Input Impedance of the Lower Abdominal Aorta in Chronically Instrumented Fetal Sheep

Hobe Schröder, Emine Cetin, Bernd Hünke, Martin Carstensen

Abstract Five fetal sheep (gestational age, 118 to 125 days) were instrumented to measure impedances of the lower abdominal aorta. Four days after surgery, flow and pressure pulses were recorded during control conditions and during infusion of norepinephrine (0.3 to 3 μg·min⁻¹·kg⁻¹ body weight) or angiotensin II (0.2 to 3 μg·min⁻¹·kg⁻¹). The protocol was repeated after injection of hexamethonium (10 mg·kg⁻¹). Moduli and phases of impedances for the first ten harmonics were calculated by fast Fourier transformation. During control, input resistance was 4300±940 dyne·s·cm⁻⁵ (mean±SD) at a mean blood flow of 13.3±2.4 cm²·s⁻¹ and pressure of 54 960±6980 dyne·cm⁻². Moduli fell to 50% of input resistance between 2 and 3 Hz and, declining continuously, reached a minimum of 20% near 10 to 12 Hz, then increased slightly to 30% at about 30 Hz. At the first three harmonics, flow was always leading pressure. Infusion of angiotensin or norepinephrine increased impedance moduli significantly. Resistance increase was largest with angiotensin (19 800±11 370 dyne·s·cm⁻⁵), but no difference was detectable between angiotensin and norepinephrine when related to the same increase of input resistance. The position of the minimum seemed to be unchanged at high resistance values, but relative impedance moduli were smaller than during control, and low-frequency phases were significantly more negative. An analog of inerterance, compliance, and resistance to steady flow was used to simulate impedances, and the effect of resistance increases on flow waveforms in the fetal abdominal aorta was calculated. It is concluded that input impedances in the fetal lower abdominal aorta largely reflect properties of the umbilical arteries, supplemented with some compliance from the descending aorta and its other branches. (Circ Res. 1994;74:641-649.)

Key Words: • input impedances • fetal sheep • abdominal aorta • flow waveform

The fetal circulation is unique because nearly 50% of cardiac output is directed to one organ, the placenta. It might be anticipated that this situation will be reflected by values of circulatory parameters (eg, pressure, flow rate, resistances) that are unusual for adult animals. Whereas this is well-known for steady-state or "time-averaged" data, only two studies so far have investigated the hemodynamic (in its true sense) properties of the fetal circulation. Recently, the input impedance of umbilical arteries and the thoracic aorta have been determined in fetal sheep. It was found that the impedance pattern of the umbilical arteries is different from large adult vessels, but input impedances of the fetal thoracic aorta resemble those known from adult animals. In fetal sheep, the umbilical arteries are direct continuations (not side branches) of the lower abdominal aorta, which therefore is of interest as a "transitional zone" between thoracic aorta and umbilical arteries.

We measured abdominal aortic input impedance because no such data are available for this vessel and because impedance influences the flow waveform, which can be routinely observed with ultrasound Doppler technology (as frequency or velocity waveforms). In obstetrics, transcutaneous Doppler velocimetry allows the noninvasive and innocuous investigation of the fetal cardiovascular system, which otherwise is almost inaccessible. The flow-dependent waveforms are characterized by indices that frequently are the basis for clinical assessment of the fetal circulatory state. Knowing the relation between flow waveforms and vascular impedance (which determines blood flow rate), therefore, is of considerable importance. This applies especially to the umbilical arteries and to the lower abdominal aorta, in which blood flow rates and blood flow waveforms may depend on properties of the umbilical-placental circulation.

Impedances, which are resistances to pulsatile flow, in combination with the shape of pressure wave, determine the flow waveform. To adequately describe input impedances, frequencies have to be varied as well as resistances. We used infusion of norepinephrine and angiotensin into fetal sheep to increase peripheral resistance while measuring pulsatile blood flow and pressure in the lower abdominal aorta. Because fetal heart rate decreases because of the baroreceptor reflex, the experiments were repeated after injection of hexamethonium to suppress the reflex.

