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The Role of HMG-COA Reductase Inhibitors in the Treatment of Stable Angina Pectoris in Representatives of Different Professional Groups in Society

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Baseline clinical-functional and metabolic characteristics of patients with stable angina from different professional groups

1.1. General characteristics of the groups, occupational profile, and risk factors

Before the initiation of therapy, the baseline clinical characteristics of the study groups were assessed. The comparability of the groups is presented in Table 1.1.

Table 1.1 Baseline clinical and demographic comparability of the study groups

Group	N	Age, years	CHD duration, years	BMI, kg/m²	Smoking, n (%)	AH, n (%)
I	30	43.2±5.4	—	24.8±1.7	3 (10.0%)	8 (26.7%)
II	30	43.4±4.3	2.9±1.0	27.0±2.2	13 (43.3%)	12 (40.0%)
III	30	43.2±5.2	3.1±1.1	26.9±2.0	8 (26.7%)	12 (40.0%)
IV	30	42.9±4.3	3.3±1.0	27.6±1.8	13 (43.3%)	18 (60.0%)
V	30	42.2±4.2	3.2±1.1	27.5±2.1	13 (43.3%)	20 (66.7%)

Note. Data are presented as M±SD or n (%). No statistically significant differences were found between the clinical groups in age, CHD duration, or BMI, p>0.05.

According to the data in Table 1.1, the age distribution in groups II–V was close to one another. The mean age was 43.4±4.3 years in group II, 43.2±5.2 years in group III, 42.9±4.3 years in group IV, and 42.2±4.2 years in group V. The difference between the smallest and largest group mean values did not exceed 1.2 years.

The duration of coronary heart disease (CHD) in patients also fell within a narrow range: 2.9±1.0 years in group II, 3.1±1.1 years in group III, 3.3±1.0 years in group IV, and 3.2±1.1 years in group V. The maximum difference between groups II and IV did not exceed 0.4 years. This is important for interpreting the chapter's findings: the longer stable angina persists, the higher the probability of accumulated ischemic episodes, myocardial remodeling, changes in patient habits, and a more complex medication history. No such time-related shift between groups was observed in this study.

In the control group, body mass index (BMI) was 24.8±1.7 kg/m², whereas in groups II–V it ranged from 26.9±2.0 to 27.6±1.8 kg/m². The clinical groups as a whole approached the overweight range. The lowest mean BMI was recorded in group III at 26.9±2.0 kg/m², and the highest in group IV at 27.6±1.8 kg/m². Differences among groups II–V were small, but the shift relative to control was clinically meaningful: when overweight, arterial hypertension, and atherogenic dyslipidemia occur together, a denser cardiometabolic background for angina forms even within the same functional class.

Groups II–V could not be regarded as fully identical with respect to smoking. In group II, 13 patients (43.3%) smoked; in group III, 8 patients (26.7%); and in groups IV and V, 13 patients each (43.3%). The difference between group III and the other clinical groups reached 16.6 percentage points. This did not undermine the overall baseline comparability of the sample, but it also means the risk profiles were not perfectly matched. Smoking was taken into account in the subsequent analysis of symptom and laboratory-marker dynamics, since this factor alone can influence vascular reactivity and oxidative stress independently of the treatment regimen.

The most notable baseline difference concerned arterial hypertension (AH). In groups II and III, it was recorded in 12 of 30 patients each (40.0%). In group IV, hypertension was found in 18 patients (60.0%), and in group V, in 20 patients (66.7%). The difference between groups II and V reached 26.7 percentage points. The control group served as a physiological reference; hypertension was recorded in 8 of 30 examined subjects (26.7%). Thus, at baseline the patient contingent differed from control not only in the presence of angina but also in a denser hemodynamic risk profile.

Table 1.2 Occupational characteristics and risk factors in patients with stable angina, by occupational category

	N	Smoking, n (%)	AH, n (%)	Dyslipidemia, n (%)	Overweight, n (%)	Night shifts, n (%)	Stress load, n (%)	Hypodyn., n (%)
Healthcare worker	33	9 (27.3%)	19 (57.6%)	22 (66.7%)	19 (57.6%)	27 (81.8%)	28 (84.8%)	7 (21.2%)
Programmer	28	9 (32.1%)	13 (46.4%)	18 (64.3%)	16 (57.1%)	20 (71.4%)	21 (75.0%)	7 (25.0%)
Marketer	25	8 (32.0%)	11 (44.0%)	15 (60.0%)	9 (36.0%)	17 (68.0%)	19 (76.0%)	10 (40.0%)
Driver	34	21 (61.8%)	19 (55.9%)	24 (70.6%)	16 (47.1%)	13 (38.2%)	23 (67.6%)	12 (35.3%)

Note. Categorical indicators are presented as n (%).

The main differences in occupational risk between patient types in the study groups are visualized in Fig. 1.1.

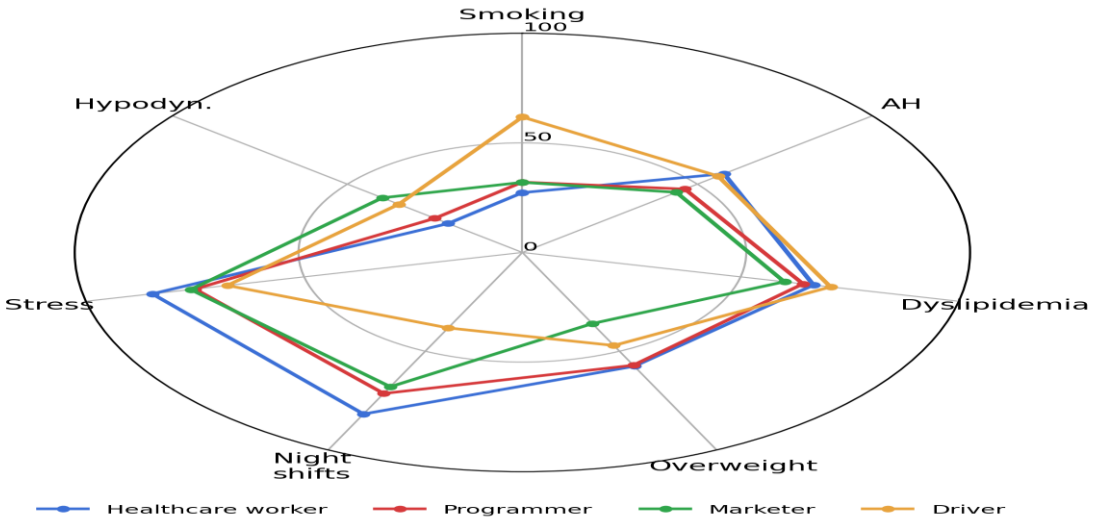


Fig. 1.1. Risk factor profile by occupational category.

