full bladder, whereas after voiding, the sound beam traversed the abdominal wall and anteverted uterine fundus. Each study was conducted using a 3.5 MHz mechanical sector transducer. Calibration data were recorded after completion of each *in vivo* experiment. Data from both groups were pooled for analysis. Assuming (1) the sound path through the full bladder is loss less, the insertion loss (IL_{FULL}) should represent the insertion loss for the abdominal wall ($IL_{ABD\ WALL}$) 8.2 ± 5.6 dB; whereas (2) for the empty bladder condition, (IL_{EMPTY}) represents ($IL_{ABD\ WALL} + IL_{UTERUS}$). Subtracting IL_{FULL} from IL_{EMPTY} yields IL_{UTERUS} = 5.8 ± 6.8 dB. Therefore, knowing the respective path lengths and normalizing for frequency, the mean tissue attenuation coefficients (A) are estimated to be $A_{ABD\ WALL}$ = 1.39 dB/cm-MHz and A_{UTERUS} = 0.14 dB/cm-MHz. These attenuation data suggest that the abdominal wall is the principal source of ultrasonic energy loss.

Key Words: **Ultrasound, Exposimetry, *In situ*.**

INTRODUCTION

Although the clinical use of ultrasound in the reproductive sciences continues to increase exponentially, scientists have still not resolved the issues of attendant bioeffects and potential biohazards, especially since scientific data establishing the safety of ultrasound are limited. Data from animal studies would unequivocally suggest that high intensity ultrasound has definite and biologically harmful, even lethal, effects (Dunn and Fry 1971; Frizzell et al. 1977; Fry et al. 1970; O'Brien 1984, 1991). Whether clinical instruments with lower power settings pose similar but less obvious biohazards remains unknown. The issue is further clouded by virtue of minimal *in vivo* exposimetry data defining actual energy levels to which gonadal or embryonic tissues are exposed during the course of a routine "clinical" ultrasound examination. The scientific basis for estimating *in vivo* exposures is becoming even more important given the recently approved Standard for Real-Time Display of Thermal and Mechanical Indices on Diagnostic Ultrasound Equipment (AIUM 1992) which, in general, will allow for higher fetal exposure levels than in the past.

Over the previous four years, we have constructed an *in vivo* exposimetry system, developed and tested customized software and determined selected "first-order ultrasonic quantities," *i.e.*, pressure waveform, peak compressional pressure (p_c), peak rarefactional pressure (p_r), as well as selected "second-order ultrasonic quantities," *i.e.*, spatial peak, temporal average intensity (SPTA), spatial peak, pulse average intensity (SPPA), spatial peak, temporal peak intensity (SPTP) during a routine ovarian ultrasound examination (Daft et al. 1990; Siddiqi et al. 1991). Although our published *in vivo* exposimetry data define the average coefficient of tissue attenu-

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ation or insertion loss which occurs in a routine ovarian ultrasound examination, the contribution by each of the interposing maternal tissue layers, *i.e.*, skin, subcutaneous fat, fascia, skeletal muscle and myometrium remains unknown.

The current study was therefore designed to determine the specific contribution of the maternal abdominal wall and the myometrium, to the average insertion loss during a routine non-pregnant reproductive ultrasound examination. We tested the hypothesis that the maternal abdominal wall is the principal source of ultrasonic energy loss whereas the myometrium is a low loss tissue.

METHODS

Diagnostic imaging system

A 3.0 MHz frequency, mechanical sector transducer (focal zone 5.5–13 cm, focal point 8 cm, crystal diameter 19 mm) in combination with an ATL Ultramark 4 Model (Advanced Technology Systems, Bothell, WA, USA) diagnostic ultrasound imaging system was used for all studies. Exposure time after obtaining an acceptable real-time image was 5 min. Power setting for the instrument was 100% at all times.