The results demonstrate that in the lower abdominal aorta, the spectrum of relative moduli is comparable to that of the umbilical arteries, but phases resemble those seen in the thoracic aorta.

Methods

Experiments and surgery were approved by the state public health agency.

Surgery

General anesthesia was induced in five pregnant sheep (merino, whiteface) with single fetuses (gestational age, 118 to 125 days) with 4 mL bupivacaine 0.5% (intrathecal; Carbostin, Astra Chemicals, Wedel, FRG) and barbiturate (Suri-
tal, Parke Davis, Freiburg, FRG) and maintained with barbiturate and dihydroxy-xylidino-thiazin (Rompun, Bayer, Leverkusen, FRG) as required. The ewe was placed in the left lateral position, and the uterus and fetus were exteriorized into a translucent plastic bag. After uterotomy the fetal lower abdominal aorta was approached cranial from the left iliac spine, leaving the (Peritoneum unopened. An inductive flow probe (Omnica Medical Series SFC 400, circumference 18 or 20 mm; Carolina Medical, King, NC) was placed around the aorta 1 to 2 cm below the renal arteries. A catheter (Sensodyn F-PO-3F-1, side opening; B. Braun Melsungen AG, FRG) with pressure tip transducer was inserted through the left femoral artery into the abdominal aorta; its opening was found during later autopsy to be 2 to 15 mm distal from the flow probe. Catheters (outer diameter, 1.5 mm) were inserted through the right femoral vessels into the iliac artery or lower abdominal aorta distal from the pressure transducer and into the fetal inferior vena cava. A catheter was placed in the amniotic/allantoic cavity.

All attached to the ewe’s left flank.

Catheters were flushed daily (heparin 1000 U/mL of saline), and both mother and fetus received 1 g/d cefotaxime (Claforan, Hoechst, Frankfurt/Main, FRG) for antibiotic protection.

Flow Measurement

In fetal sheep, heart rate may increase to almost 300 beats per minute or 5 Hz. If the first 10 harmonics are to be used for impedance calculations, flow or pressure waves up to 50 Hz will be of importance.

Flow was inductively measured with a Cliniflow II (model 701D, Carolina Medical, 50-Hz version) flowmeter, which yielded pulsatile and mean blood flow rates simultaneously. The frequency response (−3 dB, or about one-third reduction) was adjustable to 25, 50, and 100 Hz. Most measurements were made at 25 Hz, but this was increased to 50 or 100 Hz depending on the signal-to-noise ratio. The amplitudes of flow harmonics were later corrected according to frequency response curves supplied by the manufacturer (see below). Signal processing in the flowmeter causes a constant time delay of 12 to 15 milliseconds. (Phase correction included an additional slight time delay of 0.4 millisecond for flows caused by the multiplexing sampling of flow and pressure data; see below.)

Pressure

Both the pressure tip transducer and the custom-built bridge amplifier had a gain of 1 for frequencies up to 50 Hz (−3 dB beyond 10 kHz) and no phase delay. Pressure harmonics and phases therefore were not corrected. Pulsatile pressure and flow were not measured precisely at the same location. At an aortic pulse wave velocity of about 4 m · s−1, pulse wave delay could be at most 3 milliseconds. Data were not corrected for this delay.

The other vascular and amniotic fluid catheters were coupled with strain-gauge pressure transducers (Statham P23Gb and P23BB, Gould Inc, Cleveland, Ohio) or piezoelectric transducers (Honeywell 156PC06GW2, Offenbach, FRG) arranged at the same level and with the appropriate amplifiers (Hellige, Freiburg, FRG; Gould Inc; Honeywell). Fetal heart rate was derived from arterial pressure pulse (Biotach, Gould Inc).

