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# ANALYSIS OF THORACIC DUCT FLOW WAVES USING FAST FOURIER TRANSFORM IN SHEEP

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*ABSTRACT*

*We measured the lymph flow of the thoracic duct using an ultrasound transit-time flowmeter and then analyzed the obtained flow signals by fast Fourier transform. We found that the wave form included a low frequency component (~ 0.1 Hz) as well as high frequency components which represented cardiac pulsation and respiratory movement. The low frequency component signified an intrinsic thoracic duct pulsation. When venous outflow pressure was increased, the frequency of the thoracic duct pulsation increased, whereas the frequencies of cardiac pulsation and respiratory movement were unchanged. These findings suggest that thoracic duct pulsation is independent of cardiac pulsation and respiratory movement.*

We previously studied the flow pattern of the intact thoracic duct in sheep using an ultrasound transit-time flowmeter (1). We reported that the thoracic duct flow was pulsatile and that the pulsation frequency was approximately 5 $min^{-1}$. However, the flow was measured through a low pass filter (LPF) with a flowmeter setting of 0.1 Hz. Therefore, we were unable to exclude the possibility that the low frequency pulse wave was fusion from cardiac pulsation and respiratory movement. Moreover, even if an original pulsatile flow of the thoracic duct existed, we could not detect it when the pulsation frequency increased to more than 0.1 Hz, because of the 0.1 Hz LPF. To resolve these issues, we recorded the thoracic duct flow through a 10 Hz LPF in this study and detected any pulsatile components in the flow wave by analysis of flow signals by fast Fourier transform (FFT). We found that the wave form of the thoracic duct consists of cardiac pulsation, respiratory movement, and an intrinsic thoracic duct pulsation. Furthermore, we found that thoracic duct pulsation increased to more than 0.1 Hz when venous outflow pressure increased.

## *METHODS*

This study was approved by the University Committee on Animal Resources and conformed to the guiding principles of our institution in the care and use of animals.

### *Lymph Flow Measurement and Its Analysis*

Lymph flow of the thoracic duct was measured with an ultrasound transit-time flowmeter (Model T106, Transonic Systems, Ithaca, NY) (2). The output signal from the ultrasound transit-time flowmeter was filtered at 0.1 or 10 Hz by LPF. The lymph flow signals through 0.1 or 10 Hz LPF were digitally recorded on a computer hard disk with 100 samplings $s^{-1}$. To determine the pulse components of the thoracic duct flow, we analyzed the flow signal using FFT. To perform FFT, we extracted signals for 150 s from the recording and took them as one

unit. We then divided this into three equal parts (50 s). Signals for 40.96 s in each part were put into a Hunning window and we performed 4096 points FFT. We obtained, thereby, the power spectrums of the thoracic duct flow wave. We calculated the mean value of the power spectrums from each of the three parts (40.96 s) to express the data of the period (40.96 x 3 s). This analysis is characterized by a maximum frequency value of 50 Hz and a resolution of 0.0244 Hz.

To express the volume flow of the thoracic duct, we calculated the mean lymph flow rate by an integration of the computer recordings with the zero offset values. Lymph flow was expressed as ml $min^{-1}$.

*Animal Preparation*

Ten adult sheep (mixed strain Corriedale and Suffolk) of similar size (50±10 kg) were fasted for 12 hours before general anesthesia was administered. Five minutes after intramuscular injection of ketamine hydrochloride (10 mg $kg^{-1}$), a catheter was introduced into the right external jugular vein by a direct puncture, through which thiamylal sodium (10 mg $kg^{-1}$) was injected. Through this catheter we infused a Ringer's lactate solution at a rate of 0.2 $kg^{-1}$ $min^{-1}$ throughout the experiment. After endotracheal intubation, the sheep were maintained on 0.5% halothane and 35-45% oxygen in air using a positive-pressure ventilator (Harvard model No. 607, Natick, MA) with a tidal volume of 400-500 ml, a respiratory rate of 18-25 $min^{-1}$ and an end expiratory pressure of 5 cm $H_2O$. A catheter was inserted through the right external jugular vein to measure the venous outflow pressure of the thoracic duct using a transducer (model LPU-0.1-350-0-II, Nihon Kohden, Japan). Arterial blood pressure was monitored from a catheter in the right carotid artery connected to a transducer (model MPV-0.5-290-0-III, Nihon Kohden, Japan). Airway pressure was monitored using a transducer (model LPU-0.1-350-0-II, Nihon Kohden, Japan). Arterial blood gas was measured appropriately using a gas analyzer (ABL2, Radiometer, Copenhagen, Denmark).

