ORIGIN OF THE 3-RINGED APPEARANCE OF HUMAN ARTERIES BY ULTRASOUND: MICRODISSECTION WITH ULTRASONIC AND HISTOLOGIC CORRELATION

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Investigators have disagreed on the origin of the 3-ringed ultrasound appearance of normal and minimally atherosclerotic human arteries. To define the origin of each of these acoustic interfaces we performed microdissection on formalin fixed human arteries. For each arterial segment we studied regions with intima alone, intima and media, media alone, media and adventitia, adventitia alone or adventitia and intima. Subsequently intravascular imaging was performed with a 5 French 30 MHz ultrasound system providing a 360° degree cross-sectional image of a blood vessel in a plane perpendicular to the catheter tip. Microsurgical removal of the arterial intima and internal elastic lamina reduced the bright inner echo reflection. Removal of the media and the adventitia results in a bright echo signal from the remaining isolated intima. Removal of the media alone accentuates the echolucent zone between intima-adventitia. Removal of intima and media results in an isolated echo-dense outer ring. Removal of adventitia and intima with only residual media results in a weak but detectable echo signal. **Conclusion:** These microdissection studies identify the 3-ringed appearance of human vessels to emanate from the intima-internal elastic lamina (inner ring), media (middle, echolucent ring) and external elastic lamina-adventitia (outer ring).

HOW RELIABLE ARE INTRAVASCULAR ULTRASOUND AND FIBEROPTIC ANGIOSCOPY IN THE ASSESSMENT OF THE PRESENCE AND DURATION OF INTRAARTERIAL THROMBOSIS IN ATHEROMATOUS VESSELS WITH COMPLEX PLAQUES?

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We have previously shown that high-frequency intravascular ultrasound (IU) can detect intraluminal thrombi in *normal* vessels. In this in vitro study, we compared IU and fiberoptic angioscopy (FA) in examining (1) whether they can detect thrombi (TH) occurring within *arteries with complex atheromas (which themselves cause various patterns of ultrasound reflections)* and, (2) whether these techniques could gauge the age of intraarterial TH. Using both IU and FA, we imaged 33 coronary and peripheral atherosclerotic human arterial segments before and after experimental human TH. For IU imaging we used a 20 MHz catheter (IVUS) and a 30 MHz IU catheter (CVIS). The images were randomly mixed with images from 10 other diseased arteries without thrombi, and were blindly interpreted. Then, in 8 atherosclerotic arteries, human TH of varying duration were imaged both by IU and FA at 1 hr, 6 hrs, 12 hrs, 24 hrs and 7 days. Results: FA detected TH with 100% sensitivity and 100 % specificity. IU identified TH even in these atheromatous vessels with 92% sensitivity and 92% specificity: In both 30 and 20 MHz IU catheter images, atheromatous intima, media, and calcific, cystic, and necrotic areas in the wall, all were seen to cause ultrasound patterns different from those caused by TH. TH appeared as granular or speckled echo signals with gray level shades softer than the atheroma signals and bright calcification signals. We were able to detect not only intraluminal TH but also TH within necrotic or dissected areas in the wall. In judging TH duration, TH of all ages appeared dark red on FA and showed abnormal granular or speckled echoes on IU images. Neither FA nor IU was able to discriminate fresh from old TH. We conclude that the duration of intraarterial thrombosis can not be determined by IU or FA, but both techniques are highly capable of detecting intraarterial TH even in severely atheromatous arteries. Intravascular ultrasound provides added information on the architecture of the wall and atheroma.

INTRAVASCULAR ULTRASOUND: SIGNIFICANCE OF THE THREE-LAYERED APPEARANCE OF NORMAL MUSCULAR ARTERIES

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Normal muscular arteries have been shown to have a three-layered appearance on intravascular ultrasound (IU) scanning. Although the dark middle band and bright outer band of the IU image appear to correspond to media and adventitia histologically, the echogenic inner layer (EIL) of the image is too broad to represent to intima. We performed a three-phased study to determine the origin of the EIL:

1. Histologic correlation of 86 post-mortem human arteries showed that the EIL was prominent only in normal arteries with a well-defined internal elastic lamina. The EIL was significantly thicker than the histologic intima plus internal lamina (mean 308 ± 140um vs 31 ± 20um), presumably due to "blooming" (broadening) of the EIL on the image because of strong reflectance from the elastic tissue. In support of this conclusion, the thickness of the EIL plus dark middle band on the IU images correlated closely with the thickness of intima plus media histologically (mean 688 ± 20um vs. 657 ± 113um).

2. In 10 arterial specimens intima and internal lamina were removed progressively by surgical excision. Removal of intima alone had no significant effect on the EIL, but removal of the lamina caused a marked reduction in the prominence and thickness of the EIL. Significant reduction in the EIL was also seen when the lamina was enzymatically lysed with elastase in 4 specimens.

3. Five fresh bovine coronary artery segments were perfused over a graded range of pressures and the thickness of the EIL measured. At low pressures, the EIL was thick and bright corresponding to a dense, corrugated lamina; at physiologic pressures there was significant thinning of the EIL as the lamina was stretched.

We conclude: The EIL of the IU image of a normal muscular artery is primarily caused by the internal elastic lamina. The prominence of this layer is exaggerated in non-pressure fixed, in vitro specimens.

Monday, March 19, 1990
2:00PM–3:30PM, Room 41
Cardiac Function and Atrial Natriuretic Factor

DIRECT EFFECT OF ATRIAL TACHYCARDIA ON RELEASE OF ATRIAL NATRIURETIC FACTOR IN MAN

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Constant atrial distension increases accompany episodes of paroxysmal tachycardia in man. Previous studies have been unable to provide convincing evidence for a direct effect of atrial tachycardia on Atrial Natriuretic Factor (ANF) release. In order to eliminate the effects of atrial distension, we measured in 7 patients (60±9 yrs), 12 hours after coronary bypass graft surgery, mean arterial pressure (MAP); transmural right atrial (RAPTm) and pulmonary capillary wedge pressures (PcwPTm) (Swan-Ganz KT and esophageal balloon) and plasmatic ANF concentrations (pANF) (pulmonary artery samples, RIA, Normal value : 48±12 pg/ml). Protocol : control (C1); lower body positive pressure was applied ± fluid infusion to increase RAPTm and PcwPTm for 30 min (LBPP); rapid LBPP deflation was followed by 5 min of atrial tachycardia (AT) (pacing) to decrease RAPTm and PcwPTm during AT (AT); 45 min after AT (C2).

	C1	LBPP	AT	C2
HR (b/min)	84±16	79±8	150±16 #	77±10 #
pANF (pg/ml)	142±79	377±245 *	676±406 #	326±218 #
RAPTm (mmHg)	2.2±2.6	11.5±4 #	7±4 #	5±2.3
PcwpTm (mmHg)	3.2±4.9	14±3.9 #	10.5±4 #	6.4±4.4
MAP (mmHg)	83±20	100±23 #	81±27 #	95±22

HR : Heart Rate;* : $p < .05$,# : $p < .01$ Vs previous set (mean ± SD).

Two fold pANF increases were observed during AT, despite RAPTm and PcwPTm decreases. This demonstrates a direct stimulating effect on ANF release, separate from atrial stretch, providing an explanation for the extreme elevation in pANF during congestive heart failure and clinical tachycardias.