Exposimetry instrumentation

The customized exposimetry equipment and software for *in vivo* and calibration studies have previously been reported in detail (Daft et al. 1990). In summary, instrumentation has been developed to measure the acoustic pressure field during a diagnostic reproductive system ultrasound examination. The ultrasonic field was sampled using a calibrated 7-element linear array hydrophone of polyvinylidene difluoride transducers. This hydrophone was placed in the anterior vaginal fornix superior to the cervix. The radio frequency (RF) signals from the hydrophone were digitized at 50 Ms/s, and the maximum amplitude in waveform received in the examination was recorded along with that RF waveform. The reference output of the clinical real-time scanner was obtained by placing the hydrophone in a 37°C water bath at the same distance from the clinical transducer as that used to obtain the *in vivo* recording. From the hydrophone recordings, 10 exposimetry quantities were determined, five under *in vivo* conditions and five under *in vitro* conditions. The five quantities were the maximum peak compressional pressure p_c, the maximum peak rarefactional pressure p_r, the spatial peak, temporal average intensity I_{SPTA}, the spatial peak, pulse average intensity I_{SPPA}, and the spatial peak, temporal peak intensity I_{SPTP} (Daft et al. 1990; Siddiqi et al. 1991).

Subject population

Healthy, non-pregnant female volunteers were recruited for the study. Each subject was counselled and asked to sign an informed consent statement as approved by the University of Cincinnati Medical Center Institutional Review Board. Each subject was studied under two conditions: first, in the presence of a full urinary bladder where the ultrasound beam traversed the anterior abdominal wall and distended bladder prior to visualization of the hydrophone in the anterior vaginal fornix (Fig. 1a); and second, immediately after emptying the urinary bladder where the ultrasound beam traversed the anterior abdominal wall and anteflexed uterus (myometrium) prior to visualization of the hydrophone in the anterior vaginal fornix (Fig. 1b).

Hydrophone placement and study protocol

Each subject was asked to force fluids for 1 h prior to the planned time for the study. When each subject felt her urinary bladder was distended and there was an urge to void, she was placed in a supine position with hips abducted, knees flexed and externally rotated, *i.e.*, in a "frog-leg" configuration. The custom designed hydrophone was then introduced into the vagina and placed in the anterior vaginal fornix superior to the cervix. Placement was checked using real-time imaging and care was taken to ensure that the urinary bladder was in fact full with complete displacement of the pelvic organs by the distended bladder. The lower end of the hydrophone has a recognizable round flange (Figs. 1a and 1b) to help ensure appropriate placement and orientation of the linear array of transducers. The total distance from the transducer to the hydrophone, the thickness of the anterior abdominal wall and the bladder width (excluding the anterior and posterior bladder walls) were then measured on-line using real-time imaging.

In vivo data were then obtained at 100% power settings for the diagnostic imaging system. The transducer was moved across the abdominal wall surface with real-time imaging ensuring constant hydrophone visualization. The largest hydrophone signal was saved for analysis. Reference data were recorded immediately after completion of the *in vivo* study from a tank filled with water at body temperature (37°C) and with the transducer and hydrophone fixed at the same total distance as the *in vivo* state. Again, the largest signal recorded during the *in vitro* procedure was saved for data analysis.

Each subject was then immediately asked to empty her urinary bladder and was studied in the empty bladder state. The same custom-designed hydrophone used previously was reinserted into the va-

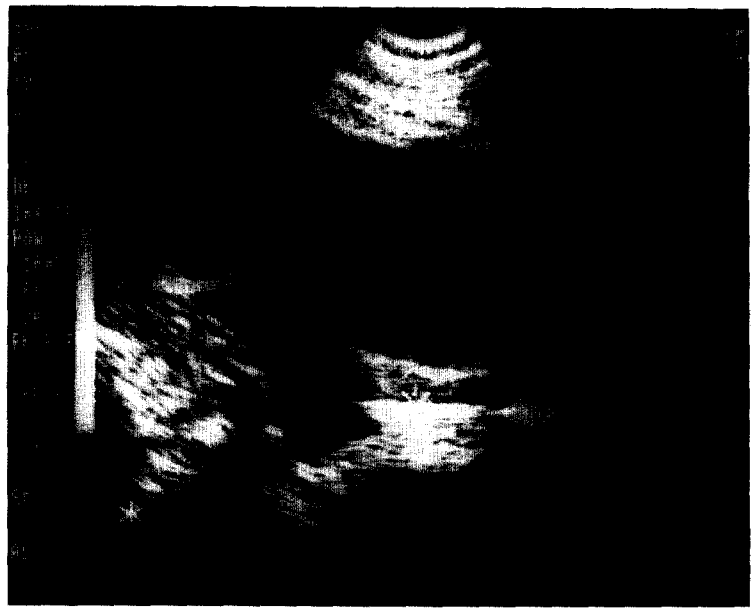

Fig. 1(a). Representative sonogram of full bladder (b) condition with hydrophone *in situ* (straight arrow) and uterus (curved arrow).

