Computerised analysis of auscultatory sounds associated with vascular patency of haemodialysis access

H. A. Mansy[1,2,3] S. J. Hoxie[1] N. H. Patel[4] R. H. Sandler[1,3]

[1]The Biomedical Acoustics Research Group, Department of Pediatrics, Rush Medical College, Chicago, USA
[2]Department of Mechanical, Materials & Aerospace Engineering, Illinois Institute of Technology, Chicago, USA
[3]Department of Bioengineering, University of Illinois at Chicago, USA
[4]Section of Interventional Radiology, Department of Diagnostic Radiology & Nuclear Medicine, Rush University Medical Center, Chicago, USA

Abstract—*Vascular access for renal dialysis is a lifeline for about 120 000 individuals in the United States. Stethoscope auscultation of vascular sounds has some utility in the assessment of access patency, yet can be highly skill-dependent. The objective of the study was to identify acoustic parameters that are related to changes in vascular access patency. The underlying hypothesis is that stenoses of haemodialysis access vessels or grafts cause vascular sound changes that are detectable using computerised data acquisition and analysis. Eleven patients participated in the study. Their vascular sounds were recorded before and after angiography, which was accompanied by angioplasty in most patients. The sounds were acquired using two electronic stethoscopes and then digitised and analysed on a personal computer. Vessel stenosis changes were found to be associated with changes in acoustic amplitude and/or spectral energy distribution. Certain acoustic parameters correlated well (correlation coefficient = 0.98, $p < 0.0001$) with the change in the degree of stenosis, suggesting that stenosis severity may be predictable from these parameters. Parameters also appeared to be sensitive to modest diameter changes (>20%), ($p<0.005$, Wilcoxon rank sum test). These results suggest that computerised analysis of vascular sounds may be useful in vessel patency surveillance. Further testing using longitudinal studies may be warranted.*

Keywords—*Haemodialysis access stenosis or occlusion, Haemodialysis graft, conduit or fistula, Vascular sounds, Auscultation, Computerised analysis, Digital signal processing*

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1 Introduction

1.1 *Vascular access for haemodialysis*

THERE ARE approximately 120 000 people in the United States with chronic renal failure (HODGES *et al.*, 1997). Without some form of dialysis, most would rapidly succumb to their disease. Haemodialysis is the most frequently used technology, but problems with the necessary vascular access are common. Either an arteriovenous (AV) fistula or a graft typically provides long-term vascular access for haemodialysis patients. The former is fashioned by direct anastomosis of a vein to an artery (usually forearm vessels), and the latter is created through conduit interposition. Bypass of the high-resistance vessels (arterioles and capillaries) facilitates the high flow rates necessary for efficient haemodialysis.

The principal proximate cause of access failure is thrombosis, but the underlying cause is usually progressive venous outflow stenosis (BESARAB *et al.*, 1995; PALDER *et al.*, 1985; ETHEREDGE *et al.*, 1983; BEATHARD, 1995; BURGER *et al.*, 1990; WINDUS *et al.*, 1990). A comprehensive review by the National Kidney Foundation-Dialysis Outcomes Quality Initiative (NATIONAL KIDNEY FOUNDATION 1997) concluded that: over 90% of graft loss is caused acutely by thrombotic events; over 90% of thrombotic events occur within stenotic vessels; and clinically significant compromise of blood flow and thrombosis rarely occur in vessels with $\leq$50% stenosis. Furthermore, graft salvage is much more likely if balloon angioplasty is performed before, rather than after, thrombosis has occurred (78.9% against 40% patency after three months) (NATIONAL KIDNEY FOUNDATION 1997; BEATHARD, 1995; KATZ and KOHL, 1995). Therefore early

Correspondence should be addressed to Dr Hansen A. Mansy; email: hmansy@rush.edu

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detection and treatment of the stenosed vessels are expected to increase access longevity.

1.2 *Current methods for monitoring patency*

Aside from physical examination, longitudinal monitoring of access flow rates is currently the least expensive method for detection of significant stenosis (BEATHARD, 1995). Limitations of the latter method include the need to standardise tubing, needle size and haemodialysis machines (BURGER *et al.*, 1990). Duplex Doppler ultrasonography, various dilution techniques, magnetic resonance imaging, contrast venography and other approaches have also been used. Although useful, limitations include expense, availability and observer skill requirements (NATIONAL KIDNEY FOUNDATION 1997).