Fig. 1.1 brings together the factors that most strongly differentiated the occupational contingents and are of the greatest clinical relevance for further analysis. Visually, the groups standing out most from the overall array are drivers for smoking (61.8%) and healthcare workers for the combination of night shifts (81.8%) and stress load

(84.8%). Programmers occupy an intermediate position, with night shifts at 71.4% and stress at 75.0%. Marketers do not appear "low-risk": although overweight is less common (36.0%), stress (76.0%) and physical inactivity (40.0%) remain elevated.

1.2. Clinical course of stable angina and functional diagnostics

The clinical course of stable exertional angina in the study group was not assessed on the basis of complaints alone. In this category of patients, some symptoms are described vaguely, some load-related episodes pass without an immediate cessation of activity, and the routine of service duties may shift the subjectively perceived pain threshold. For this reason, the baseline status was recorded using three levels of indicators: the frequency of attacks and the need for nitroglycerin, daily monitoring parameters, and the results of the bicycle ergometry test.

The discrepancy between the frequency of anginal episodes and actual nitrate intake is of particular interest. In the general civilian population, these values are often close to one another, whereas in a military environment the correspondence is weaker. The decision to take medication is influenced both by the service situation and by individual habits of tolerating load-related discomfort until a task or shift is completed.

Table 1.3 Baseline clinical profile of the patients

Indicator	I	II	III	IV	V	Between groups, p
Attacks/week	0.0	5.3±1.4	5.6±1.5	5.5±1.5	6.0±1.8	0.348
Nitroglycerin/week	0.0	5.1±1.3	5.2±1.1	4.5±1.1	5.5±1.3	0.018
Daytime HR, bpm	68.0±5.9	80.6±7.2	80.4±7.5	80.5±6.6	81.9±5.8	0.806
SBP, mmHg	125.7±8.0	150.6±10.3	151.0±8.0	149.4±12.0	151.6±9.3	0.860
DBP, mmHg	78.4±4.2	91.4±7.3	89.2±6.5	89.1±6.1	89.9±5.1	0.479
ST episodes/day	0.00	1.66±0.65	1.63±0.65	1.80±0.65	1.88±0.65	0.374
ST depth, mm	0.00	1.37±0.36	1.43±0.33	1.41±0.35	1.57±0.27	0.095

Note. ANOVA calculated between groups II–V. Comparison of each clinical group with control for attack frequency, nitroglycerin need, and ST parameters was statistically significant relative to the healthy control group, $p<0.001$.

Regarding attack frequency, groups II–V were close to one another. In group II, anginal episodes averaged 5.3±1.4 per week; in group III, 5.6±1.5; in group IV, 5.5±1.5; and in group V, 6.0±1.8; the between-group test gave p=0.348. The interval between the minimum and maximum mean values did not exceed 0.7 attacks per week. This allowed the baseline symptomatic load to be regarded as comparable across the main clinical marker of angina.

A different picture emerged for nitroglycerin need. It was 5.1±1.3 tablets per week in group II, 5.2±1.1 in group III, 4.5±1.1 in group IV, and 5.5±1.3 in group V; p=0.018 between clinical groups. The difference between groups IV and V reached 1.0 tablet per week under conditions of similar attack frequency. Nitroglycerin need reflects not only ischemia intensity but also the patient's pain-relief behavior, how quickly the load is halted, and the subjective threshold for resorting to medication. This indicator therefore received particular attention in the subsequent comparison of therapy dynamics.

In the control group, mean daytime heart rate was 68.0±5.9 bpm. In groups II–V it ranged from 80.4±7.5 to 81.9±5.8 bpm, roughly 12–14 bpm higher than control. The lowest group value was 80.4±7.5 bpm in group III, and the highest was 81.9±5.8 bpm in group V; p=0.806 between clinical groups. Here, both the fact of

tachycardization relative to control and its consistency across all clinical groups are important. An accelerated rhythm increases the myocardial oxygen requirement and, even on the same anatomical substrate, heightens vulnerability under load [84].

Blood pressure was significantly higher than control in all clinical groups. Systolic blood pressure (SBP) was 125.7±8.0 mmHg in control versus 150.6±10.3, 151.0±8.0, 149.4±12.0, and 151.6±9.3 mmHg in groups II, III, IV, and V, respectively. Diastolic blood pressure (DBP) was 78.4±4.2 mmHg in control versus 91.4±7.3, 89.2±6.5, 89.1±6.1, and 89.9±5.1 mmHg in groups II–V, respectively. Between-group p-values were 0.860 for SBP and 0.479 for DBP, i.e., the clinical groups were close to one another in this respect. At the same time, the difference from control was large enough that hemodynamic load can be regarded as a common baseline feature of the entire angina sample.

Daily episodes of ST-segment depression were not recorded in the control group but were present in all clinical groups. Episode frequency was 1.66±0.65/day in group II, 1.63±0.65 in group III, 1.80±0.65 in group IV, and 1.88±0.65 in group V; p=0.374. The depth of ST depression was 1.37±0.36, 1.43±0.33, 1.41±0.35, and 1.57±0.27 mm, respectively; p=0.095. These values are moderate but consistent with a real ischemic load profile that cannot be explained by the subjective description of pain alone. This is fundamental for the baseline characterization: functional ischemia was present before treatment began, and its magnitude was similar across groups II–V.

Symptoms, hemodynamics, and daily ST episodes formed the first layer of objectification of the disease. Holter monitoring, combining rhythmic load and ischemic changes, provided a more detailed picture.

