“Vascular Age” Is Advanced in Children With Atherosclerosis-Promoting Risk Factors

Joseph Le; Danna Zhang, MS; Spencer Menees; Jie Chen, PhD; Geetha Raghuveer, MD, MPH

Background—Obesity and familial dyslipidemia in children are associated with accelerated atherosclerosis by pathological examination. We sought to determine whether these children had increased carotid artery intima-media thickness (CIMT), a measure of subclinical atherosclerosis similar to 45-year-old adults. Adult CIMT percentile tables were used for comparison because normative CIMT data for children are limited.

Methods and Results—Seventy children, ages 6 to 19 years, with obesity- and atherosclerosis-promoting risk factors such as dyslipidemia, hypertension, insulin resistance, and tobacco smoke exposure, or with familial dyslipidemia, underwent carotid artery ultrasound. Advanced “vascular age” (VA) was defined as having maximum CIMT that was ≥25th percentile for race- and sex-matched 45-year-old adults. Mean age was 13.0±3.3 years. Forty (57%) of 70 children had body mass index ≥95th percentile for age and sex. Maximum CIMT for obese children was 0.53±0.05 mm and for familial dyslipidemic children was 0.52±0.04 mm. Advanced VA was seen in 30 (75%) of obese children and 22 (73%) of familial dyslipidemic children. Thirty (75%) of obese children had ≥3 mutable atherosclerosis-promoting risk factors; these children had a nonsignificantly higher maximum CIMT compared with obese children with ≤3 risk factors (0.54±0.06 mm versus 0.52±0.03 mm, P=0.07). Obese children with high fasting triglyceride levels were more likely to have advanced VA.

Conclusions—VA is advanced and comparable in obese children with atherosclerosis-promoting risk factors and in children with familial dyslipidemia. Advanced VA is prevalent in obese children with high fasting triglyceride levels. (Circ Cardiovasc Imaging. 2010;3:8-14.)

Key Words: carotid arteries ■ cholesterol ■ obesity ■ pediatrics ■ vasculature

Clinical Perspective on p 14

Autopsies done on apparently healthy, young adult casualties of the Korean and Vietnam wars demonstrated a 45% to 77% prevalence of atherosclerosis of the coronary arteries, suggesting its childhood origins.11,12 These observations were made more than 50 years ago, when the prevalence of childhood obesity was much lower than it is at present. In 1990, the investigators from the Pathobiological Determinants of Atherosclerosis in Youth research group reported the link between atherosclerosis and risk factors such as dyslipidemia, tobacco smoke exposure, hypertension, diabetes, and age.13 A follow-up investigation by this group suggested a link between obesity, especially abdominal obesity and coronary artery lesions in youth.14 In addition, the Bogalusa study follow-up has confirmed that the atherosclerotic lesions in the coronary arteries are more prevalent in youth with multiple risk factors15 and that the effects of childhood obesity on the adult vasculature are cumulative.16,17

Carotid artery intima-media thickness (CIMT) can be used as a noninvasive tool for assessment of subclinical atherosclerosis as the disease process is asymptomatic in children. Increased CIMT has been associated with cardiovascular risk factors,18 coronary artery disease,19 stroke,20 and progression of coronary atherosclerosis.19,21–24 Pediatric epidemiological studies have shown that high total cholesterol, BMI, and LDL cholesterol in childhood25 are associated with an increased CIMT in adulthood. Case-control and observational studies in children have confirmed increased CIMT in the presence of risk factors such as hypertension, dyslipidemia, diabetes mellitus, and obesity.27–30

In our clinic, we evaluated 2 groups of high-risk children: (1) children with familial dyslipidemia who typically have...
elevations in their total and LDL cholesterol levels and are known to develop premature cardiovascular disease as young adults and (2) children with obesity and multiple atherosclerosis-promoting risk factors. We examined these 2 groups of high-risk children to determine whether they had developed comparable vascular changes at a young age. Because availability of normative CIMT data for children is limited, we compared their distribution of CIMT with normative data for 45-year-old adults to gauge progression of the disease process. Because CIMT is age dependent, comparing a child’s CIMT against the percentile charts available for a race- and sex-matched adult population may be a useful concept to quantify subclinical atherosclerosis. Such assessments of “vascular age” (VA) have been shown to be a useful tool in accurately reclassifying risk in adults.