After intravenous injection of pancuronium bromide (0.08 mg $kg^{-1}$), we put a flowprobe (model 2SB, Transonic Systems) on the thoracic duct as previously described (1). In brief, we performed a right thoracotomy in the fifth intercostal space. Through a small incision of the parietal pleura we exposed the thoracic duct approximately 1 cm cranial to the median mediastinal lymph node and positioned the ultrasound transit-time flowprobe on the thoracic duct. This procedure was done with meticulous care to completely remove the fatty tissue around the thoracic duct in order to improve acoustic conductivity.

*Protocols*

Arterial, venous and airway pressures as well as lymph flow were continuously recorded on a polygraph (model RM-6000, Nihon Kohden, Japan). The zero level of each pressure was adjusted to the level of the cranial vena cava at its entrance to the heart.

Baseline lymph flow signals were recorded on the computer hard disk from the flowmeter through a 0.1 Hz LPF or a 10 Hz LPF. After the baseline measurement, the cranial vena cava was partially obstructed by constriction using a tape encircling the cranial vena cava in order to increase venous outflow pressure. Venous pressure was increased until the pressure produced a significant depression of lymph flow. After thoracic duct flow became stabilized, we recorded the signals on the computer hard disk from the flowmeter through a 0.1 Hz or a 10 Hz LPF. Then we analyzed the flow wave in the manner described above.

At the end of the study, we performed *in situ* calibration, then killed the sheep by exsanguination.