Table 1.4 Baseline Holter ECG monitoring parameters

Indicator	I	II	III	IV	V	Between groups, p
Daytime HR, bpm	68.0±5.9	80.6±7.2	80.4±7.5	80.5±6.6	81.9±5.8**	0.01
Night-time HR, bpm	53.8±0.8	62.8±0.8	62.5±0.8	62.9±0.8	62.6±0.8*	0.05
Maximal HR, bpm	107.7±9.7	127.5±8.0**	128.1±12.2**	130.0±15.0**	129.9±11.5**	0.01
ST depression episodes, n/day	not recorded	1.66±0.65***	1.63±0.65***	1.80±0.65***	1.88±0.65***	0.001
ST depression depth, mm	not recorded	1.37±0.36***	1.43±0.33***	1.41±0.35***	1.57±0.27***°	0.001; 0.05
Extrasystoles/hour	1.0±1.1	5.0±2.2***	4.7±2.2***	5.3±2.2***	6.4±2.2***°	0.001; 0.05

Note. Data are presented as M±SD. No ST depression episodes or their depth within a clinically meaningful range were recorded in the control group.

Daytime heart rate was elevated in all angina patients, changing from 68.0±5.9 bpm in control to a range of 80.4±7.5–81.9±5.8 bpm. Night-time heart rate also remained above control: 62.8±0.8 bpm in group II, 62.5±0.8 in group III, 62.9±0.8 in group IV, 62.6±0.8 in group V, versus 53.8±0.8 bpm in control. The between-group difference in night-time HR was not large, p=0.278, but the incomplete slowing of rhythm during night hours corresponds well with the proportion of night shifts and sleep-pattern disruption shown in Table 1.2.

On daily monitoring, maximal heart rate rose from 107.7±9.7 bpm in control to 127.5±8.0, 128.1±12.2, 130.0±15.0, and 129.9±11.5 bpm in groups II–V, p=0.796. Differences among the clinical groups were minimal, but the rise above control level indicates a higher degree of daily hemodynamic mobilization. This is particularly

important for service members: beyond laboratory-based load, there are periods during the working day when heart rhythm accelerates that do not always prompt medical attention.

No ST depression episodes were recorded in control. In groups II–V their frequency ranged from 1.63±0.65 to 1.88±0.65 per day, and their depth from 1.37±0.36 to 1.57±0.27 mm. The deepest ST depression was recorded in group V (1.57±0.27 mm), and the smallest in group II (1.37±0.36 mm). Since p=0.095 for ST depth, these differences do not place the groups into distinct clinical-functional categories, but they do mark the boundary between milder and more strained variants of baseline ischemic load.

Extrasystole deserves separate attention. In the control group, extrasystole frequency was 1.0±1.1 per hour. In patients, values rose to 5.0±2.2 in group II, 4.7±2.2 in group III, 5.3±2.2 in group IV, and 6.4±2.2 in group V. The between-group difference was statistically significant, p=0.026. This is one of the few functional indicators for which the clinical groups were not fully identical before treatment began.

Taken together, the Holter monitoring data define a complex functional phenotype rather than an isolated pain syndrome. In angina patients, an accelerated rhythm, daily ischemic episodes, and a higher arrhythmic burden coexist simultaneously.

The daily frequency and depth of ischemic episodes are shown in Fig. 1.2.

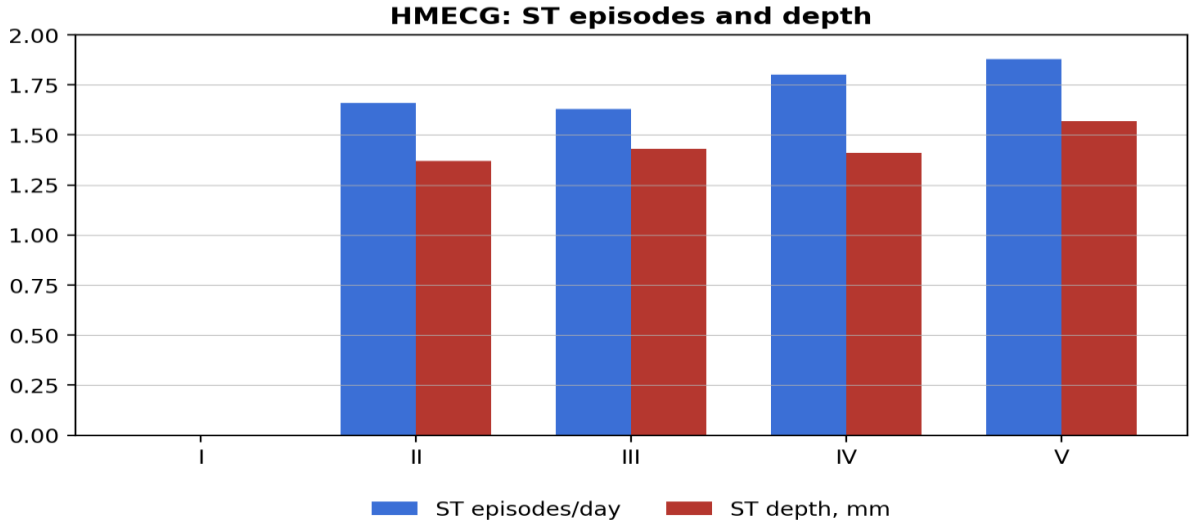


Fig. 1.2. Ischemic load according to Holter ECG monitoring data.

Fig. 1.2 brings together the two most notable parameters of Holter ischemic load, namely the frequency of ST depression episodes and their depth, in a single field. In group II these indicators were 1.66 episodes and 1.37 mm per day; in group III, 1.63 and 1.43 mm; in group IV, 1.80 and 1.41 mm; and in group V, 1.88 and 1.57 mm. The control group remained at zero on both criteria. In terms of daily ST load, group V showed a somewhat more strained baseline profile, while in group III, despite a similar depression depth, episode frequency was lower.

Baseline ambulatory blood pressure monitoring (ABPM) results are presented in Table 1.5.

