using pECG-CCTA and rECG-CCTA protocols were 1.7±0.1 mSv and 3.8±1.7 mSv respectively. (See table 1) None of the studies had coronary images which prevented an accurate assessment of the coronary segments.

Table 1 Comparison of effective radiation dose between pECG-CCTA and rECG-CCTA

<table>
<thead>
<tr>
<th>Sample population</th>
<th>Effective radiation dose (mSv)</th>
<th>Mean dose reduction (%)</th>
<th>BMI pECG-CCTA</th>
<th>rECG-CCTA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal weight (18.5−25)</td>
<td>1.7±0.2 (range, 1.3−2.2 mSv)</td>
<td>2.9±0.9 (range, 1.5−4.8 mSv)</td>
<td>41.4</td>
<td></td>
</tr>
<tr>
<td>Over weight (25−&lt;30)</td>
<td>1.7±0.1 (range, 1.6−1.7 mSv)</td>
<td>3.8±1.7 (range, 1.9−5.9 mSv)</td>
<td>65.3</td>
<td></td>
</tr>
</tbody>
</table>

Conclusions Using a pECG-CCTA protocol as compared to a rECG-CCTA protocol, there was 41.4% and 65.3% mean radiation dose reduction for the normal and overweight BMI groups respectively.

Related Subjects: Vascular Medicine

THE STUDY OF EFFECT OF ANGIOTENSIN II ON THE BIOLOGICAL BEHAVIOUR OF RAT VASCULAR SMOOTH MUSCLE CELLS IN VITRO
doi:10.1136/hrt.2010.208967.704

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Objective To study the effect of Angiotensin II on the proliferation, migration and apoptosis of vascular smooth muscle cell (VSMC) in rats.

Methods The recombinant adenoviral vector, AdCMV-AT2R, containing rat AT2 receptor gene was constructed by homologous recombination, and then it was used to transfer AT2 receptor gene to rat VSMC in vitro. The expression of AT2R mRNA was detected by RT-PCR and the rate of expression in VSMC was determined by flow cytometer. Cell proliferation was determined by incorporation of bromodeoxyuridine (BrdU). The modified Boyden’s chamber method was used to test the migration of VSMC. Apoptosis was quantified by flow cytometer.

Results RT-PCR showed that the expression of AT2R mRNA increased obviously in transferred VSMC, and the peak value of expression rate was about 89.51% at 48 h. When the expression of AT2R was at peak value, the OD value of BrdU incorporation were reduced by 51.6% (p<0.01), and the number of VSMC migration was also decreased by 62.2% (p<0.05). The ratio of apoptosis in VSMC was increased from 7.6±1.6% in control group to 32.1±5.5% in treated group.

Conclusion The results indicated that the expression of AT2R can inhibit the proliferation and migration of rat VSMC and induce its apoptosis.

EVALUATION OF INTRAVENTRICULAR FLOW IN DCM PATIENTS USING VECTOR FLOW MAPPING
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Aims To assess the change of intraventricular flow due to DCM (Dilated Cardiomyopathy) using VFM (Vector Flow Mapping).

Methods DCM patients and healthy candidates were included into two groups. 3C and 5C images were stored using VFM. Vector velocity of intraventricular flow was measured at the different planes and compared among groups.

Results Six DCM patients and 11 candidates were included. The vector velocity measured at LV outflow tract is lower in DCM group; a flow in direction of LV apex was found in the middle and apical planes of DCM patients while not in healthy candidates; the vector velocity of isovolumic contraction measured at basal plane is found to be higher in healthy group, but lower in the same group at middle and apical plane.

Conclusions In DCM patients, the vector velocity of LVOT is lower, which is accompanied by an abnormal flow distribution in middle and apical parts of left ventricle in both systole and isovolumic contraction. VFM can be used to evaluate the change of the intraventricular blood flow.