gina and placed in the anterior vaginal fornix with slight forward pressure so as to further flex the anteverted uterus thereby ensuring closest proximity between the hydrophone and uterus. (Subjects with retroverted uteruses were excluded from the study.) The total distance from the transducer to the hydrophone, the thickness of the anterior abdominal wall, and the uterine thickness (myometrium) were measured separately on-line in real-time. *In vivo* and *in vitro* data were then obtained in the same manner as described above.

Although an attempt was made to ensure that the hydrophone was placed in the same mid-line axial plane in both the full and empty bladder conditions (using the umbilicus as a reference) the study design precluded absolute accuracy (the hydrophone had to

Fig. 1(b). Representative sonogram of empty bladder (b) condition with hydrophone *in situ* (straight arrow) and overlying anteflexed uterus (curved arrow).

be removed to allow the subjects to void). Similarly, estimates of repeatability were not possible because of the study design using human volunteers.

Data analysis

For each subject, two complete data sets of *in vivo* and *in vitro* pressure waveforms were obtained (full and empty bladder states, respectively) with corresponding sonograms for tissue-path distance measurements. Six insertion loss values (loss as determined by the measurement procedure) were calculated for each complete data set, *i.e.*,

$$IL = 20 \times \log_{10}(\textit{in vitro}\ \text{pressure}/\textit{in vivo}\ \text{pressure}) \quad (1)$$

where the pressure ratios were for p_c, p_r and $p_c + p_r$, and

$$IL = 10 \times \log_{10}(\textit{in vitro}\ \text{intensity}/\textit{in vivo}\ \text{intensity}) \quad (2)$$

where the intensity ratios were for I_{SPTA}, I_{SPPA} and I_{SPTP}. An average insertion loss value of the six insertion loss values represented the insertion loss value ($\langle IL \rangle$) for each of the two data sets (full and empty bladder) for each subject for subsequent calculations.

Calculations were based on the overlying tissue model (Siddiqi et al. 1991) which assumes that attenuation consists only of that due to intact tissue and the fluid path (bladder urine) contributes no attenuation and not the fixed attenuation model (Carson et al. 1989a). The overlying tissue model attenuation coefficient, A_0, for each subject was calculated by dividing the average insertion loss ($\langle IL \rangle$) by the center frequency, f_c, and the distance of the nonfluid path, d_{nf}, *i.e.*,

$$A_0 = \langle IL \rangle / (f_c d_{nf}) \quad (\text{in dB/cm-MHz}). \quad (3)$$

The center frequency was 2.4 MHz as determined from a spectrum analysis of the hydrophone signal (Siddiqi et al. 1991). The overall attenuation coefficient was calculated as the mean value of the 23 samples for each data set. This latter calculation does not take into consideration the average insertion loss as a function of distance which is discussed below.

Statistical methods

The dependency of the experimentally determined $\langle IL \rangle$ data on the volunteers' anatomy (thickness of abdominal wall and myometrium) was analyzed statistically using simple regression analysis. The dependent variable (Y) was the appropriate average insertion loss in units of dB and the independent variable (X) was the appropriate anatomical distance in units of centimeters. All of the dependencies were evaluated against the three simple regression models:

$$Y = a + bX \quad \text{(linear model)} \quad (4a)$$

$$Y = aX^b \quad \text{(multiplicative model)} \quad (4b)$$

$$Y = \exp(a + bX) \quad \text{(exponential model)} \quad (4c)$$

where a represents the estimate of the intercept and b represents the estimate of the slope in each model. Probability levels (p) for the estimates of the intercept and slope were determined along with their standard error (SE) for each regression model. Probability levels (p), correlation coefficients (r) and standard error of the estimates (SE) were determined using analysis of variance.

The full and empty bladder conditions individually ($n = 23$ for each data set) were evaluated using the three regression models listed above. Predicted mean and 95% confidence interval data are presented for the appropriate best fit models.