1.3 *Previous acoustic studies of vascular stenosis*

Several acoustic studies have investigated the acoustic characteristics of occluded blood vessels. A common finding of these studies (TOBIN, 1976; FREDBERG, 1977; KIRKEEIDE *et al.*, 1977; WANG *et al.*, 1990; SEMMLOW *et al.*, 1991; KUROKAWA *et al.*, 1994) was that sound intensity tends to increase with stenosis, especially at high frequencies and at certain locations. The source of this acoustic energy is believed to be associated with turbulence produced by partially occluded blood vessels (FREDBERG, 1977; WANG *et al.*, 1990; AKAY *et al.*, 1994). These high-amplitude sounds occur slightly downstream of the stenosis region and tend to decay within a few centimetres (TOBIN *et al.*, 1976; FREDBERG, 1977). In addition, sounds associated with coronary stenoses were more detectable during diastole (WANG *et al.*, 1990; SEMMLOW *et al.*, 1991, AKAY *et al.*, 1992).

1.4 *Objectives and achievements*

The objective of this study was to identify acoustic characteristics that could be indicative of changes in vessel patency using computerised analysis of vascular sounds. This could foster development of new technologies for the diagnosis and monitoring of vascular stenosis. The underlying hypothesis was that stenoses of haemodialysis access cause detectable vascular sound changes. Achievements of the study include

(a) presenting new data demonstrating the effects that dialysis vascular access stenosis may have on vascular sounds
(b) Identifying some acoustic parameters that correlate well with the severity of vascular access stenosis
(c) Determining optimum frequency bands that could maximise the utility of the acoustic parameters
(d) Investigating initial stethoscope placement options and finding that a small number of auscultation sites, which are not necessarily located directly at the stenosis site, may be sufficient for the current approach.

2 Methods

2.1 *Data gathering*

Experiments were conducted in the Department of Diagnostic Radiology & Nuclear Medicine at Rush University Medical Center, after approval of the Human Institutional Review Board. Subjects were recruited from those referred for angiography either for routine three-month surveillance or for access dysfunction noted at recent dialysis sessions. Patients underwent the angiography and, if needed, endovascular interventions as part of their routine clinical care. Patients were not subjected to any additional radiation or intervention for the purposes of the study. Subjects who could not co-operate with the acoustic examination or who had a physical limitation (e.g., wound infection, dressing barrier) preventing adequate sensor placement were excluded from the study.

After informed consent had been obtained, and the location and configuration of the haemodialysis access (graft or arteriovenous fistula) had been determined, its patency and any dysfunction at recent dialysis sessions were noted. A skin marker was used to mark chosen sensor sites to ensure reproducible pre- and post- angiography/angioplasty sensor positioning. Two electronic stethoscopes* were placed over the field of the haemodialysis circuit. For grafts, one sensor was placed on the venous limb, and the other was placed on the arterial limb of the graft. For arteriovenous fistulae, sensors were placed 5 cm apart over the outflow vein.

Acoustic measurements were acquired for 15 s on a digital multitrack minidisk recorder,† while patients rested comfortably in the supine position. Sensors were then removed, and the patient's arm was prepped and draped in the usual sterile fashion.

After completion of angiography (with or without endovascular intervention, e.g. by angioplasty), the two acoustic measurements were again repeated. The presence and location of any stenotic lesion(s) were noted. Also, if performed, the type of endovascular intervention used to treat the significant stenotic lesion(s) and its angiographic outcome were recorded. The diameter of the stenotic lesion(s) was measured from the angiograms both before and after endovascular intervention using a Vernier calliper (0.02 mm resolution). For cases without intervention, the diameter was assumed invariant. The percent diameter change (PDC) with angioplasty was calculated as the diameter increase divided by the native vessel diameter.

For the purposes of this study, 'small' PDC cases were taken as those with PDC $\leq 20\%$, and 'large' PDC cases were taken as those with PDC $>20\%$. The 20% cutoff value was chosen because the data later showed that acoustic differences started to be detectable around this PDC value. Therefore this cutoff choice was indicative of the level that could separate detectable and undetectable PDC changes using the current acoustic method. In other words, small PDC cases were those with undetectable acoustic signatures, and large PDC cases were those with propably detectable acoustic changes.

2.2 *Distances between sensors and stenosis*

Stenosis sites were not known prior to angiography, and sensors were placed with respect to other anatomical landmarks (as described in Section 2.1). This approach was preferred, as it entailed standardising sensor locations while minimising interference with patient care and reducing the need for expert placement of sensors. This also helped avoid the complexity of carrying out acoustic recordings at a large number of sensor locations in the current pilot study. Furthermore, this approach would be helpful in showing that acoustic changes may be detectable at locations beyond the immediate neighbourhood of the stenosis.