Methods

Approval from Children’s Mercy Hospital Institutional Review Board was obtained before data collection. This study involved a retrospective chart review of 70 children ages 6 to 19 years who visited the Children’s Mercy Hospital Preventive Cardiology Clinic over a 1-year period (January 1, 2007, to December 31, 2007). These children were referred for evaluation of obesity with dyslipidemia and other atherosclerosis-promoting risk factors or familial dyslipidemia. Because of the retrospective nature of the study, the requirement for individual child assent/consent was waived.

Family history of premature cardiovascular disease was defined as death, myocardial infarction, or angina due to coronary artery disease or stroke at ≤55 years of age in a first-degree relative. History of exposure to tobacco smoke was ascertained via a questionnaire and interview in clinic, which addressed if the child indulges in tobacco use or if anybody living in the home where the child resides indulges in tobacco use. Demographic and anthropometric data that included age, sex, race, weight, height, and BMI were collected. In addition, information on blood pressures and fasting lipid profiles was collected. Blood pressures were obtained from the left arm in a sitting position (Dinamap). Fasting lipid profiles were obtained using a standard enzymatic assay in a clinical laboratory.

Inclusion Criteria

1. Children classified in the familial dyslipidemia group had BMI <95th percentile for age and sex, an increase in total and LDL cholesterol with no abnormalities in the rest of the lipid profile, normal blood pressure for age, sex, and height (<95th percentile), normal insulin levels, and a parent with a similar lipid profile and/or a history of premature coronary artery disease.

2. Children classified in the obese group had BMI ≥95th percentile for age and sex along with multiple atherosclerosis-promoting risk factors such as high triglyceride, high total and LDL cholesterol, low HDL cholesterol, high blood pressures for age, sex, and height (≥95th percentile), and high insulin levels.

Exclusion Criteria

1. Children who had not had carotid artery ultrasound examinations were excluded.

2. Children who were obese but had elevations of total and LDL cholesterol alone with no abnormalities in the rest of their lipid profile and with normal insulin and blood pressure levels were excluded from the obese group because it was deemed that they probably had familial dyslipidemia and were, in addition, obese.

3. Children with BMI <95th percentile for age and sex but with multiple atherosclerosis-promoting risk factors were excluded from the familial dyslipidemia group.

The 2 groups were mutually exclusive.

Carotid artery ultrasound for estimation of CIMT was offered as part of cardiovascular risk assessment. The carotid arteries were imaged using a standard ultrasound machine (Philips IE 33, Bothell, Wash) and a high-resolution, L9–3 MHz linear array transducer. The carotid imaging was performed by trained sonographers who followed a standardized institutional scanning protocol that was developed for pediatric imaging from modifications of a previously published protocol that had been used to develop the percentile data for adults (Appendix 1). The images were stored digitally as clips for offline reading.

The far wall of the common carotid artery was used for measurements of CIMT. The digital clips acquired from both the left and right common carotid arteries were analyzed offline using a semi-automated, edge detection software (Philips, QLAB). We used this software to measure the CIMT within a 10-mm-wide box (region of interest) that was placed along the far wall of the common carotid artery within 2 cm of the carotid bifurcation. Each measurement was accompanied by a “success rate,” which was the percentage of the intima-media within the region of interest that was able to be accurately measured. We only tabulated measured frames that had a success rate of 90% or above (Figure 1). Each study consisted of measuring CIMT on an average of >100 such frames of both the right and left carotid arteries.

CIMT measurements were tabulated in a spreadsheet and the grand maximum CIMT (mCIMT) and the grand mean CIMT (MCIMT) for each patient were derived. The MCIMT was obtained by deriving the mean of the maximum CIMT measurements from both the left and right carotid arteries. The MCIMT was plotted against CIMT percentile tables that have been published for a sex- and race-matched 45-year-old population (Table 1). The mCIMT was obtained by deriving the mean of all the CIMT measurements from both the left and right carotid arteries. The VA was defined as advanced if the child’s MCIMT plotted ≥25th percentile on the sex- and race-matched 45-year-old CIMT chart; the VA was defined as not advanced if the MCIMT was <25th percentile. Thus, we dichotomized the CIMT data of this population.