*Data Analysis*

Data are presented as mean ± S.D. The

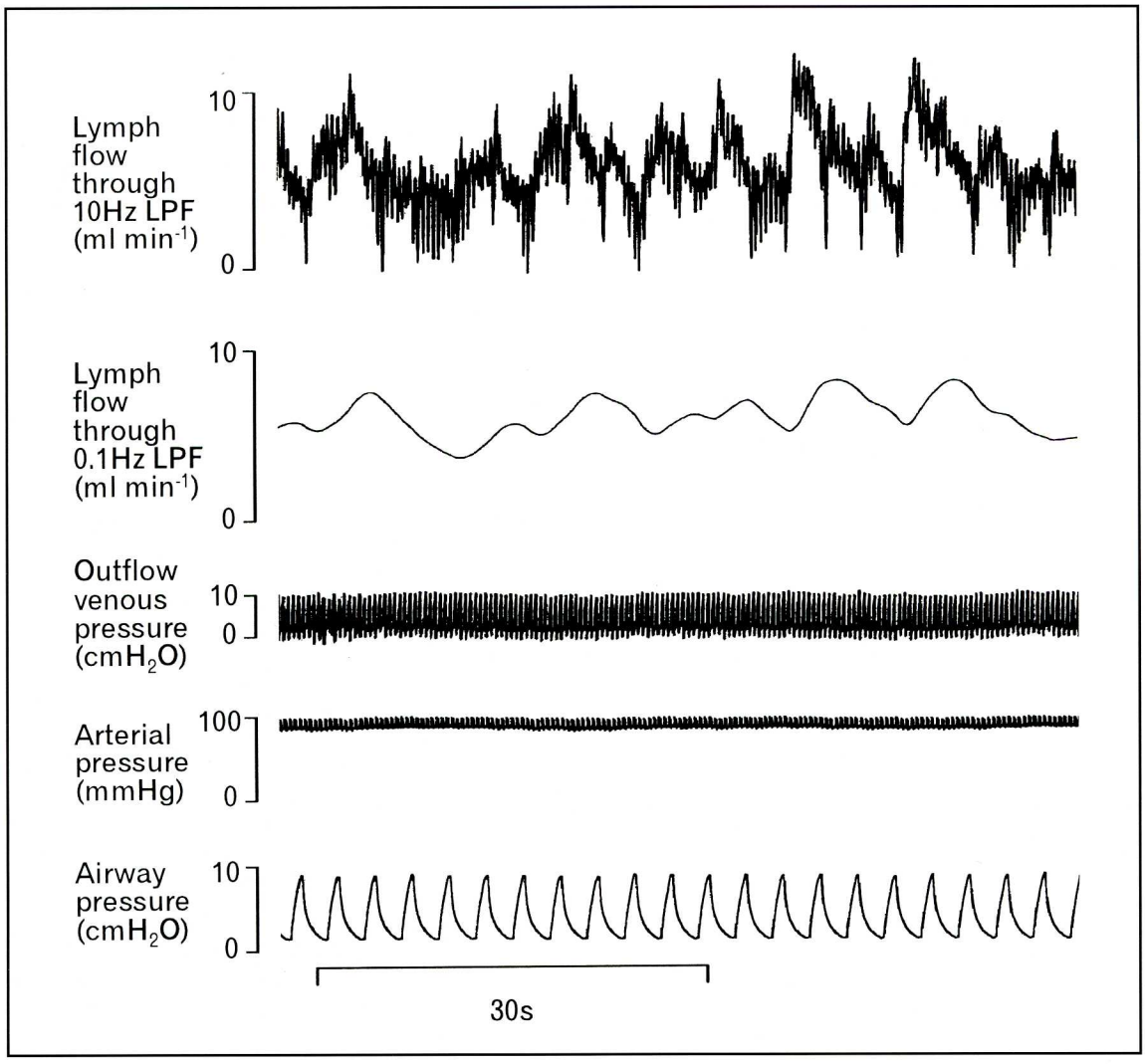


*Fig. 1. Recording from an anesthetized, mechanically ventilated sheep. Lymph flow of the thoracic duct was recorded through two low pass filters (LPF) of the flowmeter. Upper tracing of lymph flow (10 Hz LPF) is affected by cardiac pulsation and by airway pressure fluctuation. Lower tracing of lymph flow (0.1 Hz LPF) demonstrates low frequency pulsation. It is uncertain whether or not the waveform is simply a fusion of cardiac pulsation and respiratory fluctuation.*

paired *t* test was applied for comparisons. A value of $p<0.05$ (two-tailed) was accepted as statistically significant.

*RESULTS*

Lymph flow of the thoracic duct was recorded both through a 10 Hz LPF and a 0.1 Hz LPF on the polygraph *(Fig. 1)*. Lymph flow through a 10 Hz LPF showed cardiac pulsation (180 $min^{-1}$) and airway pressure fluctuations (24 $min^{-1}$) synchronously. Lymph flow through a 0.1 Hz LPF showed only a low frequency wave. The flow signal through each LPF was recorded on the computer hard disk sequentially during the same period for FFT analysis.

