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rather than on correction of hemostatic function.
should thus be focused on the correction of lipid abnormalities
appear to be the principal risk factor. Prevention and intervention
development of arterosclerosis in HAL and lipid abnormalities
ion does not seem to contribute in a major way to the
positive family histories. Therefore, abnormal hemostatic func-

Test	HDL	HAL	Patients	Controls	n:16
Total cholesterol, mg/dL	171.0	328.4 ^a)	184.1		
Triglycerides, mg/dL	(98-245)	(211-451)	(130-221)		
HDL-cholesterol, mg/dL	103.6	92.3	77.1		
Total triglycerides, mg/dL	(55-178)	(41-150)	(36-129)		
Plasma fibrinogen, g/dL	0.30	0.29	0.26		
Factor VII coagulant activity, %	(84-108)	(101-131)	(87-100)		
Von Willebrand factor, %	107.9	99.9	85.0		
Antithrombin III, %	101.9	106.3 ^b)	(68-105)		
Factor VIII coagulant activity, %	95.0	99.9	85.0		
von Willebrand factor antigen, %	90-129	(93-106)	(90-100)		
Fibrinopeptide A, pg/ml	111-146	(101-130)	(90-125)		
Antithrombin III, %	127.7	115.0	106.9		
Factor VIII coagulant activity, %	84-108	(101-131)	(87-100)		
Plasminogen, mg/dL	0.25-0.34)	(0.26-0.31)	(0.23-0.29)		
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