Data Acquisition

Fetal signals (fetal arterial blood pressure, amniotic fluid pressure, mean aortic blood flow) were continuously recorded (1 cm · min−1) as 4-second means on a polygraph recorder (Servogor 462, Metrawatt, Nürenberg, FRG). The output of each channel was sampled every 3 seconds by a personal computer (Compaq Portable II) with a 12-bit analog-to-digital (A/D) converter (Tecmar Labpac, Scientific Solutions Inc, Solon, Ohio) and stored on disk as 30-second averages.

The zero position of pressures and the zero offset values of the flowmeter were checked frequently and adjusted when necessary.

The pulsatile pressure and flow signals were sampled when required at 200 Hz for 2.5 seconds and stored with date and time information. Depending on fetal heart rate, each record contained 4 to 10 cardiac cycles. The A/D converter readings were later converted to flow rates or pressures using calibration signals from the flowmeter or using a calibration procedure for the tip pressure transducer after the experiment. Fetal arterial blood pressure (from the fluid-filled catheter) during periods of stable blood pressure was compared with the mean of the pulsatile pressure (tip transducer), and the latter was adjusted (zero offset) if necessary.

Fetal blood gas analysis was performed with an ABL2 (Radiometer, Copenhagen, Denmark). Fetal temperature was taken to be 0.5°C higher than maternal rectal temperature, and blood values were corrected accordingly. Oxygen saturation was measured with an OSM2 (Radiometer).

Data Processing

Amniotic fluid pressure was subtracted from fetal arterial blood pressure and from the pressure pulses to obtain true arterial blood pressure. Resistance to steady flow (input resistance) was calculated as mean aortic pressure divided by mean flow from the pulsatile recordings.

Using a commercial program package for signal analysis (FAMOS, imc-Mess-Systeme, Berlin, FRG), usually 5 (range, 2 to 10) consecutive cardiac cycles were chosen from each pressure and flow record for analysis. The quality of flow and pressure waveforms was estimated on the basis of signal stability, amount of noise, and similarity of pulses and scored accordingly (1 to 6). Only records (n=357) with scores 3 or smaller were evaluated (Fig 1). After the flow signal was shifted according to the respective constant time delay of the flowmeter, pressure and flow data of the chosen cycles were extracted from the records, measuring from flow peak to flow peak. Fast Fourier transformation was performed to obtain the amplitude and phase of pressure and flow as a function of frequency. From these results, moduli and phases were accepted that corresponded to moduli peaks (harmonics) of the complex flow. The peaks usually were well defined and occurred only at frequencies that were whole-number multiples of the heart rate (beats per minute divided by 60). The flow moduli were corrected to account for the flowmeter gain. Pressure moduli then were divided by flow moduli, and flow phases were subtracted from pressure phases to obtain the moduli and phases of impedances for each harmonic. Thus, impedances at zero frequency (=input resistance or resistance to steady flow) and at the first 10 harmonics were obtained. Finally, relative impedance moduli were calculated by dividing the impedance moduli 1 to 10 by the input resistance of each recording.

Pressures are given as dyne · cm−2 (1 mm Hg=1334 dyne · cm−2) and flow rates as cm3 · s−1. Therefore, the unit of resistance or impedance is given in volumetric units as dyne · s · cm−3, in accordance with a large body of literature. Phases are expressed as degrees (−180 to +180).

FAMOS was also used to calculate the analog model of the circulatory system (see below).