RESULTS

Study population

A total of 23 individuals were studied, 14 were nulliparous and 9 were multiparous. Each individual was studied under two conditions, first with a full bladder and then immediately after voiding. A two-tailed t-test found no difference between the nulliparous and multiparous subjects with respect to (1) (a) total distance, (b) abdominal wall thickness, (c) bladder width, and (d) average insertion loss, in the "full bladder" condition; and (2) (a) total distance, (b) abdominal wall thickness, (c) uterine thickness, and (d) average insertion loss, in the "empty bladder" condition (Tables 1 and 2). Therefore, data from the nulliparous and multiparous subjects were pooled for analysis into two experimental conditions: full bladder state ($n = 23$) and empty bladder state ($n = 23$).

Overall insertion loss evaluation

The mean insertion loss values ($\langle IL \rangle$) for the full and empty bladder conditions were 8.2 ± 5.6 dB and 13.9 ± 5.8 dB, respectively. As expected, the empty bladder condition resulted in a greater insertion loss value (because of a greater thickness of soft tissue traversed) than the full bladder condition even though the total distance values were not too different under the two conditions (Tables 1 and 2).

Table 1. Total distance from transducer to hydrophone, including abdominal wall thickness, bladder width and mean insertion loss ($\langle IL \rangle$) in the full bladder condition (mean ± standard deviation).

	n	Total distance (cm)	Abdominal wall thickness (cm)	Bladder width (cm)	$\langle IL \rangle$ (dB)
Nulliparous	14	8.20 ± 1.79	2.53 ± 0.79	5.67 ± 1.26	7.64 ± 4.49
Multiparous	9	7.93 ± 0.97	2.39 ± 0.66	5.54 ± 0.76	8.99 ± 7.27
Pooled	23	8.10 ± 1.50	2.47 ± 0.73	5.67 ± 1.07	8.17 ± 5.62

Differential insertion loss

In the full bladder condition, assuming that the sound path through urine is loss-less, the average insertion loss, (IL_{FULL}), should represent the anterior abdominal wall insertion loss ($IL_{\text{ABD WALL}}$), *i.e.*,

$$IL_{\text{FULL}} = IL_{\text{ABD WALL}} - (IL_{\text{URINE}} = 0). \quad (5)$$

The mean $IL_{\text{ABD WALL}}$ ($n = 23$) was therefore 8.2 ± 5.6 dB (range: 1.7–22.7 dB), and the mean abdominal wall thickness, ($d_{\text{ABD WALL}}$) was 2.5 ± 0.7 cm (range: 1.6–4.5 cm).

Evaluating each of the 23 $IL_{\text{ABD WALL}}$ values at their respective $d_{\text{ABD WALL}}$ values and at a center frequency (f_o) of 2.4 MHz, the mean value for the attenuation coefficient for the abdominal wall ($A_{\text{ABD WALL}}$) was obtained by:

$$A_{\text{ABD WALL}} = \frac{1}{23} \sum_{n=1}^{23} \left(\frac{IL_{\text{ABD WALL}}}{d_{\text{ABD WALL}} f_o} \right) \quad (6)$$

which yields a value $A_{\text{ABD WALL}} = 1.39 \pm 0.89$ dB/cm-MHz (range 0.40–4.01 dB/cm-MHz).

Figure 2 shows the relation between abdominal wall insertion loss ($IL_{\text{ABD WALL}}$) and abdominal wall distance ($d_{\text{ABD WALL}}$) where the multiplicative regression analysis provided the best fit ($p < 0.056$), *i.e.*,

$$IL_{\text{ABD WALL}} = 2.8(d_{\text{ABD WALL}})^{0.99} \quad (7)$$

where the $IL_{\text{ABD WALL}}$ (which is assumed to equal IL_{FULL}) and $d_{\text{ABD WALL}}$ (measured directly) demonstrate the slope to be 2.8 dB/cm at 2.4 MHz. This is essentially a linear model over the applicable $d_{\text{ABD WALL}}$ range since the exponent is essentially unity. Normalizing the data to frequency, yields a value 1.17 dB/cm-MHz for the abdominal wall.