Table 1.5 Baseline ABPM parameters

Indicator	I	II	III	IV	V	Between groups, p
ABPM SBP, mmHg	125.7±8.0	150.6±10.3**	151.0±8.0**	149.4±12.0**	151.6±9.3**	0.01

Indicator	I	II	III	IV	V	Between groups, p
ABPM DBP, mmHg	78.4±4.2	91.4±7.3**	89.2±6.5*	89.1±6.1*	89.9±5.1**	0.01; 0.05

Note. The table shows mean 24-hour SBP and DBP values presented uniformly across the final groups I–V. Statistics were significant relative to the healthy control group, p<0.05; p<0.01.

Mean 24-hour systolic blood pressure was 125.7±8.0 mmHg in control, versus 150.6±10.3, 151.0±8.0, 149.4±12.0, and 151.6±9.3 mmHg in groups II–V. Diastolic pressure rose from 78.4±4.2 mmHg in control to 91.4±7.3, 89.2±6.5, 89.1±6.1, and 89.9±5.1 mmHg, respectively. There were no differences among groups II–V: p=0.860 for SBP and p=0.479 for DBP.

A higher mean 24-hour blood pressure in angina increases afterload, raises the myocardial oxygen requirement, and accelerates mechanical strain on the vascular wall. Together with the data in Table 1.1, particularly the frequent occurrence of arterial hypertension in groups IV and V, this creates an important background for evaluating the subsequent dynamics of antianginal therapy.

Table 1.2 shows that 81.8% of healthcare workers and 71.4% of programmers work during the second half of the day into the night, with high stress reported in 84.8% and 75.0%, respectively. This occupational context helps explain why blood pressure remains persistently elevated even under monitored conditions. This reflects not only the disease itself but also the everyday circumstances that sustain it.

The next objective block concerns tolerance to graded exercise. While Holter monitoring and ABPM capture the everyday functional background, bicycle ergometry allows measurement, under controlled conditions, of the threshold at which coronary reserve becomes insufficient. Baseline bicycle ergometry results are given in Table 1.6.

Table 1.6 Baseline bicycle ergometry (VEM) parameters

Indicator	I	II	III	IV	V	Between groups, p
Threshold power, W	120.4±12.7	79.9±9.7	78.8±9.3	78.1±8.5	77.1±9.0	0.675
Exercise duration, min	9.8±1.1	6.3±0.8	6.3±0.8	6.4±1.0	6.3±1.0	0.957

Note. The table allows direct comparison, without recalculation, of the categorical test results — threshold power and exercise duration — across groups I–V.

Exercise tolerance decreased uniformly in patients with stable angina. Threshold power was 120.4±12.7 W in control, falling to 79.9±9.7 W in group II, 78.8±9.3 W in group III, 78.1±8.5 W in group IV, and 77.1±9.0 W in group V. The between-group test gave p=0.675.

Exercise duration was 9.8±1.1 minutes in control and approximately 6.3–6.4 minutes in patients: 6.3±0.8 min in group II, 6.3±0.8 in group III, 6.4±1.0 in group IV, and 6.3±1.0 in group V, p=0.957. The difference from control reached roughly three and a half minutes. In this type of exercise test, such a difference is not a minor detail; it reflects earlier attainment of the ischemic threshold and faster exhaustion of coronary reserve.

The similarity of groups II–V in power and exercise duration is also important for subsequent between-group analysis of therapy. Had one group started with a substantially higher exercise threshold, subsequent improvement could have been partly attributable to that baseline advantage rather than to the treatment regimen. No such shift

was present here. The clinical groups were close not only in symptom reports at baseline but also on objective functional testing.

The comparison of power and exercise duration across groups is shown in Fig. 1.3.

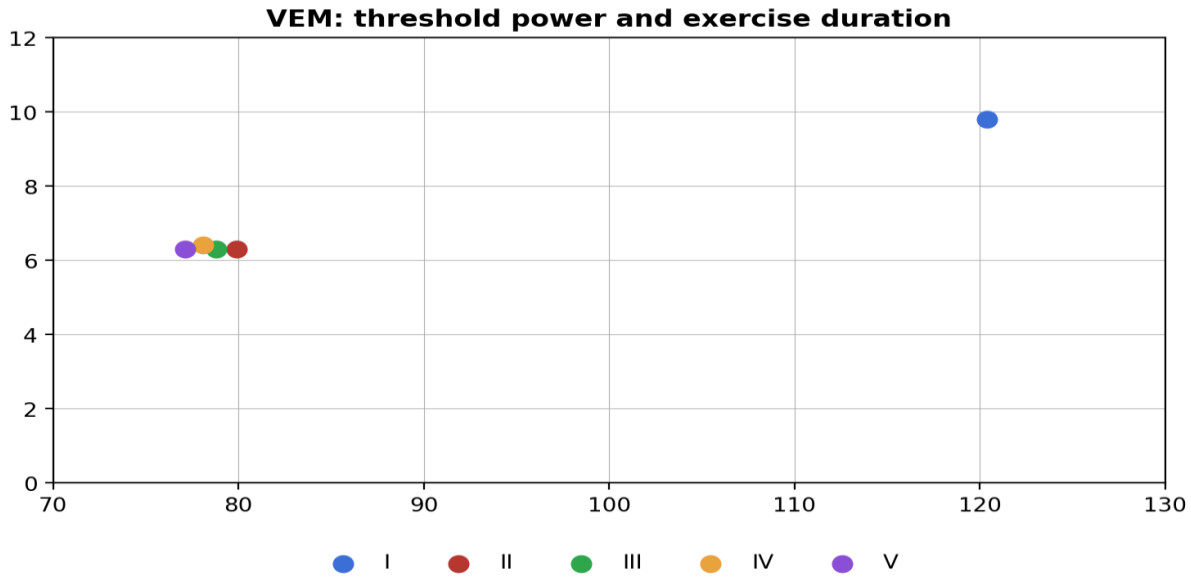


Fig. 1.3. Threshold power and exercise duration by group.

The control group combined a threshold power of 120.4 W with an exercise duration of 9.8 minutes. In groups II–V, power fell to 77.1–79.9 W and duration to 6.3–6.4 minutes. Within the clinical block, the curves virtually overlap.