Experimental Procedure

Experiments were performed on at least 2 days starting at day 4 after surgery. On each day, a control period (0.5 to 1 hour) was followed by periods of drug infusion with norespinephrine hydrochloride (0.3 to 3 µg · min−1 · kg−1; Arterenol, Hoechst) and then angiotensin (0.2 to 3 µg · min−1 · kg−1; angiotensin II amide, Hypertensin, Ciba-Geigy, Wehr, FRG) into the fetal vena cava catheter. The recovery time between the two latter experimental periods was at least 1 hour. Infusion time depended on the observed
impedances. Data displayed in the Table or used for statistics were averaged for each animal and for each experimental period. Differences from control were evaluated by the two-tailed Wilcoxon test for paired differences (n=5). Impedance spectra were compared relating moduli and phases of the respective harmonics. As a measure of the complete spectrum of relative moduli, the sum of moduli at harmonics 1 to 10 was used. Significance level was P<.05. Linear correlations were calculated where appropriate. Calculations were performed with spss/pc (SPSS Inc, Chicago, Ill).

**Results**

The mean fetal weight was 3.7±0.9 kg. Fetal blood gas values, mean arterial pressure, heart rate, mean aortic blood flow rate, and resistance to steady flow are summarized for the experimental periods in the Table. Infusion of norepinephrine or angiotensin increased arterial blood pressure and resistance, whereas blood flow rate and fetal heart rate decreased. The heart rate reduction was suppressed by hexamethonium. Hexamethonium "unmasked" the cardiotropic effects of norepinephrine (Table), because blood flow rate during infusion of norepinephrine increased despite raised input resistances.

Blood gas values deteriorated during periods of greatly increased resistance and reduced flow rates (Table).

**Flow and Pressure Pulses**

Fig 1 displays pressure and flow patterns of one animal under control conditions (top) and during infusion of angiotensin (bottom). A large variety of waveforms was seen within one animal during "stable" control conditions and between animals as well, considerable changes sometimes occurring within two or three beats. This was especially true during diastole, when flow rates could be slowly decreasing or constant or (as shown here) display a small oscillation. In general, pressure "pulsatility" (the pulse amplitude as related to the mean) was much less than flow pulsatility, and the diastolic variability of flows was more distinct than that of pressures. During periods of large resistance increases (Fig 1, bottom), the flow pattern could demonstrate two distinct diastolic fluctuations with reversal of the flow direction, whereas the pressure waves displayed only a small (Fig 1, bottom) or even no diastolic wave.

**Impedances**

**Moduli (Amplitudes)**

Fig 2 displays the medians (±25%) of impedance moduli under control conditions and during the experimental periods. As expected, infusion of norepinephrine or angiotensin significantly increased input resistances and impedance moduli as well, regardless of heart rate decreases or increases (blockage with hexamethonium), whereas injection of hexamethonium alone had no consistent effect. Impedance moduli 1, 2, 3, 4, and 6 of all experimental periods (except hexamethonium) were significantly different from control. Evidently, moduli decreased with frequency up to 10 to 12 Hz, demonstrating more or less clearly a shallow trough.

No difference was detectable when angiotensin and norepinephrine impedance spectra were compared at the same level of input resistance (on average, input resistance was larger with angiotensin than with norepi-
Circulatory Parameters and Arterial Blood Gas Values During Experimental Periods