It is interesting to compare the results from the multiplicative model to the linear regression model ($p < 0.11$), *i.e.*,

$$IL_{\text{ABD WALL}} = 2.67 d_{\text{ABD WALL}} + 1.57 \quad (8)$$

since over the applicable $d_{\text{ABD WALL}}$ range, the slope of 2.67 dB/cm is essentially the same as that for the multiplicative model.

For the empty bladder condition, IL_{EMPTY}, represents the sum of insertion losses through the abdominal wall ($IL_{\text{ABD WALL}}$) and uterus (myometrium) (IL_{UTERUS}), *i.e.*,

$$IL_{\text{EMPTY}} = IL_{\text{ABD WALL}} + IL_{\text{UTERUS}}. \quad (9)$$

From eqn (5) we can derive the average insertion loss for the uterus (IL_{UTERUS}), *i.e.*,

$$IL_{\text{UTERUS}} = IL_{\text{EMPTY}} - IL_{\text{FULL}}. \quad (10)$$

The mean IL_{UTERUS} was 5.8 ± 6.8 dB (range: −4.8–20.2 dB) and the mean uterine thickness (d_{UTERUS}) was 5.2 ± 1.3 cm (range: 2.9–7.3 cm).

Evaluating each of the 23 IL_{UTERUS} values with their respective uterine thickness values (d_{UTERUS}) and at a center frequency (f_o) of 2.4 MHz, the mean value for the attenuation coefficient for the uterus (A_{UTERUS}) was obtained by:

$$A_{\text{UTERUS}} = \frac{1}{23} \sum_{n=1}^{23} \left(\frac{IL_{\text{UTERUS}}}{d_{\text{UTERUS}} f_o} \right) \quad (11)$$

Table 2. Total distance from transducer to hydrophone, including abdominal wall thickness, uterine (myometrial) thickness and mean insertion loss ($\langle IL \rangle$) in the empty bladder condition (mean ± standard deviation).

	n	Total distance (cm)	Abdominal wall thickness (cm)	Uterine thickness (cm)	$\langle IL \rangle$ (dB)
Nulliparous	14	7.65 ± 2.09	2.53 ± 0.79	5.12 ± 1.44	14.35 ± 1.19
Multiparous	9	7.80 ± 1.28	2.39 ± 0.66	5.41 ± 1.00	13.31 ± 5.38
Pooled	23	7.71 ± 1.78	2.47 ± 0.73	5.23 ± 1.27	13.94 ± 5.78

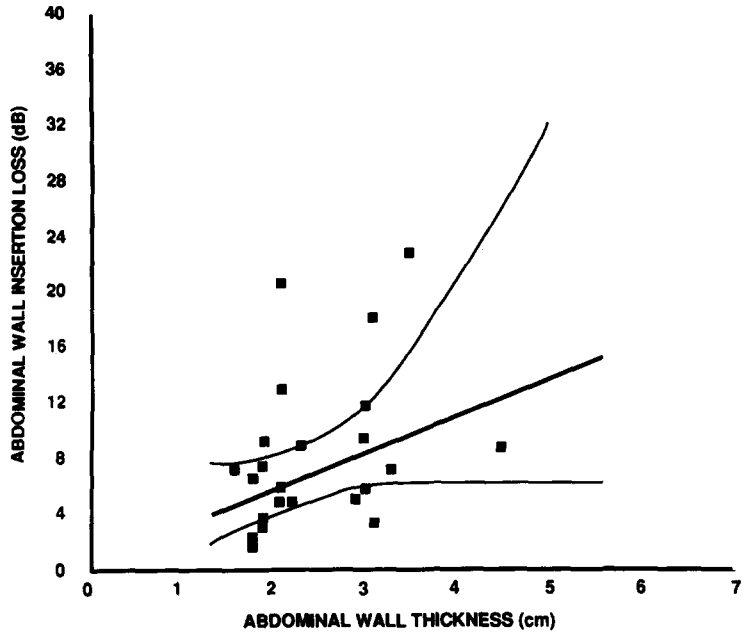

Fig. 2. Predicted mean and 95% confidence limits defining the relationship between insertion loss in dB and abdominal wall thickness in centimeters using the multiplicative regression model ($p < 0.05$).

which yields a value $A_{\text{UTERUS}} = 0.14 \pm 0.17$ dB/cm-MHz (range −0.14–0.55 dB/cm-MHz).