Consequently, the distances between the stenosis and acoustic measurements sites (mean = 7.5 cm, range = 0–9 cm) and the inter-sensor distances (mean = 5.5, range = 4–8 cm) varied considerably in the current study. The details of these distances are listed in Table 1. The shunt location (seven of the 11 cases were upper arm, and the rest were forearm) and configuration (one was a fistula, and ten were grafts), as well as the stenosis geometry, varied significantly. These variations may have made it impossible to show trends associated with

*Electromax, Labtron, Graham-Field, Hauppage, NY, USA
†Model MD 8, Yamaha, Japan

Table 1 Distance of Sensor upstream from stenosis (cm) and shunt type

Subject	a	b	c	d	e	f	g	h	i	j	k
Sensor 1	7	1	11	−1	5	10	5	8	10	10	NA
Sensor 2	2	−6	4	−5	0	15	1	2	2	6	NA
Shunt type	graft	fistula	graft	graft	graft	graft	graft	graft	graft	graft	graft

specific geometries, especially given the small total number of subjects in this pilot study.

2.3 *Data analysis*

Custom software was developed in Matlab[‡] to digitise and analyse the acoustic signals on a personal computer[**] equipped with an analogue-to-digital card[††]. The software implemented the following steps:

(i) digitise the acoustic signals from both electronic stethoscopes at 8192 Hz
(ii) calculate the auto spectrum of each 250 ms segment of the digital data (120 segments with 50% overlap, Hanning window) using an FFT (corresponding to a 4 Hz frequency resolution)
(iii) calculate the mean power spectral density (in decibels) by averaging results from all data segments
(iv) calculate the difference between the pre- and post-angioplasty spectra (in decibels) for a pre-determined frequency band (e.g. 0–1000 Hz)
(v) calculate the mean (MEAN) of the spectral difference
(vi) calculate the root-mean-square (RMS) of the spectral difference (after subtracting the mean difference)
(vii) calculate the true RMS (TRMS) of the spectral difference as the RMS value without subtracting the mean
(viii) Repeat the data processing procedure for the second sensor location
(ix) Calculate the maximum of the MEAN, RMS and TRMS as the larger of the two corresponding values from the two sensors.

2.4 *Statistical analysis*

The Wilcoxon rank sum test was used to compare the values of the acoustic parameters for large (>20%) and small PDC cases. A p-value <0.05 was taken to indicate that differences between the values corresponding to the two groups were statistically significant. The correlation coefficient between the PDC and the spectral parameters was calculated to evaluate the association between these two parameters. Here, a correlation coefficient >0.90 was taken to indicate strong association, given that the relationship is statistically significant ($p < 0.05$).

3 Results

Eleven patients (three males) of mean age 60 were recruited for this study over a 3 month period. Pairs of pre- and post-angiography spectra (solid and broken lines, respectively) are shown in Figs 1 *a–k* for all study subjects. The PDC is also shown for each patient. Subjects are arranged such that the PDC continuously decreases from Fig. 1*a* to Fig. 1*k*. The data suggest several trends. First, the spectral distributions showed wide intersubject variability, which may be owing to the broad anatomical differences between subjects. Secondly, the acoustic energy tended to decrease with increasing frequency, which is a common feature of vascular and other biological sounds (TOBIN *et al.*, 1976; FREDBERG, 1977; WANG *et al.*, 1990; AKAY *et al.*, 1994; PASTERKAMP *et al.*, 1995; MANSY *et al.*, 2002). Thirdly, there were noticeable differences between each spectral pair, even when no angioplasty was performed (Fig. 1*k*). Finally, greater spectral differences seemed to take place with larger diameter changes.

To describe spectral differences using a few parameters, the MEAN, RMS, and TRMS of spectral differences (in the 0–1000 Hz band) were calculated. These three parameters are shown as functions of the PDC in Fig. 2–4, respectively. It can be observed from these Figures that all three parameters seem to increase with PDC increase.

4 Discussion

4.1 *Characteristic spectral changes*

The spectral differences between the pre- and post-angioplasty states (Fig. 1) were clear, especially for cases with relatively large (>20%) changes in vessel diameter. In the latter group, differences as large as >10 dB can be appreciated. Although the spectral changes did not follow a single trend, they can be broadly placed under one or both of two different categories. The first category is a general sound amplitude change, and the second is a redistribution of acoustic energy among different frequencies. In certain patients, one of these types may be more prominent than the other. For example, Fig. 1*f* shows a case of general amplitude change (first type) dominance. Here, spectral energies changed relatively uniformly over the entire frequency range. Conversely, Fig. 1*e* shows little change (<1.5 dB) in mean signal amplitude, with significant energy redistribution among frequencies (seen as an increase in the high frequencies and a decrease in low frequencies). The majority of the subjects (four of six) with large PDC, however, showed simultaneous general amplitude change and energy redistribution. To quantify both types of changes the MEAN and RMS of the spectral differences (in the 0–1000 Hz band) were calculated and are shown as functions of PDC in Figs 2 and 3, respectively.