Presence of mutable atherosclerosis-promoting risk factors was noted, based on recommended cutoff levels previously published or norms accepted in our laboratory and clinic. These were as follows: obesity (BMI ≥95th percentile for age and sex), total cholesterol ≥170 mg/dL, HDL cholesterol ≤45 mg/dL, triglyceride ≤120 mg/dL, systolic blood pressure (SBP) ≥95th percentile for age, sex and height, fasting insulin levels ≥18 uIU, and history of exposure to tobacco smoke. The relation between the number of coexisting, mutable, atherosclerosis-promoting risk factors in obese children and MCIMT was further explored.

Inter-Reader and Intra-Reader Reliability

To estimate inter-reader and intra-reader reliability, a total of 106 frames (53 of the right common carotid artery and 53 of the left common carotid artery) were read by different readers. One reader read all these frames on 2 different occasions, blinded to the other reading for the purpose of determining intra-reader reliability. Three readers read all these frames independently and blinded to each others’ readings to establish inter-reader reliability. The intraclass correlation was evaluated using established guidelines in which values >0.75 indicate excellent reliability and values between 0.4 and 0.75 indicate good reliability. The intra-reader, intraclass correlation value was 0.81 (95% confidence interval [CI], 0.74 to 0.87), indicating excellent reliability. The inter-reader, intraclass correlation value was 0.67 (95% CI 0.57 to 0.76), indicating good reliability. The primary author (J.L.) read all the carotid scans for this study.

Statistical Methods

Continuous variables are described as mean±standard deviation and range, and the groups were compared using the Student t test. Categorical variables were described as percentages, and the groups were compared using the χ² test. A probability value of <0.05 was considered statistically significant. Simple linear and multiple linear regression analyses were used to investigate the association between atherosclerosis-promoting risk factors (chronological age, family history, BMI, SBP, total cholesterol, HDL cholesterol, triglyceride, history of exposure to tobacco smoke, and insulin levels) and MCIMT. Significant outliers were taken into consideration when finding the best simple linear regression model between MCIMT and
any one of the risk factors listed above. A multiple linear regression model was first used to determine the most significant predictors for MCIMT. Further model building was performed using standard techniques, such as mean shifted outlier testing, and variable-transformations. SAS (version 9.1, SAS Institute, Inc, Cary, NC) was used for statistical analysis.

Results

For the entire group of 70 children, the mean chronological age was $13.0 \pm 3.3$ years (range, 6 to 19 years). Sixty-two (89%) were white and 34 (49%) were male. There was a family history of premature cardiovascular disease in 58 (83%) children. Exposure to tobacco smoke was reported in 25 (36%). Forty (57%) were obese (BMI $25.6 \pm 6.0$ kg/m$^2$) with BMI $\geq$ 95th percentile for age and sex. Demographic, anthropometric, and blood pressure information of obese and familial dyslipidemic children are compared in Table 2.

When compared with familial dyslipidemic children, obese children had a significantly elevated SBP. Eighteen of the 40 (45%) obese children had SBP $\geq$ 95th percentile for age, sex, and height, 26 (65%) had LDL cholesterol $\geq 110$ mg/dL, 29 (73%) had HDL cholesterol $\leq 45$ mg/dL, 26 (65%) had triglyceride levels $\geq 120$ mg/dL, and 8 (20%) had insulin levels $\geq 18$ uIU/L. The most serious elevations of total cholesterol and LDL-C were noted in the familial dyslipidemic group. Obese children had significantly elevated triglyceride, very low-density lipoprotein cholesterol, glucose, and insulin levels and significantly lower HDL-C levels (Table 3).

The MCIMT data for the various age groups are categorized in Table 4. MCIMT for obese, dyslipidemic children was $0.53 \pm 0.05$ mm; for familial, dyslipidemic children, $0.52 \pm 0.04$ mm. Advanced VA was noted in 30 (75%) of obese children and 22 (73%) of familial dyslipidemic children (Table 5). Furthermore, 10 (25%) obese children and 7 (23%) familial dyslipidemic children had MCIMT that placed them $\geq$ 50th percentile for a race- and sex-matched 45-year-old.