The negative values of IL_{UTERUS} may be due to the assumption that bladder urine is loss-less and not taking into account reflective losses at membrane surfaces. Assuming that the attenuation coefficient of bladder urine is between that of water (0.008 dB/cm at 2.4 MHz and 37°C [Herzfeld and Litovitz 1959]), and amniotic fluid (0.02 dB/cm at 1.7 MHz and 25°C; 0.03 dB/cm at 2.8 MHz and 25°C [Zana and Lang 1974]), a value of 0.01 dB/cm at 2.4 MHz was chosen. Therefore, in order to account for bladder urine losses (IL_{URINE}), *i.e.*,

$$IL_{\text{ABD WALL}} = IL_{\text{FULL}} - IL_{\text{URINE}} \qquad (12)$$

where IL_{URINE} is the product of 0.01 dB/cm and $d_{\text{BLADDER WIDTH}}$ in cm. Therefore:

$$IL_{\text{UTERUS}} = IL_{\text{EMPTY}} - (IL_{\text{FULL}} + IL_{\text{URINE}}). \qquad (13)$$

Under conditions in which ultrasonic loss in bladder urine is considered, $A_{\text{ABD WALL}}$ is 1.37 ± 0.89 dB/cm-MHz and A_{UTERUS} is 0.14 ± 0.17 dB/cm-MHz. There is very little change in the values. Evaluating the ultrasonic transmission loss due to specular reflection at the bladder/tissue interface, assuming the bladder impedance (product of ρ and c) is 1.512 Mrayls ($\rho = 993$ kg/m^3 and $c = 1523$ m/s) and the tissue impedance is 1.872 Mrayls ($\rho = 1200$ kg/m^3 and $c = 1560$ m/s) yields a transmission loss at normal incidence of 0.05 dB.

Other possible sources of error responsible for the measurement uncertainty, which in turn would be responsible for the calculated negative insertion loss values are: (1) slightly different parts of the abdominal wall being traversed in the full and empty bladder conditions, and (2) changes in the beam pattern caused by traversing heterogenous tissue under the two bladder conditions.

Therefore, ultrasonic loss in bladder urine and specular reflection at the bladder/tissue interface cannot account for the calculated negative insertion losses of the uterus (myometrium). The IL_{UTERUS} is determined from the difference between IL_{EMPTY} and IL_{FULL} and, given an estimated error of approximately 20% in the measurement of each, may be responsible for some of the IL_{UTERUS} values being negative which is, of course, not possible.

There is no statistically significant regression model which describes the relation between uterine insertion loss (IL_{UTERUS}) and uterine thickness (d_{UTERUS}). Figure 3 demonstrates the relation between IL_{UTERUS} and d_{UTERUS} where the linear regression analysis provided the best fit ($p < 0.28$), *i.e.*,

$$IL_{\text{UTERUS}} = 1.25 d_{\text{UTERUS}} - 0.80. \qquad (14)$$

This, when normalized to frequency, yields an attenuation coefficient of 0.52 dB/cm-MHz.

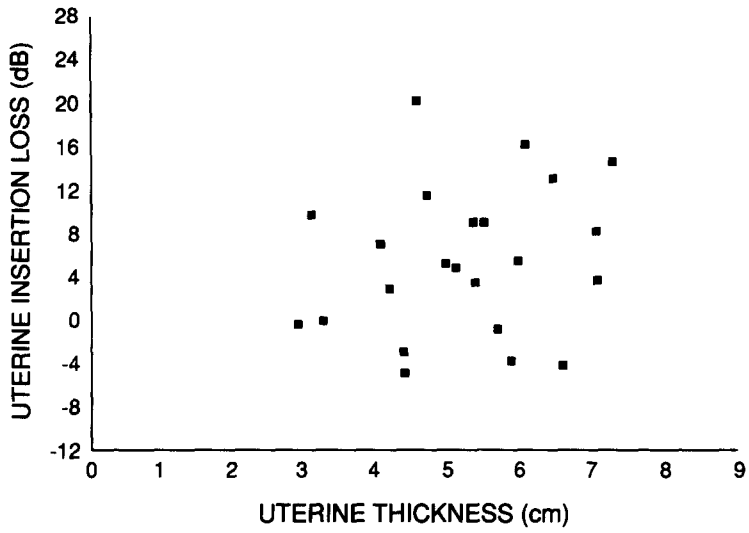

Fig. 3. This figure demonstrates uterine insertion loss in dB when plotted against uterine thickness in centimeters. There is no significant statistical relationship noted.