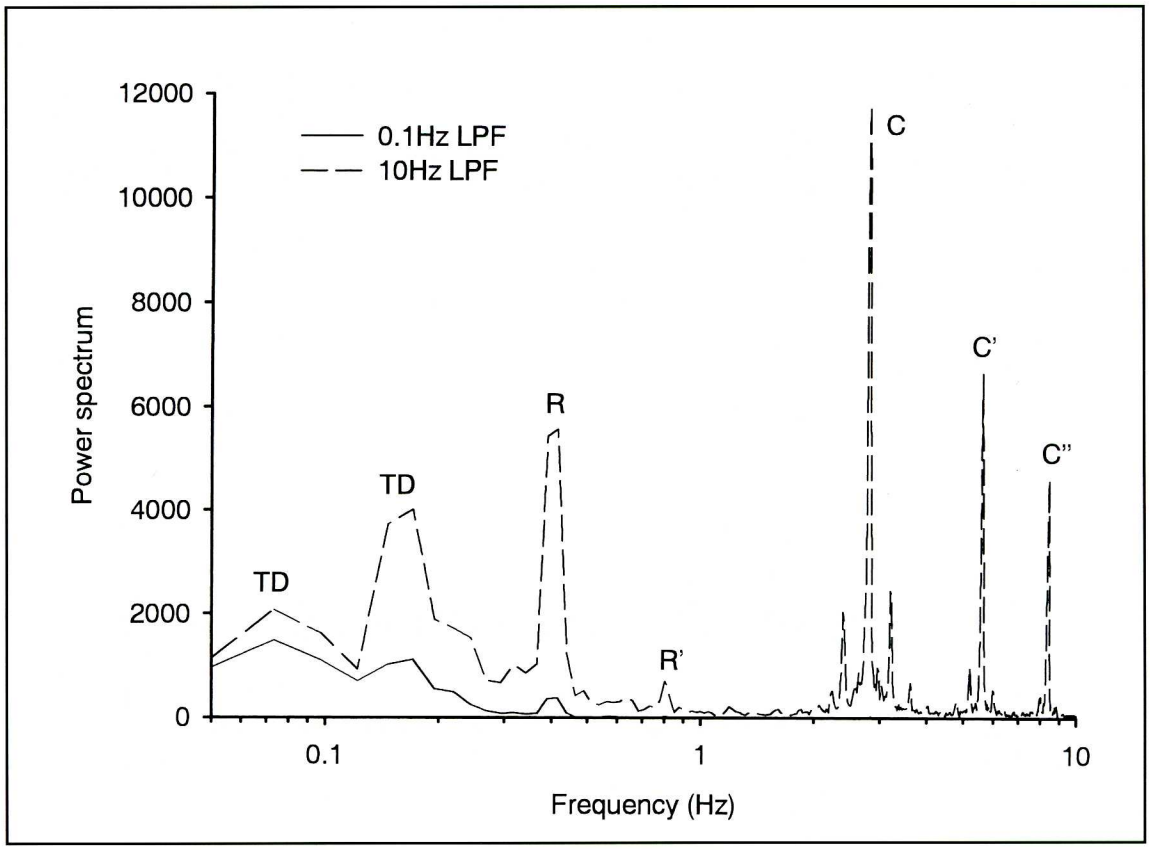


*Fig. 2. Power spectrums obtained by fast Fourier transform from lymph flow signals both through a 10 Hz low pass filter (LPF) and a 0.1 Hz LPF of the flowmeter during the baseline period in an experiment. The power spectrum from 10 Hz LPF (dotted line) contains cardiac pulsation (C) and its harmonics (C', C"), respiratory fluctuations (R) and its harmonics (R'), and low frequency components which signify an intrinsic spectrum of thoracic duct pulsation (TD). In the power spectrum through a 0.1 Hz LPF, only the component of thoracic duct pulsation appeared and the components of cardiac pulsation and respiratory fluctuation almost disappeared.*

*Fig. 2* shows the power spectrums obtained by FFT from recorded signals through a 10 Hz LPF and a 0.1 Hz LPF during the baseline period in one experiment. The power spectrum of the lymph flow wave through a 10 Hz LPF contains cardiac pulsation (2.8 Hz) and its harmonics, and airway pressure fluctuations (0.4 Hz) and its harmonics. Besides these spectrums, there were low frequency components which may signify intrinsic spectrums of thoracic duct pulsation. The pattern of the spectrums appeared in all sheep (n=10). In the power spectrums of flow signals through a 0.1 Hz LPF, low frequency components appeared, and they were of the same frequency which appeared through a 10 Hz LPF. The components of cardiac pulsation and respiratory movement almost disappeared in spectrums through a 0.1 Hz LPF.