### Table 1. Right and Left Common CIMT 25th Percentile for Sex and Race in 45-Year-Old Adults as Published in the Atherosclerosis Risk in Communities Study

<table>
<thead>
<tr>
<th>Percentiles</th>
<th>Black</th>
<th>Black</th>
<th>White</th>
<th>White</th>
</tr>
</thead>
<tbody>
<tr>
<td>RCCA</td>
<td>Women, mm</td>
<td>0.51</td>
<td>Men, mm</td>
<td>0.52</td>
</tr>
<tr>
<td>LCCA</td>
<td>Women, mm</td>
<td>0.49</td>
<td>Men, mm</td>
<td>0.53</td>
</tr>
<tr>
<td>RCCA indicates right common carotid artery; LCCA, left common carotid artery.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>From Reference 32.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
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Table 2. Demographic and Anthropometric Data

<table>
<thead>
<tr>
<th></th>
<th>Entire Cohort</th>
<th>Obese (n=40)</th>
<th>Familial Dyslipidemia (n=30)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>13.0±3.3</td>
<td>13.2±2.9</td>
<td>12.7±3.8</td>
<td>NS</td>
</tr>
<tr>
<td>White race*</td>
<td>62 (89%)</td>
<td>35 (88%)</td>
<td>27 (90%)</td>
<td>NS</td>
</tr>
<tr>
<td>Male*</td>
<td>34 (49%)</td>
<td>23 (58%)</td>
<td>11 (37%)</td>
<td>NS</td>
</tr>
<tr>
<td>Family history*</td>
<td>58 (83%)</td>
<td>29 (73%)</td>
<td>29 (97%)</td>
<td>NS</td>
</tr>
<tr>
<td>Tobacco smoke exposure*</td>
<td>25 (36%)</td>
<td>18 (45%)</td>
<td>7 (23%)</td>
<td>NS</td>
</tr>
</tbody>
</table>

| Weight, kg         | 64.0±23.4     | 76.0±19.8    | 48.0±17.8                   | <0.01   |
| Height, cm         | 155.4±18.0    | 159±15       | 151±21                      | NS      |
| BMI, kg/m²         | 26±6          | 30±4         | 20±3                        | <0.01   |
| BMI, Z score       | 1.37±0.98     | 2.09±0.28    | 0.41±0.71                   | <0.01   |
| Systolic blood pressure, mm Hg | 120±14 | 124±14 | 114±13 | <0.01 |
| Diastolic blood pressure, mm Hg | 65±8 | 66±7 | 64±8 | NS |

Data are presented as mean±SD or n (%). NS indicates not significant. *χ² tests with df=1; all other tests were Student t tests with df=68.

Table 3. Fasting Lipid Profile, Glucose, and Insulin Data

<table>
<thead>
<tr>
<th></th>
<th>Entire Cohort</th>
<th>Obese (n=40)</th>
<th>Familial Dyslipidemia (n=30)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cholesterol, mg/dL (&lt;170 mg/dL)*</td>
<td>223±58</td>
<td>204±42</td>
<td>249±65</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>LDL, mg/dL (&lt;110 mg/dL)*</td>
<td>150±59</td>
<td>130±66</td>
<td>175±66</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>HDL, mg/dL (&gt;45 mg/dL)*</td>
<td>46±13</td>
<td>42±11</td>
<td>52±14</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Triglycerides, mg/dL (&lt;120 mg/dL)*</td>
<td>152±105</td>
<td>187±121</td>
<td>104±53</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>VLDL, mg/dL (&lt;24 mg/dL)*</td>
<td>30±21</td>
<td>37±24</td>
<td>21±10</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Glucose, mg/dL† (&lt;120 mg/dL)*</td>
<td>87±8</td>
<td>89±8 (n=37)</td>
<td>84±6 (n=21)</td>
<td>0.01</td>
</tr>
<tr>
<td>Insulin, μU/mL‡ (&lt;18 μU/mL)</td>
<td>13.0±7.6</td>
<td>16±8 (n=23)</td>
<td>9±4 (n=17)</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

Data are presented as mean±SD.
*Considered optimal levels.
Student t test with df=68, except †df=56 and ‡df=38.