It is clear from Fig. 2 that the absolute value of the MEAN spectral difference tended to increase as the PDC increased. The Figure also shows that, for the large PDC cases (PDC > 20%, $n = 6$), the MEAN spectral difference was positive ($n = 4$), negative ($n = 1$) or close to zero (−0.4 dB, $n = 1$). This result is interesting, as it suggests that, in the majority of cases (four out of six), increased vessel stenosis may result in quieter sounds ($f = 0$–1000 Hz).

These sound amplitude changes (described here by the MEAN spectral difference) can be understood based on possible flow alterations owing to local stenoses. More specifically, a local stenosis is expected to increase vessel resistance and reduce blood flow velocity through the graft or fistula (BOSMAN *et al.*, 1997; TESSITORE *et al.*, 2003) (except possibly at the stenosis site), which tends to reduce vascular sounds. On the other hand, a high-speed jet can form at the localised stenosis site (ASK *et al.*, 1995), which tends to increase local

[‡]Version 6.5, The Mathworks, Natick, MA, USA
[**]Inspiron 8200, Dell, Austin, TX, USA
[††]Card 6062-E, National Instruments, Austin, TX, USA

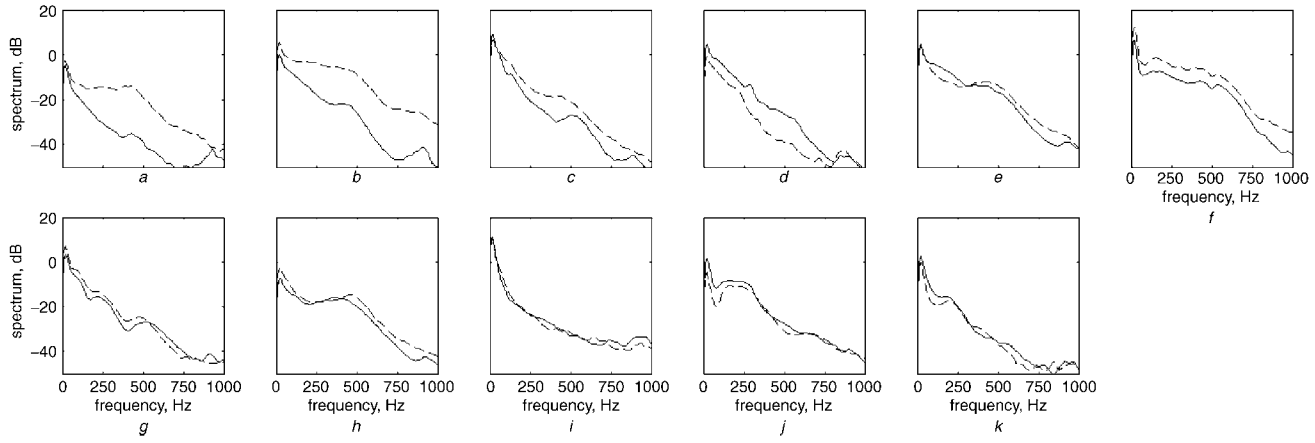

Fig. 1 *Acoustic spectra for (–) pre- and (– – –) post-angioplasty for all subjects. PDC for each case is as follows: (a) 48%; (b) 42%; (c) 30%; (d) 29%; (e) 23%; (f) 22%; (g) 18%; (h) 15%; (i) 14%; (j) 5%; (k) 0%. Although there was wide inter-subject variability, differences between each spectral pair were greater when diameter changes were larger*

turbulent vascular flow sounds (TOBIN *et al.*, 1976; FREDBERG, 1977). These two competing effects (increased sound amplitude near the stenosis and decreased sounds elsewhere) can balance at an intermediate spatial location, leading to minimum amplitude changes, masking the effects of diameter change. To avoid this pitfall, the acquisition of signals over several locations would be helpful. Given that the sound amplitude tended to decrease with increasing stenosis (in four out of the six large PDC cases), it can be concluded that the effects of blood flow reduction were more dominant in the current study.

A common finding of previous studies (TOBIN *et al.*, 1976; FREDBERG, 1977; KIRKEEIDE, 1977; WANG *et al.*, 1990; SEMMLOW *et al.*, 1991; KUROKAWA *et al.*, 1994) was that sound intensity tends to increase with stenosis, especially at high frequencies and at certain locations. These high-amplitude sounds occur slightly downstream from the stenosis region and tend to decay within a few centimetres (TOBIN *et al.*, 1976; FREDBERG, 1977). Most of the spectral distributions of Fig. 1 demonstrated the effect of decreased flow velocity due to the stenosis rather than the increase in high-frequency sounds. This may be primarily owing to the fact that recording sites used in the current study (Table 1) were usually not close enough to the high acoustic generation location that may exist downstream from the stenosis site.