Clinical attacks, daily ischemic load, ABPM parameters, and exercise testing all point in the same direction. At baseline, patients in groups II–V shared not only a similar symptomatic profile but also a comparable degree of objective functional deficit.

1.3. Baseline lipid, apolipoprotein and inflammatory profile

The task of the laboratory block was to link atherogenic load, inflammatory background, and subsequent functional limitations within a single baseline model of the disease. For this purpose, standard lipidogram parameters were used together with apolipoprotein B (Apo-B) as a marker of atherogenic particle number, and C-reactive protein (CRP) as an indicator of low-grade inflammation. This approach was chosen because, in patients with functional class II stable angina, disease severity is not always determined by attack frequency alone. Two patients with similar symptoms may differ substantially in the number of Apo-B-containing particles, the atherogenic coefficient, and the degree of inflammatory activation. Thus, the baseline laboratory profile serves not only to describe lipid metabolism but also to establish how metabolically "saturated" the coronary risk was before treatment began.

Lipid spectrum, apolipoprotein B, and CRP values are shown in Table 1.7.

Table 1.7 Baseline lipid profile, Apo-B, and CRP

Indicator	I	II	III	IV	V	Between groups, p
TC, mmol/L	4.38±0.40	6.21±0.44	6.22±0.55	6.22±0.45	6.44±0.42	0.157
TG, mmol/L	1.13±0.21	2.10±0.28	2.10±0.35	2.11±0.28	2.06±0.37	0.912

Indicator	I	II	III	IV	V	Between groups, p
HDL-C, mmol/L	1.41±0.16	0.91±0.11	0.91±0.13	0.93±0.11	0.91±0.13	0.794
LDL-C, mmol/L	2.40±0.36	4.13±0.48	4.16±0.49	4.11±0.46	4.29±0.45	0.407
AC, arb. units	2.14±0.38	5.91±1.03	6.01±1.25	5.76±1.05	6.26±1.23	0.394
Apo-B, g/L	0.75±0.09	1.23±0.17	1.23±0.18	1.21±0.17	1.24±0.16	0.904
CRP, mg/L	0.91±0.38	3.46±0.72	3.41±0.57	3.45±0.78	3.52±0.76	0.944

Note. In all clinical groups compared with control, LDL-C, HDL-C, atherogenic coefficient, Apo-B, and CRP differed significantly, $p<0.001$.

According to Table 1.7, a marked atherogenic profile had formed in patients with stable angina. Total cholesterol (TC) was 4.38±0.40 mmol/L in control versus 6.21±0.44, 6.22±0.55, 6.22±0.45, and 6.44±0.42 mmol/L in groups II–V. The between-group difference among patients did not reach statistical significance, p=0.157. This level alone indicates that all angina groups entered the study with a similar degree of hypercholesterolemia.

Triglycerides (TG) were also above control values: 1.13±0.21 mmol/L in control versus 2.10±0.28 mmol/L in group II, 2.10±0.35 in group III, 2.11±0.28 in group IV, and 2.06±0.37 in group V; p=0.912 between clinical groups. There were virtually no differences within the clinical block, indicating that the rise in TG was not a feature of a single therapeutic subgroup but part of the shared metabolic background.

The shifts in LDL-C and HDL-C proved most fundamental. LDL-C was 2.40±0.36 mmol/L in control, rising to 4.13±0.48, 4.16±0.49, 4.11±0.46, and 4.29±0.45 mmol/L in groups II–V. Every clinical group differed significantly from control, p<0.001, but there was no significant difference among groups II–V, p=0.407. At the same time, HDL-C fell from 1.41±0.16 mmol/L in control to 0.91±0.11, 0.91±0.13, 0.93±0.11, and 0.91±0.13 mmol/L in patients (p<0.001 versus control; p=0.794 among clinical groups). The combination of elevated LDL-C and reduced HDL-C forms the classic atherogenic lipid configuration.

The atherogenic coefficient (AC) reinforced the impression given by individual lipid fractions. It was 2.14±0.38 arbitrary units in control, versus 5.91±1.03 in group II, 6.01±1.25 in group III, 5.76±1.05 in group IV, and 6.26±1.23 in group V. Even the lowest value among patients was more than 2.5-fold higher than control. The between-group p-value within block II–V was 0.394. Thus, the clinical groups entered the study not merely with elevated LDL-C, but with a consistently shifted overall balance of atherogenic to anti-atherogenic fractions.

Apolipoprotein B clarified this conclusion at the level of atherogenic particle number. Its concentration was 0.75±0.09 g/L in control. In patients it ranged from 1.21 to 1.24 g/L: 1.23±0.17 g/L in group II, 1.23±0.18 in group III, 1.21±0.17 in group IV, and 1.24±0.16 in group V. Differences from control were significant at p<0.001, while among clinical groups p=0.904.

CRP was 0.91±0.38 mg/L in control. In groups II–V, CRP was recorded at 3.46±0.72, 3.41±0.57, 3.45±0.78, and 3.52±0.76 mg/L. All clinical groups differed reliably from control, p<0.001, with no difference among groups II–V, p=0.944. Thus, in the examined service members, baseline coronary risk involved not only dyslipidemia but also a low-grade inflammatory background capable of sustaining the biological activity of the atherosclerotic process.

Groups II–V were sufficiently uniform in their lipid-inflammatory parameters, so that the subsequent comparison of dynamics was not confounded by a baseline advantage of any single regimen. At the same time, the contrast with control was pronounced across all major positions: LDL-C, HDL-C, AC, Apo-B, and CRP. This indicates

that the functional limitations identified in section 3.2 developed against a background of pronounced atherogenic and inflammatory load.

The generalized atherogenic contour across groups is visualized in Fig. 1.4.