Discussion

Autopsy studies have confirmed that premature atherosclerosis is a notable finding in children and youth with risk factors. In this study, we estimated the VA of children with obesity and atherosclerosis-promoting risk factors and children with familial dyslipidemia by comparing their CIMT with 45-year-old adults and found a distribution of CIMT similar to that age group suggesting “advanced” VA in these children. CIMT values that were ≥25th percentile on a 45-year-old race- and sex-matched percentile chart was interpreted as indicative of an advanced VA because then the CIMT would undoubtedly fall in the expected range for a 45-year-old adult population. Obese children and children with familial dyslipidemia had equivalent progression in their VA.

The concept of VA is useful as the atherosclerotic burden of children with the same chronological age can vary based on the presence of single or multiple risk factors, risk factor interactions, length of exposure to the risk, genetic factors, or undocumented risks. About three quarters of all the children attending our clinic had an “advanced” VA, that is, their MCIMT was similar to that of a race- and sex-matched 45-year-old adult. There was, on average, a 0.07-mm increment in CIMT in children with an “advanced” VA. A 0.01-mm increase in CIMT per year is considered as an aging-associated normal progression in adults. Thus, in our population of high-risk children with an “advanced” VA, there may be an accelerated progression of CIMT.

Although there are several atherosclerosis-promoting risk factors contributing to premature vascular aging in obese children, these children most frequently had elevated fasting triglyceride levels (Figure 3). Thus, an elevated fasting triglyceride level in an obese child can be considered a marker for advanced VA. This association may be due to the effects of an associated insulin resistance. In our data set,
fasting insulin levels had not been performed in all patients (Table 3). However, it was noted that obese children as a group had higher fasting insulin levels signifying insulin resistance. Furthermore, obese children with >3 mutable atherosclerosis-promoting risk factors were noted to have a higher MCIMT compared with obese children with ≤3 risk factors. However, this did not reach statistical significance, which probably was a result of small sample size.

Total cholesterol, a significant predictor of atherosclerosis as demonstrated in other studies, was not found to be a predictor of MCIMT in our subjects by multiple linear regression analysis even though it was related to the increased MCIMT in the familial dyslipidemic group. This lack of significance in the multiple linear regression analysis may be due to our small sample size and inclusion of 40 obese children who had mild elevations in total cholesterol levels but had marked abnormalities in other atherosclerosis-promoting risk factors in this analysis.

Weight reduction via behavior modification, aerobic and strength training, or gastric bypass surgery in obese children is reported to be associated with a decrease in LDL-C, triglycerides, insulin levels, and SBP and an improvement in HDL-C.39–43 The efficacy and relative safety of statin therapy as a lipid-modifying drug in children with familial dyslipidemia have also been established.29,44 An intriguing question would be if the premature aging of the vasculature is reversible with lifestyle alterations or with pharmacological therapy of the risk factors. These structural vascular improvements have been reported after interventions in adults.45 A few pediatric reports have also described short-term, favorable changes in vascular function46–48 and CIMT measures29,48 after statin therapy or an exercise program in children with atherosclerosis-promoting risk factors. Because the adverse vascular changes are early in their evolution in children, resolution of the changes may be more likely. Of note, the mean age of our study children was comparable to children who underwent statin therapy in a large placebo-controlled trial that demonstrated CIMT regression with statin therapy and CIMT progression in the placebo group.29

We have also demonstrated that the performance of carotid artery ultrasound and measurement of CIMT is feasible in an

![Figure 2](http://circimaging.ahajournals.org/)

**Figure 2.** Significant difference (P<0.01) in MCIMT (mm) values in children with similar chronological ages but with “normal” and “advanced” VA are shown.

![Figure 3](http://circimaging.ahajournals.org/)

**Figure 3.** Bar graph representing the number of obese children with triglyceride levels ≥120 mg/dL and <120 mg/dL in the “normal” and “advanced” VA groups.