<table>
<thead>
<tr>
<th>Parameters</th>
<th>CTRL</th>
<th>HXMT</th>
<th>ANGI</th>
<th>ANHX</th>
<th>NEPI</th>
<th>NEHX</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pressure, dyne · cm⁻²</td>
<td>54 ±690±69870</td>
<td>46 ±960±8070</td>
<td>86 ±130±6030*</td>
<td>89 ±680±1240*</td>
<td>80 ±970±5040*</td>
<td>90 ±707±830</td>
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<tr>
<td>Flow, cm³ · s⁻¹</td>
<td>13.3±2.4</td>
<td>12±1.9</td>
<td>6.4±3.3*</td>
<td>9.6±4.0*</td>
<td>11.5±2.2*</td>
<td>14.4±2.8</td>
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<tr>
<td>Resistance, dyne · s · cm⁻⁵</td>
<td>4300±940</td>
<td>4030±1060</td>
<td>19 ±800±11 370*</td>
<td>11 ±480±4600*</td>
<td>7540±1700*</td>
<td>6520±1090*</td>
</tr>
<tr>
<td>FHR, bpm</td>
<td>165±14</td>
<td>148±7*</td>
<td>146±23</td>
<td>174±21</td>
<td>149±15</td>
<td>188±26</td>
</tr>
<tr>
<td>pH</td>
<td>7.324±0.041</td>
<td>7.313±0.025</td>
<td>7.126±0.165*</td>
<td>7.226±0.089*</td>
<td>7.308±0.033*</td>
<td>7.313±0.033</td>
</tr>
<tr>
<td>P0₂, mm Hg</td>
<td>25.3±3.9</td>
<td>24.7±4.5</td>
<td>19.5±7.2</td>
<td>18.7±10.4</td>
<td>26.9±3.0</td>
<td>24.2±5.4</td>
</tr>
<tr>
<td>Pco₂, mm Hg</td>
<td>55.5±4.3</td>
<td>56.8±4.3</td>
<td>85.2±29.8</td>
<td>69.4±12.6*</td>
<td>58.2±5.2</td>
<td>55.2±4.8</td>
</tr>
<tr>
<td>So₂, %</td>
<td>42.5±6.0</td>
<td>40±6.7</td>
<td>14.8±8.3*</td>
<td>20.5±13.3*</td>
<td>43.9±8.5</td>
<td>43.3±14.5</td>
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<tr>
<td>BE, mmol · L⁻¹</td>
<td>1.5±2</td>
<td>1.2±1.2</td>
<td>-4.5±6.2*</td>
<td>-1.1±3.0</td>
<td>1.3±0.9</td>
<td>0.6±1.5</td>
</tr>
</tbody>
</table>

CTRL indicates initial control; HXMT, after injection of hexamethonium; ANGI, during infusion of angiotensin; ANHX, after injection of hexamethonium and during infusion of angiotensin; NEPI, during infusion of norepinephrine; NEHX, after injection of hexamethonium and during infusion of norepinephrine; FHR, fetal heart rate; bpm, beats per minute; P0₂, oxygen partial pressure; Pco₂, carbon dioxide partial pressure; So₂, oxygen saturation; and BE, base excess. Pressure, flow, and resistance are means in the abdominal aorta.

Values are mean±SD from the averaged (five experimental period) values of five animals. For fetial circulation, values are based on 30-second averages within 0.5 minute of the corresponding pulsatile pressure and flow recordings. Fetal blood gas values are from arterial blood samples during control, 10 to 15 minutes after HXMT injection, or at the end of experimental periods. *Significant differences between control and experimental periods.

nephrine; Table). Thus, the increase of input resistance per se was more important than the vasoactive substance that had induced the increase.

When moduli were related to input resistance, the dependence of these relative moduli on frequency was similar for the experimental periods. Only during angiotensin was the sum of the relative moduli at harmonics 1 to 10 significantly smaller than the control value. Regression analysis of the dependence of relative moduli on resistance revealed, however, that all relative moduli decreased with resistance (for these calculations, the logarithm of resistance was taken for linearization).

Because of variation of resistances within each experimental group, therefore, group analysis according to the experimental periods masked the changes of relative moduli with resistance. When relative moduli are displayed as depending on resistance and frequency, the relative moduli of all experimental data can be represented as a surface (Fig 3). Besides some "bumps," the surface has two characteristic features: relative moduli decrease with increasing frequency to a minimum at 10 to 15 Hz and decrease with increasing resistance. At high resistances, the increase of moduli at higher frequencies is less distinct than at low resistances, or even absent. The same basic features could be observed for each individual animal and all experimental periods combined and for all animals combined and each experimental period, with the exception of control and hexamethonium, for which the variation of resistance was insufficient.