There were two subjects (29 and 23% PDC cases) where one of the data acquisition sites was right at the stenosis location. Here, the sensor nearest to the stenosis showed increased amplitude with stenosis (Fig. 1*d*), whereas the far sensor (located about 5 cm upstream) showed little amplitude variation (Fig. 1*e*). This is consistent with the expected spatial variations of vascular sounds discussed above.

4.2 *Acoustic parameters as indicators of vessel stenosis*

The data in Figs 2–4 showed that a larger PDC gave rise to larger MEAN (absolute value), RMS and TRMS values. The correlation coefficient was calculated between these parameters and PDC for PDC > 20%. The coefficients were 0.92, 0.90 and 0.95 ($p < 0.0001$) for the data of Figs 2–4, respectively. The slightly higher coefficient for TRMS suggests that the TRMS parameter may have a slightly better association with PDC (compared with MEAN and RMS), while combining both amplitude and energy redistribution changes into a single parameter. In addition, regression analysis showed

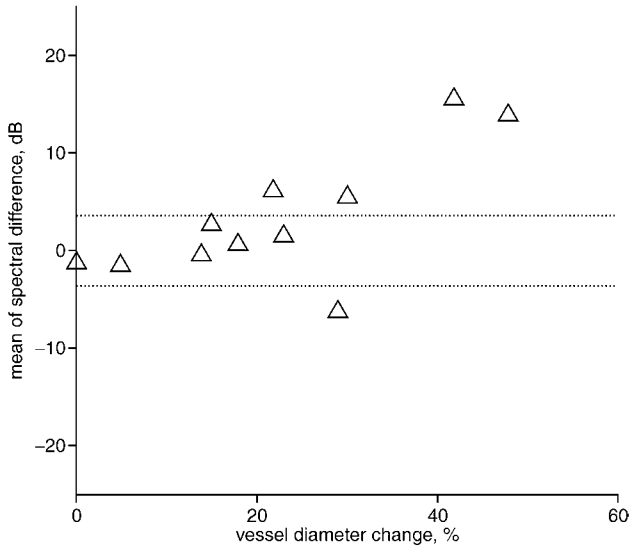

Fig. 2 *MEAN of spectral difference (in 0–1000 Hz band) as function of vessel diameter change*

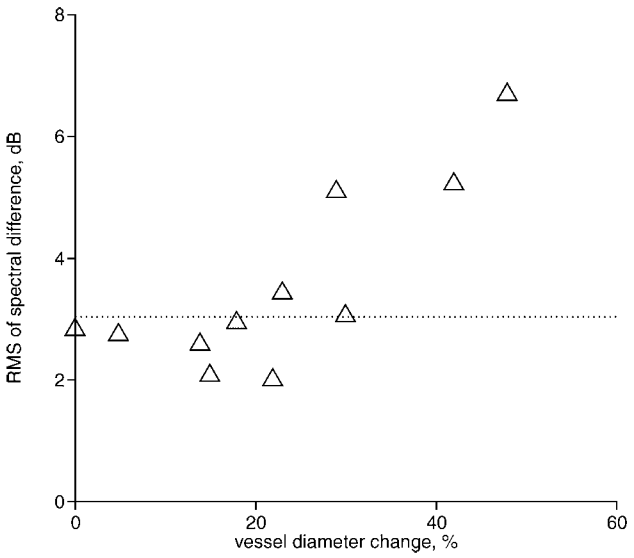

Fig. 3 *RMS of spectral difference (after subtraction of mean difference, 0–1000 Hz) as function of vessel diameter change*

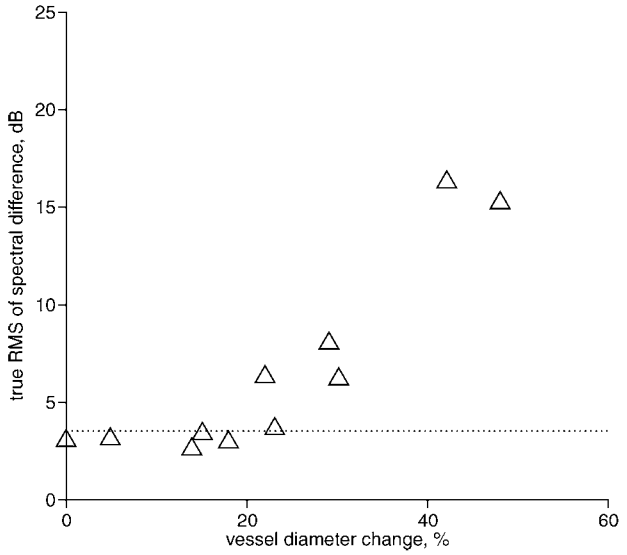

Fig. 4 *TRMS of spectral difference (without subtraction of mean difference, 0–1000 Hz) as function of vessel diameter change*

that the PDC values (PDC > 20%) can be estimated from TRMS as: PDC = 2.3 × TRMS + 10. Note that TRMS is not independent of MEAN and RMS, as it equals the square root of the sum of the squares of the MEAN and RMS values.