### Table 5. CIMT and Vascular Age Data

<table>
<thead>
<tr>
<th></th>
<th>Entire Cohort (n=70)</th>
<th>Obese Dyslipidemia (n=40)</th>
<th>Familial Dyslipidemia (n=30)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>MCIMT, mm*</td>
<td>0.53±0.05</td>
<td>0.53±0.05</td>
<td>0.52±0.04</td>
<td>NS</td>
</tr>
<tr>
<td>mCIMT, mm*</td>
<td>0.45±0.03</td>
<td>0.45±0.03</td>
<td>0.45±0.03</td>
<td>NS</td>
</tr>
<tr>
<td>Vascular age</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥25th percentile†</td>
<td>52 (74)</td>
<td>30 (75)</td>
<td>22 (73)</td>
<td>NS</td>
</tr>
</tbody>
</table>

Data are presented as mean±SD or n (%). NS indicates not significant.

*Student t test with df=68.

†χ² test with df=1.
outpatient clinic setting. Assessment of CIMT is noninvasive, technically easy to perform, reliable, relatively inexpensive, and devoid of radiation and other side effects. Because clinical cardiovascular events do not typically occur in childhood, subclinical markers of atherosclerosis such as CIMT can be considered the “end-organ effect” of exposure to all known and unknown atherosclerosis-promoting risk factors. Thus, CIMT and VA assessments may be useful in understanding the evolution of atherosclerotic vascular disease in children with obesity and multiple atherosclerosis-promoting risk factors and in following high-risk children who have had risk factor modification.

Limitations

The age range of the study group covers a period of rapid growth and associated physiological changes, and it was not possible to determine the length of time that each subject was exposed to the atherosclerosis-promoting risks factors that were tested for relationship with CIMT.

The anthropometric measures derived in this study did not include measurement of waist circumference; therefore we are unable to note the effects of central obesity on VA. The CIMT measurements used to assess VA in this study were done at a single point in time. Because normative data in this age group are limited, comparisons were made with normative data for a sex- and race-matched 45-year-old adult. CIMT is thought to increase slowly over time. However, without knowing the rate of progression of CIMT in children, the conclusions drawn from this study will need to be further validated with longitudinal observations in healthy and at risk children. This study only looked at the common carotid artery intima-media thickness and did not examine the intima-media thicknesses in the carotid bulb or the internal carotid artery; the evolution of atherosclerosis in these segments may vary.

Counseling children and families with regard to their vascular health by assigning a VA, which may be easily understood by families, may have a psychological and motivational impact, which cannot be assessed in this retrospective study. Whether such comparisons and counseling encourage families to undertake further measures that will lead to favorable changes in lifestyle beyond what would be undertaken with routine counseling must be studied.

Conclusion

VA is similarly advanced in obese children with atherosclerosis-promoting risk factors compared with children with familial dyslipidemia who are known to have premature cardiovascular disease. Obese children with multiple atherosclerosis-promoting risk factors may especially have an increased CIMT and hence should be screened for these risks. The use of carotid artery ultrasound and estimation of VA may help further stratify children who are at higher risk for developing accelerated atherosclerosis. VA estimation and the ability to relate CIMT findings to adult CIMT data may be a useful adjunct until CIMT normative data for children are well established.

Acknowledgments

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Disclosures

None.

References


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Appendix 1:

Children’s Mercy Hospital Cardiovascular Ultrasound Laboratory - CIMT Scanning Protocol

1. Patient is asked to lie down supine; EKG leads to be hooked up. Sonographer is to scan from the head end of the patient.

2. Turn patient’s head to left. Neck should be slightly hyper-extended, place towel if necessary next to the patient’s head to help maintain position.

3. Scan right carotid artery in short axis. Rotate the probe parallel to the artery. The carotid artery image should be seen horizontally across the screen now (common carotid, bulb and bifurcation). Use color Doppler and pulse wave Doppler to ascertain that the vessel being scanned is artery. Acquire clip #1.

4. Freeze with “Right” annotation and acquire clip #2.

5. Change back to 2D. Perform heel toe movements and/or change neck position (extension or lateral movement), change gain and resolution as required until the far wall of the distal common carotid artery is clearly imaged. You should see the intimal lining of the near and far wall and the carotid bifurcation in the same image. Acquire clip #s 3, 4, 5, 6, 7, 8, 9, 10.

6. Repeat steps 1 through 5 on the left side.