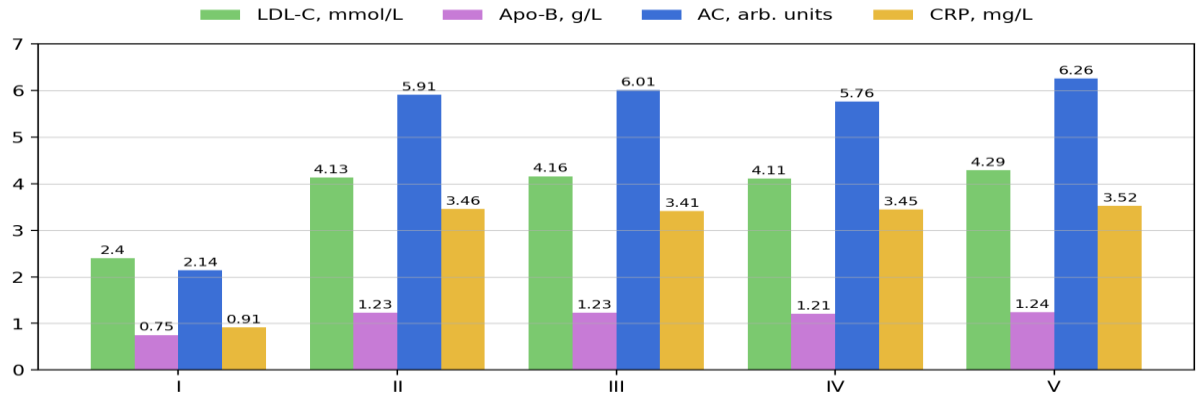


Fig. 1.4. Atherogenic profile by group.

Fig. 1.4 clearly shows that groups II–V nearly overlap in their baseline atherogenic contour. In these groups, LDL-C ranged from 4.11 to 4.29 mmol/L, Apo-B from 1.21 to 1.24 g/L, the atherogenic coefficient from 5.76 to 6.26 arbitrary units, and CRP from 3.41 to 3.52 mg/L. The control group occupied a distinct position: LDL-C 2.40 mmol/L, Apo-B 0.75 g/L, AC 2.14, and CRP 0.91 mg/L.

1.4. State of the lipid peroxidation–antioxidant system and endothelial NO-producing function

The baseline metabolic characterization included the assessment of lipid peroxidation (LPO), the antioxidant system (AOS), and endothelial NO production as an interconnected functional block. Given the expressed LPO–AOS imbalance, mandatory joint analysis with NO exchange parameters is justified. Chronic psycho-emotional strain, disrupted sleep, shift work, and variable physical load create conditions for both increased oxidative imbalance and a shift of vascular regulation toward vasoconstrictor reactions.

Baseline parameters of the LPO–AOS and NO systems are presented in Table 1.8.

Table 1.8 Baseline parameters of the LPO–AOS and NO systems

Indicator	I	II	III	IV	V	Between groups, p
MDA, nmol/mL	16.6±2.0	30.7±4.3	30.1±4.4	30.2±3.7	32.5±3.8	0.081
SOD, arb. units/mg Hb	66.8±6.5	37.1±6.6	36.2±6.1	37.2±5.6	36.0±5.1	0.796
Catalase, μkat/L	234.6±11.9	161.6±19.3	159.3±17.8	156.2±20.8	158.1±21.2	0.767
Ceruloplasmin, mg/L	257.9±16.7	157.6±20.3	163.1±22.2	159.1±21.3	160.5±18.6	0.763
NOx, μmol/L	70.0±5.4	39.2±5.2	41.7±4.6	40.9±6.5	39.0±5.6	0.162

Note. LPO–AOS — lipid peroxidation and antioxidant system. Comparison of each clinical group with control for MDA, SOD, catalase, ceruloplasmin, and NO was significant at p<0.001.

Malondialdehyde (MDA) proved the most contrasting indicator in this block. Its level was 16.6±2.0 nmol/mL in control, rising to 30.7±4.3 nmol/mL in group II, 30.1±4.4 in group III, 30.2±3.7 in group IV, and 32.5±3.8 in

group V. All clinical groups differed from control at p<0.001, with no significant difference within block II–V, p=0.081. The magnitude of this range alone indicates that lipid peroxidation was nearly doubled in angina patients.

Superoxide dismutase (SOD) activity changed in the opposite direction. SOD was 66.8±6.5 arbitrary units/mg Hb in control. In groups II–V, values were 37.1±6.6, 36.2±6.1, 37.2±5.6, and 36.0±5.1 arbitrary units/mg Hb, respectively. The decrease relative to control was pronounced, p<0.001, with no difference among clinical groups, p=0.796. In effect, this reflects a profound weakening of enzymatic antioxidant defense against a background of already activated lipid peroxidation.

Catalase and ceruloplasmin values confirmed the same trend. Catalase was 234.6±11.9 μkat/L in control, falling to 161.6±19.3, 159.3±17.8, 156.2±20.8, and 158.1±21.2 μkat/L in groups II–V (p<0.001 versus control; p=0.767 among clinical groups). Ceruloplasmin fell from 257.9±16.7 mg/L in control to 157.6±20.3, 163.1±22.2, 159.1±21.3, and 160.5±18.6 mg/L, respectively (p<0.001 and p=0.763). These values are difficult to interpret as random fluctuation of a single marker; a coordinated shift was observed across the entire antioxidant block.

As the summary stable metabolites of nitric oxide, NOx was reduced in all patients. It was 70.0±5.4 μmol/L in control versus 39.2±5.2 μmol/L in group II, 41.7±4.6 in group III, 40.9±6.5 in group IV, and 39.0±5.6 in group V. Differences from control were significant at p<0.001, and among clinical groups p=0.162. The decrease of NOx to roughly 39–42 μmol/L indicates a persistent limitation of NO-dependent vasodilator regulation.

It is essential to view these indicators not in isolation but within a single pathophysiological sequence. The rise in MDA was not an isolated laboratory finding; it occurred together with reduced SOD, catalase, and ceruloplasmin, as well as lower NOx values. This profile is consistent with a model in which oxidative modification of lipid structures and weakened antioxidant defense coincide with reduced nitric oxide bioavailability and a strengthened vasoconstrictor background. Patients also had elevated LDL-C and Apo-B, meaning more atherogenic particles were circulating in the blood. Against a background of heightened lipid peroxidation, this environment becomes biologically more aggressive. In other words, the metabolic picture in angina patients was not limited to cholesterol transport but encompassed a complex of lipid-oxidative disturbances.