It can also be seen in Fig. 2 that MEAN values for large and small PDC cases overlap. This overlap was assessed in the current study by calculating an inter-group distance as the difference between the lowest parameter value for the large PDC cases and the highest value for the small PDC cases. A positive distance indicates no overlap, and a negative distance is the amount of overlap. Inter-group distances were −1.4, −0.9 and 0.3 dB in Figs 2–4, respectively. This indicates overlap in the MEAN and RMS and no overlap in the TRMS values.

Furthermore, thresholds that can be used to separate large and small PDC cases are shown in Figs 2–4. It can be seen that one of the large PDC (>20%) cases (PDC = 23%) would be misclassified as a small PDC using the MEAN parameter (Fig. 2), but would be correctly classified using the RMS (Fig. 3). The RMS, on the other hand, would misclassify the 22% PDC case, whereas the MEAN correctly classified this case. These misclassifications can be expected, given the morphology of the spectra of Figs 1*e* and *f*.

These data also suggest that by using either the MEAN or RMS parameters (0–1000 Hz), cases with PDC > 25–30% can still be separable from lower PDC cases, and combining decisions from both parameters (i.e. if either MEAN or RMS is high, indicating that PDC is high) may enable separation for PDC > 20%. In addition, complete separation of large PDC cases (>20%) was possible using the TRMS parameter. Here, both the previous misclassified cases (PDC = 22 and 23% of Figs 1*e* and *f*) were correctly classified ($p < 0.005$, Wilcoxon rank sum test). Although this is consistent with the absence of inter-group overlap, inter-group distance seemed small (0.3 dB), as demonstrated by the several data points that are close to the threshold. The MEAN and RMS differences between the large and small PDC groups were significant and marginal, respectively ($p = 0.009$, 0.08, respectively, Wilcoxon rank sum test).

Therefore it can be concluded that auscultation at a single sensor location may be able completely to separate large (PDC > 20%) and small PDC cases, given that TRMS or both MEAN and RMS (in 0–1000 Hz) are used.

4.3 *Number of sound acquisition locations*

Analysis was repeated for data collected at the second sensor location, and values of the acoustic parameters were combined with those of the first sensor by the choice of the 'largest deviation value' (i.e. choice of the larger of the two sensor values to represent each parameter). Combined parameter values from the two sensors resulted in correlation coefficients of 0.95, 0.93, 0.97, for the MEAN, RMS and TRMS, respectively ($p < 0.0001$). Now the relationship between PDC and TRMS can be represented by: PDC = 2.3 × TRMS + 10. The larger correlation values (compared with those of one sensor location of Figs 2–4) suggest that using two sensor locations is more likely to yield better estimates of the PDC from the values of the MEAN, RMS or TRMS spectral deviations.

The inter-group distances also improved to 0.5, −0.7, and 1.0 dB for the MEAN, RMS and TRMS, respectively (compared with −1.4, −0.9, 0.3 dB in Figs 2–4, respectively). Because of the improved inter-group distances, it can be concluded that better separation between large and small PDC cases was possible when two sensor locations were used. In that case, there were no misclassifications or group overlap, except for the RMS, where the 22% PDC case was still misclassified. The RMS values had less overlap but were still marginally different for the large (PDC > 20%) and small PDC cases ($p < 0.08$, Wilcoxon rank sum test). However, the MEAN and TRMS values were significantly different for the two groups ($p < 0.005$ for each, Wilcoxon rank sum test), with greater inter-group distances (compared with the single sensor values). Therefore stronger discrimination between the two groups can be expected when two sensors are used. It is worthwhile noting, however, that, for most patients, the changes in the acoustic spectra were similar at the two sensor locations.

Note that, although this study has focused on detecting the presence and severity of stenosis using a small number of sensors, determining the stenosis location using an auscultation approach may also be possible using sensor arrays with a larger number sensors (OWSLEY, 1998). That approach, however, is beyond the scope of the current study.