Fig 4 demonstrates the effect of input resistance on relative impedance spectra at the extreme range of resistances (<5000 dyne · s · cm⁻⁵ [mean, 3980±710] and >8000 dyne · s · cm⁻⁵ [mean, 16 000±9900]). The spectrum of relative moduli was significantly smaller in the high-resistance group, and the rise of relative moduli after the minimum seemed to be less distinct. The position of the minimum itself (about 12 Hz) was unchanged.

Phases

Phases started to vary distinctly with the fourth or fifth harmonic (Fig 2). Phases were never positive (eg, flow was leading pressure) for the first three harmonics. This was true even when phases were calculated neglecting the constant time delay of the flowmeter (see above). A consistent and significant finding was that angiotensin phases (harmonics 1 to 3) were more negative than control phases and that the phases of the first four relative harmonics were significantly more negative at high resistance values (Fig 4). There was a tendency to approach zero at higher frequencies.

Discussion

Criticism of Methods

Some problems associated with our method of collecting and evaluating data should be discussed briefly.

Pressure and flow waveforms were registered "blindly," ie, not knowing the stability and quality of the signals, and only for a short time span (2.5 seconds). Careful judging of the signals (see "Methods") overcame the first problem (while reducing the data set), but the registration of only a short train of pulses made the more effective spectrum analysis by correlation (Milnor,¹⁵ pp 351 to 357; Randall¹⁶) impossible.

Pressure and flow signals were not filtered (100 Hz) before digital sampling, which is usually done to avoid aliasing caused by high-frequency components. Because the frequency response of the flowmeter is equivalent to a filter and because physiological pressures are probably negligible above 50 Hz, we believe that aliasing does not corrupt our data.

The variation of impedance data, especially at higher harmonics, is large. Impedance moduli are ratios of pressure over flow harmonics, and therefore, variations of small flow moduli may generate extreme fluctuations.
Fig 2. Graphs showing moduli and phases during initial controls (top left), after injection of hexamethonium (top right), and during infusion of angiotensin (middle left) and norepinephrine (lower left) without and with (middle right and lower right) hexamethonium. Frequency error bars are ±1 SD. Because of logarithmic scale, the width of frequency error bars is the same for each harmonic. Error bars of moduli and phases indicate quartile range above and below the respective medians, which are centered on mean frequencies. SD of frequencies, medians, and quartile ranges are based on numbers (indicated as n on the figure panels) of pressure and flow records analyzed during the respective experimental periods.
of impedance moduli. This could be partly overcome by the use of logarithms or, as in this study, of medians.

Impedance moduli were much more stable than phases. In fact, all data were evaluated also using only single pulses of flow and pressure with no significant differences from the averaged modulus values presented in "Results." Phases, however, appeared to be reliable only (harmonics 1 to 4) when the method of evaluation described in "Methods" was applied.

Introductory Remark

In fetal sheep the terminal aorta continues into the common umbilical artery, which branches within 0.5 to 1 cm into two umbilical arteries 30 to 50 cm long. A mean input pressure of 50,000 to 55,000 dyne · cm⁻² drives a blood flow of about 3 cm³ · s⁻¹ · kg⁻¹ body weight through two vessels that have no side branches. The low resistance of the umbilical circulation gives rise to low flow pulsatility.⁴⁻⁵.⁹ The situation is unique and explains why diastolic blood flow in the fetal descending aorta under normal conditions is never zero or reversed, in contrast to common findings in adult animals.¹⁵

Our site of measurement was the descending aorta 3 to 5 cm proximal to the common umbilical artery. This is close to the large aortic continuations, and it seems likely that input impedances here will reflect, to a large extent, properties of the distal vessels, especially the umbilical arteries, the iliac and femoral arteries, etc. The predominance of the umbilical arteries will be enhanced by the experimental situation in which both femoral arteries were completely blocked by inserted catheters.

It is difficult to find a site in the adult circulation that may reasonably be compared to the fetal abdominal aorta. Perhaps the pulmonary artery (Milnor,¹⁵ pp 185 to 188) or very dilated aortic main branches (O'Rourke,¹⁴ Fig 3, middle panel) are close.