The link with functional data is also meaningful. In the same groups where 1.63–1.88 ST depression episodes per day and reduced VEM power were recorded, MDA simultaneously reached 30.1–32.5 nmol/mL and NOx 39.0–41.7 μmol/L. These parallel patterns do not by themselves establish causality, but they provide a well-grounded hypothesis for the correlation block: functional ischemia develops in an environment where atherogenic and oxidative loads are closely intertwined.

When comparing the clinical groups, most metabolic indicators showed no differences. At baseline, no clinical group had an advantage in MDA, SOD, catalase, ceruloplasmin, or NOx. This means that the subsequent dynamics of these markers can be interpreted more reliably as a treatment effect rather than as a baseline imbalance. The deviations of lipid, inflammatory, and metabolic indicators relative to control are shown in Fig. 1.5.

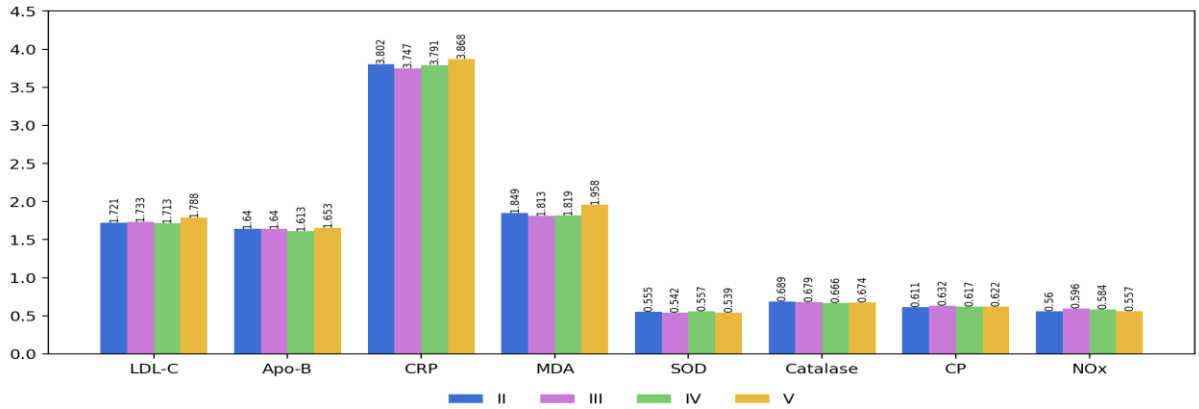


Fig. 1.5. Standardized deviation of lipid, inflammatory and metabolic parameters from control values

In groups II–V, the ratio to control values was approximately 1.7–1.8 for LDL-C, around 1.6 for Apo-B, 3.7–3.9 for CRP, and 1.8–2.0 for MDA. SOD, catalase, ceruloplasmin, and NOx shifted in the opposite direction; their relative level in patients was markedly below unity.

CRP showed the sharpest departure from control, followed by the LPO–AOS and NO system parameters. Fig. 1.5 shows that the baseline profile of stable angina patients cannot be described by the word "dyslipidemia" alone. It simultaneously encompassed an atherogenic shift, inflammatory activation, increased lipid peroxidation, and weakened antioxidant reserve. On this basis, it is logical to proceed to an integral summary table of the main deviations and to correlation analysis.

1.5. Interrelationship of clinical-functional, lipid, and metabolic disorders

This section summarizes the leading deviations across daily ischemia, blood pressure, exercise tolerance, lipid profile, LPO–AOS, and NO exchange data. The statistical relationships among these parameters are then analyzed, forming the basis for subsequent assessment of therapeutic dynamics.

The integral summary of the main baseline deviations relative to control in patients with stable angina is presented in Table 1.9.

Table 1.9 Summary of the main baseline deviations relative to control in patients with stable angina

Indicator	Control	Range in groups II–V	Direction	p vs. control	Clinical interpretation
LDL-C, mmol/L	2.40±0.36	4.11–4.29	↑	p<0.001	Increased lipid load on the vascular wall
Apo-B, g/L	0.75±0.09	1.21–1.24	↑	p<0.001	Increased number of Apo-B-containing atherogenic particles
CRP, mg/L	0.91±0.38	3.41–3.52	↑	p<0.001	Presence of a low-grade inflammatory background
MDA, nmol/mL	16.6±2.0	30.1–32.5	↑	p<0.001	Activation of lipid peroxidation
SOD, arb. units/mg Hb	66.8±6.5	36.0–37.2	↓	p<0.001	Weakened enzymatic antioxidant defense
NOx, µmol/L	70.0±5.4	39.0–41.7	↓	p<0.001	Reduced NO-dependent vasodilator regulation
ST depression episodes, n/day	not recorded	1.63–1.88	↑	p<0.001	Documented daily ischemic load
VEM threshold power, W	120.4±12.7	77.1–79.9	↓	p<0.001	Reduced exercise tolerance
ABPM SBP, mmHg	125.7±8.0	149.4–151.6	↑	p<0.001	Increased hemodynamic load on the myocardium and vascular wall

According to the table data, LDL-C rose from 2.40±0.36 mmol/L in control to 4.11–4.29 mmol/L in groups II–V; Apo-B rose from 0.75±0.09 g/L to 1.21–1.24 g/L; and CRP rose from 0.91±0.38 mg/L to 3.41–3.52 mg/L. This triad alone forms the atherogenic-inflammatory framework of the baseline condition. A metabolic block was added to it: MDA rose from 16.6±2.0 to 30.1–32.5 nmol/mL, SOD fell from 66.8±6.5 to 36.0–37.2 arbitrary units/mg Hb, and NOx fell from 70.0±5.4 to 39.0–41.7 µmol/L. At the same time, daily ST depression episodes

— not recorded in control — reached 1.63–1.88 per day; VEM threshold power fell from 120.4±12.7 W to 77.1–79.9 W; and ABPM SBP rose from 125.7±8.0 mmHg to 149.4–151.6 mmHg.

Correlations among clinical, lipid, and metabolic indicators in patients with stable angina are presented in Table 1.10.