4.4 *Optimum frequency bands*

One objective of the study was to identify acoustic parameters that may be indicative of vessel diameter changes. As the values of the three acoustic parameters identified (namely, MEAN, RMS and TRMS of the spectral differences) depend on the frequency range of analysis (initially 0–1000 Hz), the correlation coefficients between PDC and these parameters are also frequency-dependent. To find the strongest correlation, the coefficient values were calculated for all possible frequency-band choices. The optimum band was then taken as the band with the highest coefficient values. Fig. 5*a* shows the correlation coefficient between PDC and TRMS as a function of frequency band start and bandwidth. This Figure shows that the coefficient has high values for a wide range of frequencies. The optimum frequency band is around 300–600 Hz, where the coefficient reached a value of 0.98 ($p < 0.0001$). Using this frequency band, PDC can be estimated from: PDC = 1.8 × TRMS + 10. Fig. 5*a* also suggests that low frequencies were associated with very low correlation coefficients. For example, the dip in the correlation at low frequencies suggests that the low-pass cutoff needs to be greater than about 100–200 Hz to maintain high correlations.

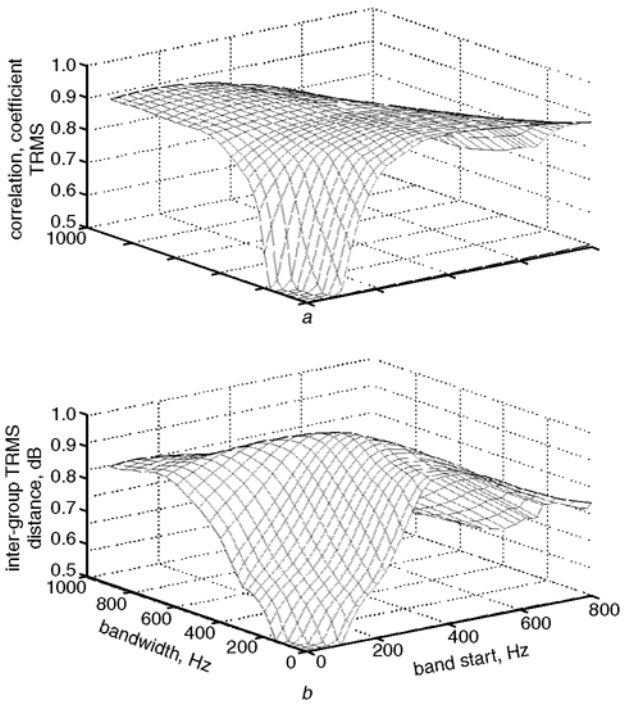

Fig. 5 *(a) Correlation coefficient between TRMS and PDC as function of frequency band choice. (b) Effect of choice of frequency band on ability to separate small and large vascular diameter alteration*

A second frequency band optimisation was implemented in this study, where the optimum band criterion was maximising separation between small and large PDC groups. Here, inter-group TRMS distance was calculated for all possible frequency bands. Results are shown in Fig. 5*b*. Here, positive values are indicative of complete separation, and negative values indicate group overlap. As seen in the Figure, the optimum frequency band (that with maximum TRMS group separation) was also found to be about 300–600 Hz.

The MEAN, RMS and TRMS values (the larger of the two sensor locations) for the optimum frequency band (300–600 Hz) are plotted in Figs 6–8, respectively. Here, the TRMS group separation has become 3.6 dB (for 300–600 Hz), compared with 1.0 dB (for the 0–1000 Hz band).

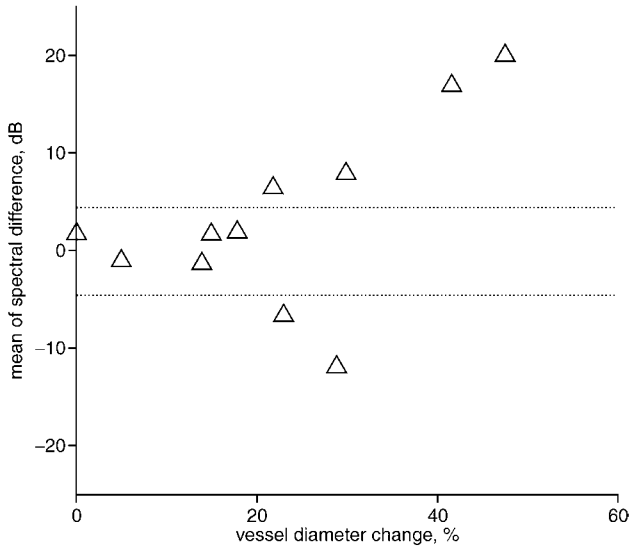

Fig. 6 *Mean of spectral difference (in 250–550 Hz band) as function of vessel diameter change*