The changes in the power spectrum of thoracic duct flow before and after the elevation of venous outflow pressure is demonstrated by the results of FFT in *Fig. 3*. Whereas the components of cardiac pulsation and respiratory movement did not change, the frequency of thoracic duct pulsation increased from 0.7 to 0.12 Hz during

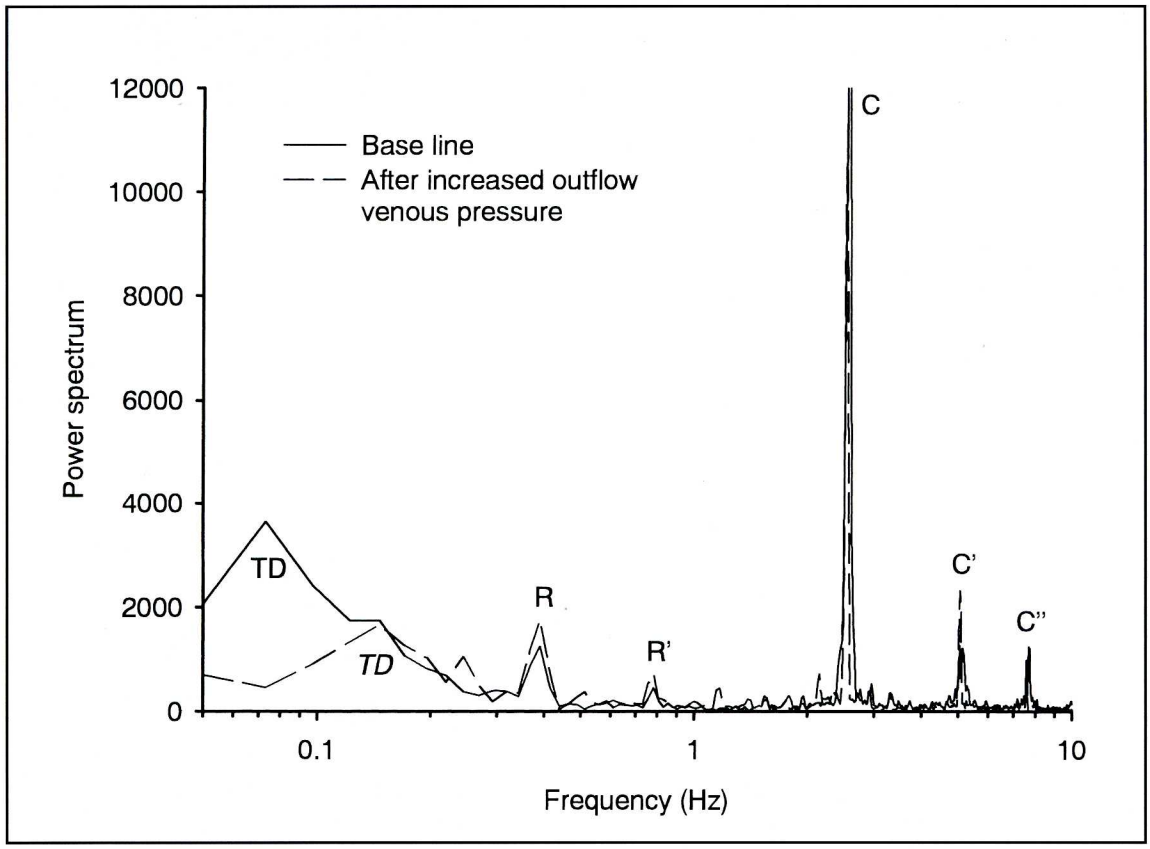


*Fig. 3. The effect of an increase in outflow venous pressure on thoracic duct pulsation. The power spectrum of thoracic duct pulsation (TD) shifted to the right (TD) (higher frequency). However, the components of cardiac pulsation (C) and its harmonics (C', C"), and respiratory fluctuation (R) and its harmonics (R') did not change.*

elevation of venous outflow pressure. Although there were several peaks which represent the spectrum originating from thoracic duct pulsation, the frequency of the highest peak was considered as the frequency of the thoracic duct pulsation at each period.

*Table 1* summarizes the data of the effect on measured parameters of an increase in venous outflow pressure (n=8). Venous pressure was increased from 6±3.5 to 17±9.5 cm $H_2O$ by partial constriction of the cranial vena cava. In response to this maneuver, thoracic duct lymph flow decreased significantly ($p<0.01$) and thoracic duct flow pulsation increased significantly ($p<0.01$). However, the frequency of cardiac pulsation and respiratory fluctuation did not change.