The absence of a thickness-dependent effect upon IL_{UTERUS} is probably because the estimated uterine (myometrial) attenuation coefficient is quite low.

DISCUSSION

Ultrasound is a physical agent and is known to produce mechanical disruption in discrete media. The physical changes associated with ultrasound include pressure changes, changes in tension, shearing stress, expansion, compression, acceleration of particles and changes in particle velocity. Ultrasound is known to produce bioeffects both *in vivo* and *in vitro* experimental models (O'Brien 1984, 1991). These bioeffects occur not only at varying ultrasonic intensities but are also related to exposure times. Despite the availability of these data, there are very few data defining the actual ultrasound intensities to which gonadal or embryonic tissues are exposed during the course of a routine "clinical" ultrasound examination. The effects of patient size, stage of gestation and duration of exposure remain completely undefined. Similarly, the relationship between *in vitro* maximum values of ultrasonic quantities measured and *in vivo* exposure levels of focused ultrasound systems remains unknown. Mathematical modeling is questionable due to the nonlinearities and the differences between free-field ultrasonic measurements in water (very low loss medium) and in tissue (much higher loss medium). In fact, the minimal information available in the older literature relates to older equipment which did not have the beam focusing capabilities that are available with current equipment.

As part of an ongoing National Institutes of Health (NIH) funded research project designed to address the issues of *in situ* exposimetry in reproductive ultrasound examinations, we have previously reported the average insertion loss during an ovarian ultrasound examination to be 6.2 ± 3.5 dB in the full bladder condition and 7.3 ± 4.9 dB in the empty bladder condition. Applying the fixed-attenuation tissue model to our data, we determined the attenuation coefficient to be 2.56 ± 1.47 dB/MHz when normalized for a 2.4 MHz frequency in the full bladder condition. The attenuation coefficients for the full and empty bladder conditions at a 2.4 MHz frequency, when the overlying tissue model was applied, were 0.89 ± 0.71 and 0.45 ± 0.32 dB/cm-MHz, respectively (Siddiqi et al. 1991). The mean values for the fixed-attenuation tissue model's attenuation coefficient were about a factor of 3 greater than the values proposed to model the attenuation coefficient (Carson et al. 1989b); the mean values for the overlying tissue model's attenuation coefficient were a factor of 2 to 3 greater than the values used by the Center for Devices and Radiological Health (CDRH) of the Food and Drug Administration (FDA) (NCRP 1983).

Our current study was designed to determine the specific contributions of the maternal abdominal wall and uterus (myometrium) to the average insertion loss during a routine non-pregnant reproductive ultrasound examination. In order to do so, calculations were of necessity based on the overlying tissue model which assumes that attenuation consists only of that due to intact tissue, and any fluid path (bladder urine) contributes negligible or no attenuation. To quantify loss of the ultrasonic signal, two terms are generally

used: attenuation and insertion loss. Attenuation generally implies the loss is due to tissue attenuation properties (absorption and scattering) whereas insertion loss generally implies loss as determined by the measurement procedure (includes tissue attenuation properties and beam property effects). In our studies, we have exclusively determined mean insertion loss values from the *in vivo* and *in vitro* measurements. In fact, the attenuation coefficients have been calculated for both the anterior abdominal wall and the uterus (myometrium) using the measured insertion loss values.

There have been a few studies, summarized in Stewart and Stratmeyer (1982) and NCRP (1983), which estimate the ultrasonic attenuation in tissues overlying the first trimester embryo, *i.e.,* the anterior abdominal wall and anterior uterine wall. These data, however, are limited in that they were obtained *in vitro* in non-perfused tissues. In the Stewart and Stratmeyer summary, they reported the insertion loss to be in the range from 2–12 dB for frequencies of 2 and 2.25 MHz. Carson et al. (1989a), on the other hand, estimated the insertion loss to be 3.9 dB at 3.5 MHz. Another calculation (Table 2.4 in NCRP 1983) estimated the loss to be 4.1 dB at 3.5 MHz. The latter 3.9 and 4.1 dB estimates are much lower than our measured insertion loss values, *i.e.,* 8.2 ± 5.6 dB for the full bladder condition and 13.9 ± 5.8 dB for the empty bladder condition. Stewart and Stratmeyer's range (2–12 dB) includes most of our values. In fact, if we assume that our full bladder condition most closely approximates the early pregnancy state (where the ultrasound beam traverses the anterior abdominal and uterine walls and amniotic fluid before reaching the embryo), our average insertion loss value is still greater by at least a factor of 2 when compared to what has been postulated in the past by modeling techniques.