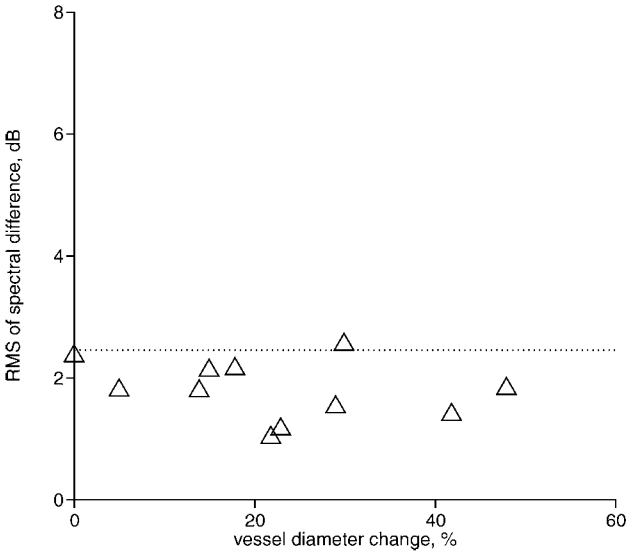

Fig. 7 *RMS of spectral difference (after subtraction of mean difference) as function of vessel diameter change*

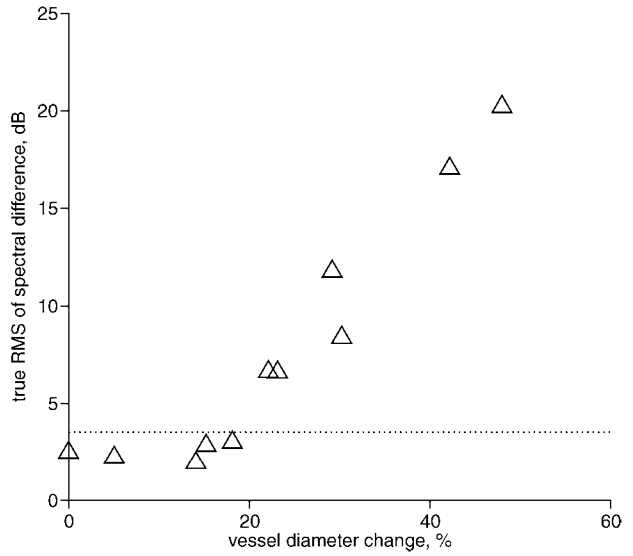

Fig. 8 *RMS of the spectral difference (without subtraction of mean difference) as function of vessel diameter change*

It can also be seen that the RMS parameter (Fig. 7) has lost its discrimination ability, as most of the large PCD cases are now misclassified. In addition, the correlation between PDC and RMS is very poor (correlation coefficient = 0.04). Further frequency band optimisations for the RMS parameter showed that broad frequency bands (width > 600 Hz) would be needed to maintain high correlation and relatively high separation ability for RMS. These results indicate that energy redistributions due to stenosis occur over broad frequency bands, whereas narrower bands primarily show amplitude changes.

5 Conclusions

The central hypothesis in the current study was that haemodialysis access stenoses cause detectable vascular sound changes. The purpose of the study was to test this hypothesis and identify acoustic parameters that are indicative of

changes in vascular access patency. Results suggested that stenoses were associated with changes in the acoustic amplitude and/or spectral energy distribution. Results also suggested that even modest diameter changes (>20%) were detectable ($p < 0.005$, Wilcoxon rank sum test) using one surface sensor. Using two sensors (or two sensor locations) provided better results, and sensors did not necessarily need to be placed in the immediate neighbourhood of the stenotic region.

The study identified certain acoustic parameters that correlated well ($r = 0.98, p < 0.0001$) with the PDC due to stenosis, suggesting that the degree of stenosis change can be estimated from measured acoustic parameters (e.g. PDC can be calculated from: $PDC = 1.8 \times TRMS + 10$). It can therefore be concluded that computerised auscultation of vascular sounds may be worthy of future studies to further assess its utility in vascular patency surveillance.

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Authors' biographies

HANSEN A MANSY received his PhD in aerospace engineering with a focus on oscillating fluid flows from Illinois Institute of Technology. He is the associate director of the Biomedical Acoustics Research Center at Rush Medical College, and is a Research Associate Professor at the Illinois Institute of Technology.

SILAS J HOXIE received his Bachelor of Arts in physics from Dartmouth College. He is currently working towards a Master's degree in biology at New York University.

NILESH H PATEL completed medical school at State University of New York at Syracuse and a radiology residency at Michigan State University. He completed his subspecialty fellowship in interventional radiology at Henry Ford Hospital. His is the Director of Interventional Radiology at Rush University Medical Center.

RICHARD H SANDLER completed medical school and a pediatrics residency at Michigan State University and a subspecialty fellowship in Pediatric Gastroenterology at Harvard Medical School. He is the director of the Biomedical Acoustics Research Center and the Section of Pediatric Gastroenterology at Rush Medical College.

A primary research focus of the authors is developing novel medical diagnostic tools using audible-frequency acoustics.