Control Values

The spectrum of aortic input impedances depends on body size and on the position of pressure and flow probes. Most measurements have been made at the ascending aorta (Milnor,¹⁵ pp 181 to 184). For example, in the ascending aorta of rabbits¹⁷ (average weight, 2.95 kg), input resistance (impedance modulus at 0 Hz) was approximately 47,000 dyne · s · cm⁻² (for volume flow, or 7500 dyne · s · cm⁻² for flow velocity). Impedance moduli in rabbits reached a first minimum at about 4 Hz with about 5% of input resistance, then rose to 10% and reached a second minimum at 12 Hz. Phases were negative (flow leading pressure), with a tendency to cross the zero line at the first impedance minimum. Basically the same pattern was seen in the thoracic aorta of fetal sheep.³ Input resistance was about 4500 dyne · s · cm⁻², which is very close to our control value. There was a first minimum at about 6 Hz; phases were approximately −20° to −40° at 3 and 6 Hz and crossed the zero line at 7 to 8 Hz.

In general, moving down the aorta, the impedance minimum will shift to higher frequencies, and the "oscillation" of input impedance (ie, the difference between minimum and maximum values as related to the mean) will increase as well as the modulus values at higher frequencies. At the origin of large aortic branches (brachiocephalic artery; Avolio et al,¹⁷ Fig 3), the relative moduli seem to be larger, especially at low peripheral resistances (femoral artery¹¹).

Our data demonstrate that input resistance at the lower abdominal aorta of fetal sheep is about 4000 dyne · s · cm⁻², a low value that reflects the low arterial
pressure (about 50% of the adult values) and the presence of the low-resistance placental circulation. Impedance then decreases to about 50% of input resistance at 2 to 3 Hz (Figs 2, top left, and 4) and reaches a minimum of 20% in the range of 11 Hz. The relative moduli thus display smaller values than in the umbilical arteries of fetal sheep, but they resemble those of large (dilated) peripheral vessels and of the fetal thoracic aorta (Langille and Adamson, Fig 3A). The position of the minimum (11 Hz, Fig 2, top left), however, is distinctly different from the fetal aorta. It is conspicuous that in Adamson et al (Figs 5 and 6), the first trough of the spectra of the umbilical arteries usually is shaped by one harmonic only, and the moduli of the second trough at 10 to 15 Hz are smaller than the modulus of the first one. The overall impression, therefore, is that in umbilical arteries there is a more or less continuous drop of relative moduli up to 10 to 15 Hz, and this pattern is close to our observation.

Up to about 9 Hz (harmonics 1 to 3), phases were always negative. This seems to be a common feature of aortic and large-vessel input impedances (Milnor, p 184) but not of the umbilical artery, in which phases of the first harmonic were consistently positive (about 8°; Adamson et al). In the lower abdominal aorta we have never seen positive phases. Therefore, the impedance spectra (relative moduli) observed by us display features that are intermediate between those of the fetal thoracic aorta and those of the umbilical arteries.
Resistance Increases

The influence of angiotensin or norepinephrine on aortic input impedance has frequently been investigated (usually ascending aorta; Milnor,15 [pp 196 to 200]). Whereas an increase of input resistance is almost a necessity, other changes are more equivocal (Adamson et al; Milnor,15 Table 7.3). Typically, because of increasing reflections, enlarged oscillations of impedance moduli and a shift of the minimum to higher frequencies were seen. These changes were seen in the fetal thoracic aorta also. However, alterations of this kind are not detectable with our data (Figs 2 and 4). In some cases (especially during infusion of angiotensin), during resistance increases, the impedance modulus minima disappeared and the moduli decreased continuously to higher frequencies, but it is uncertain whether this corresponds to a frequency shift. A lack of these changes (eg, enlarged oscillations of impedance moduli and shift of the minimum) in the umbilical artery has also been noted by Adamson et al.2 The authors put this result down to the extremely long and thick-walled umbilical arteries, which constrict preferentially to angiotensin, whereas the more compliant vessels of the cotyledons are less affected. In their experiments,2,3 infusion of angiotensin led to more negative phase shifts of input impedances (thoracic aorta and umbilical arteries), a finding in accordance with our results.