Interestingly, in a summary of five studies (Stewart and Stratmeyer 1982), distances between the abdominal surface and the uterine cavity in early pregnancy ranged between 2 to 11 cm. Using a worst-case approach (Carson et al. 1989b), similar distances were estimated to be 2.6 cm. Our mean value of 2.47 ± 0.73 cm for anterior abdominal wall thickness is quite consistent with this worst-case estimate assuming, of course, that the anterior uterine wall thickness is negligible and there is no signal loss through amniotic fluid prior to the beam reaching the embryo.

The mean abdominal wall insertion loss in our study was 8.2 ± 5.6 dB (Table 1), and the predicted average loss in dB/cm-MHz was 1.17 (Fig. 2), while the actual calculated attenuation coefficient value was 1.39 ± 0.89 dB/cm-MHz. Similarly, the mean uterine (myometrial) insertion loss in our study was 5.8 ± 6.8 dB, and the predicted average loss in dB/cm-MHz using regression analysis was 0.52 (Fig. 3). The actual calculated attenuation coefficient value (from eqn 11) for the uterus was 0.14 ± 0.17 dB/cm-MHz. In essence, we have estimated the attenuation coefficient in one of two ways for both the abdominal wall and uterus (myometrium): firstly, by the equation $A_0 = \langle IL \rangle / (f_c d_{nf})$; and secondly, by using simple regression analysis to determine the best fit model for a given set of conditions. The large discrepancy between the attenuation coefficient values for the uterus derived by the two methods may be explained by the fact that there is no statistically significant regression model which describes the relation between uterine insertion loss and uterine thickness. We propose that the value 0.14 ± 0.17 dB/cm-MHz is much more representative of the true uterine attenuation coefficient than the predicted value of 0.52 dB/cm-MHz. Interestingly, in the empty bladder condition where the sound beam does not traverse any non-fluid pathway (passes through anterior abdominal wall and then through uterus), the attenuation coefficient for the combined abdominal wall and uterine tissues is 0.77 dB/cm-MHz, which is typical for soft tissue (NCRP 1983).

As may be noted, the average insertion loss for the abdominal wall is at least twice if not more than that for the uterus (myometrium). This supports our hypothesis that the maternal abdominal wall is the principal source of ultrasonic energy loss whereas the myometrium is a low loss tissue. This difference is even greater if one considers the fact that in our study, the mean uterine (myometrial) thickness (5.23 ± 1.27 cm) was almost twice the mean thickness of the abdominal wall (2.47 ± 0.73 cm). This is because in the normal, non-pregnant state, the beam path is along the long axis of the anteverted uterus (Fig. 1b). During pregnancy, the abdominal wall does not change in thickness to any significant degree whereas the uterine wall thins significantly with advancing gestation, again supporting our hypothesis.

The issues of which of the various proposed tissue models most closely approximates experimental data, and whether current derating factors used by regulatory agencies are in fact correct, will be addressed in a separate manuscript. Suffice it to say that although the multiplicative model was the best fit for the insertion loss values for the total data set ($n = 46$), full bladder data set ($n = 23$) and the abdominal wall data set ($n = 23$); for the range of distances evaluated, the linear model assumption provided very close, almost identical values. For the empty bladder data set, the linear model was in fact the best fit while for the

uterus (myometrium) there was no statistically significant regression model although the closest fit was again the linear model. Thus, for clinical purposes one can assume that the ultrasound beam behaves according to a linear model.

Finally, we have provided ultrasound users and researchers the means to estimate embryonic and fetal exposure levels given the power output of an ultrasound system and the mean thickness of the maternal abdominal and uterine walls. Ongoing studies will allow us to improve upon our current data.

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