## *DISCUSSION*

Campbell and Heath (3) suggested the existence of thoracic duct pulsation after measuring the pressure in the thoracic duct of sheep. They reported that the pulsation frequency ranged from 2 to 20 $min^{-1}$. Hall, Morris and Woolley (4) also demonstrated pulsation of the lumbar lymph trunk. They observed an increase from 8 to 22 $min^{-1}$ in the frequency of pressure pulses when the outflow of the inserted catheter tip was raised to 68 cm above the head of the humerus. Both studies showed that major lymph duct pulsation exceeded 0.1 Hz. Previously, we demonstrated thoracic duct pulsation using an ultrasound transit-time flowmeter (1). We

**TABLE 1**
**Effect (Mean ± SD) on Measured Parameters of an Increase in Venous Outflow Pressure**

| | VP ($cm\ H_2O$) | LF ($ml\ min^{-1}$) | PF (Hz) | HR (Hz) | RR (Hz) |
|---|---|---|---|---|---|
| Baseline (6±3.5) | | 3.89±1.59 | 0.11±0.03 | 2.22±0.45 | 0.38±0.05 |
| Increased (17±9.5) | | 2.69±1.82* | 0.21±0.05* | 2.16±0.37 | 0.38±0.05 |

VP—venous outflow pressure; LF—thoracic duct lymph flow; PF—thoracic duct pulsation frequency obtained from signals through 10 Hz LPF by FFT; HR—heart rate; RR—respiratory rate
*significantly different from baseline value ($p<0.01$).

found that thoracic duct flow was regularly pulsatile and that the frequency was approximately 5 $min^{-1}$. In that study, we recorded the lymph flow through a 0.1 Hz LPF. When the pulsation frequency exceeded 0.1 Hz, flow variation was damped and appeared as a mean flow, and disappeared when venous outflow pressure was increased to more than 30 cm $H_2O$. Therefore, in the present study we recorded the thoracic duct lymph flow through a 10 Hz LPF. In the waveform through a 10 Hz LPF, cardiac pulsation and respiratory fluctuation appeared *(Fig. 1)*. Although both the pressure recordings (3,4) and our recording through a 0.1 Hz LPF (1) suggested the existence of thoracic duct pulsation, it was still uncertain whether the waveform was just a fusion of cardiac pulsation and respiratory fluctuation. To resolve this problem, we analyzed the flow signals through a 10 Hz LPF by FFT and obtained a power spectrum of the pulse frequency. We were able to demonstrate that thoracic duct lymph flow contains its own intrinsic pulsation appearing as a low frequency component independent of cardiac or respiratory fluctuation *(Fig. 2)*.

In our previous study (1) when the venous outflow pressure was increased, lymph flow decreased and pulsation frequency increased. With a further increase in venous outflow pressure, lymph flow decreased significantly together with an apparent decrease or disappearance of flow pulsation. Because we had recorded the flow signals through a 0.1 Hz LPF, there was the distinct possibility that a high frequency pulsation of more than 0.1 Hz was not able to be detected precisely at higher venous outflow pressures. Accordingly, we again tested the effect of elevating venous outflow pressure on thoracic duct pulsation but applied FFT analysis to flow signals through a 10 Hz LPF and a 0.1 Hz LPF. We confirmed that the pulsation frequency increased from 0.11 to 0.21 Hz when venous outflow pressure increased from 6 to 17 cm $H_2O$ *(Table 1)*. When venous outflow pressure increased, only the thoracic duct pulsation frequency increased and the frequencies of other components (cardiac and respiratory) did not change. These findings support that the thoracic duct pulsation is independent of cardiac pulsation and respiratory movement and is highly sensitive to a rise in venous outflow resistance.

In summary, we measured thoracic duct flow using an ultrasound transit-time flowmeter and confirmed by FFT analysis an intrinsic thoracic duct pulsation which increased in frequency when venous outflow pressure was raised.

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