Frequency shift and increased oscillation of impedance moduli after vasoconstriction are thought to be caused by increased pulse wave velocity (increased vessel distension) and increased reflection coefficients.14 In the umbilical arteries, however, the effects of angiotensin were interpreted to actually decrease reflections, because an impedance mismatch between umbilical arteries and cotyledonary arteries was reduced.2 We did not estimate the characteristic impedance of the lower abdominal aorta because of the unusual course of the modulus spectra. Reflection coefficients, therefore, cannot be calculated.

We conclude that in the fetal lower abdominal aorta, flow leads pressure at low frequencies, and moduli decline moderately with frequency. There appears to be one minimum at about 10 to 12 Hz. Thus, input impedances, measured close to the origin of the umbilical arteries, display properties that are somewhere between those of the fetal thoracic aorta and those of umbilical arteries. Because more than 80% of blood flow in the lower abdominal aorta of fetal sheep is directed to the umbilical arteries, the finding seems reasonable.

Analog Model

The two important effects of peripheral vasoconstriction, ie, decrease of relative moduli and the shift of phases, can be simulated with an analog of the terminal impedance (ie, resistance downstream of the flow probe and intravascular pressure transducer). The model is mostly formal and combines the smallest possible number of different components that could simulate our results to some extent. It is meant to mimic the behavior of pressure-flow relations in the fetal abdominal aorta but not to explain them. Fig 5 illustrates the arrangement of an inductance (L), two resistances (R<sub>L</sub>, R<sub>C</sub>) and a capacitance (C) representing inerence and steady-flow resistances to flow, and vascular compliance. When appropriate values (Fig 5) for L, R<sub>L</sub>, R<sub>C</sub>, and C are
chosen, impedance spectra as displayed in Figs 6 and 7 can be obtained.

Obviously, the model simulates only the main features of input impedances, and the numerical correspondence of resistances and phases is poor (Figs 4 and 6). It may, however, be used to explore the influence of model components on the flow waveform in the lower abdominal aorta (see introduction). In Fig 8 (top), an experimental pressure pulse was analyzed by fast Fourier transformation and reconstructed from frequencies of 0 to 20 Hz (dotted line). From the model (Fig 5), complex flows were calculated as pressure/impedance (both as complex quantities), and the resistance $R_c$ was varied as indicated in Fig 8. Pressure was increased also by multiplication factors as shown, thus leaving the pressure waveform or oscillations unchanged. Flows then were reconstructed from complex flows by inverse fast Fourier transformation, and the results are displayed in Fig 8 (bottom). Compared with Fig 1, bottom, it is evident that increasing the resistance to steady flow ($R_c$) already generates flow patterns that agree in principle with experimental observations. Thus, the occurrence of diastolic oscillations and zero and reverse flows, all ominous signs in fetal surveillance with Doppler sonography, could indeed be based on a large increase solely of resistances. This does not exclude that vascular compliances, reflection coefficients, cardiac contractility, or other factors may change also. Fig 8 illustrates as well the expected changes of measures of “pulsatility” (eg, resistance index or pulsatility index).

Our data support the conclusion of Adamson et al that the lower-body and umbilical-placental circulation of the fetus has features unknown in adult animals. It is tempting to speculate that the umbilical blood flow constitutes a mostly inertial and resistive load with no compliance, whereas the residue of the lower abdominal aorta and its branches constitutes compliance and resistance. The lower abdominal aorta of fetal sheep performs like the umbilical arteries with some additional compliance.

References

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