Table 1.10 Correlations among clinical, lipid, and metabolic indicators in patients with stable angina

Indicator pair	N	r	95% CI	p
LDL-C — MDA	120	0.41	[0.25; 0.55]	p<0.001
LDL-C — NOx	120	-0.47	[-0.60; -0.32]	p<0.001
MDA — NOx	120	-0.41	[-0.55; -0.25]	p<0.001
LDL-C — ST depth	120	0.34	[0.17; 0.49]	p<0.001
Apo-B — LDL-C	120	0.22	[0.05; 0.39]	p=0.014
CRP — MDA	120	0.28	[0.11; 0.44]	p=0.002

Note. Correlation coefficients calculated using Pearson's method.

According to Table 1.10, the strongest relationship among those presented was the inverse correlation between LDL-C and NOx: r=-0.47, 95% CI -0.60 to -0.32, p<0.001. By strength this falls into the moderate range. Its meaning is that patients with higher atherogenic cholesterol levels had lower total NO metabolites. Correlation alone does not establish causality, but it fits well with a model in which the atherogenic profile and endothelial regulation are interrelated.

The relationship between LDL-C and MDA was likewise moderate: r=0.41, 95% CI [0.25; 0.55], p<0.001. The higher the LDL-C level, the higher the recorded level of the final lipid peroxidation product. This statistically supports the pathophysiological sequence whereby a more pronounced atherogenic lipid load is accompanied by more pronounced oxidative stress. It does not directly prove oxidative modification of a specific LDL particle in a given patient, but the observed correlation is fully consistent with this hypothesis.

The correlation between MDA and NOx was inverse and of moderate strength: r=-0.41, 95% CI [-0.55; -0.25], p<0.001. Increased lipid peroxidation was accompanied by lower NOx levels. This finding links the prooxidant and endothelial analysis levels within a single model. In other words, patients with more pronounced oxidative stress also had a less favorable profile of NO-dependent regulation.

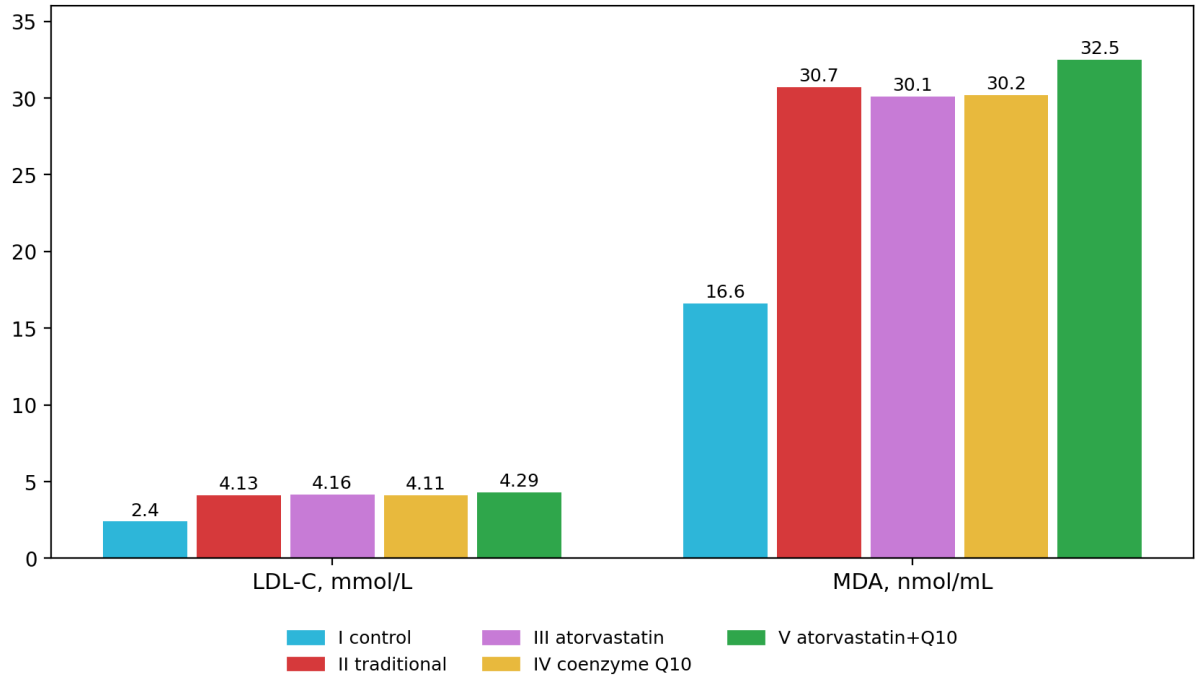
The relationship between LDL-C and ST depression depth was weaker but clinically notable: r=0.34, 95% CI [0.17; 0.49], p<0.001. This lies close to the lower boundary of a weak-to-moderate correlation. It cannot be interpreted to mean that elevated LDL-C by itself deepens ST depression. However, it does indicate that patients with a stronger atherogenic load tended to have a more severe functional ischemic response.

The relationship between Apo-B and LDL-C was the weakest among those presented: r=0.22, 95% CI [0.05; 0.39], p=0.014. Apo-B and LDL-C are related but do not describe identical aspects of risk: the former is closer to the number of atherogenic particles, the latter to the mass of transported cholesterol. The weak positive correlation underscores that a high LDL-C level does not always fully capture the burden of atherogenic particles, which justified including Apo-B in the analysis.

The correlation between CRP and MDA was r=0.28, 95% CI [0.11; 0.44], p=0.002. In strength this is weak but statistically robust. It indicates that, in this sample, inflammatory background and lipid peroxidation were not mechanistically identical but moved in a common direction. Such a relationship complements the baseline model

of stable angina as a condition in which atherogenic, inflammatory, and oxidative processes are not separable from one another.

Thus, in angina patients, the baseline deviations formed an interconnected system in which lipid profile, LPO–AOS, NO-dependent regulation, and functional ischemia were statistically coordinated. The visual comparison of mean group LDL-C and MDA values is presented in Fig. 1.6.



The control group was positioned in the zone of 2.40 mmol/L for LDL-C and 16.6 nmol/mL for MDA. Groups II–V shifted upward and to the right, forming a tight cluster with LDL-C values of 4.11–4.29 mmol/L and MDA values of 30.1–32.5 nmol/mL. The visual comparison of mean group MDA and NOx values is presented in Fig. 1.7.

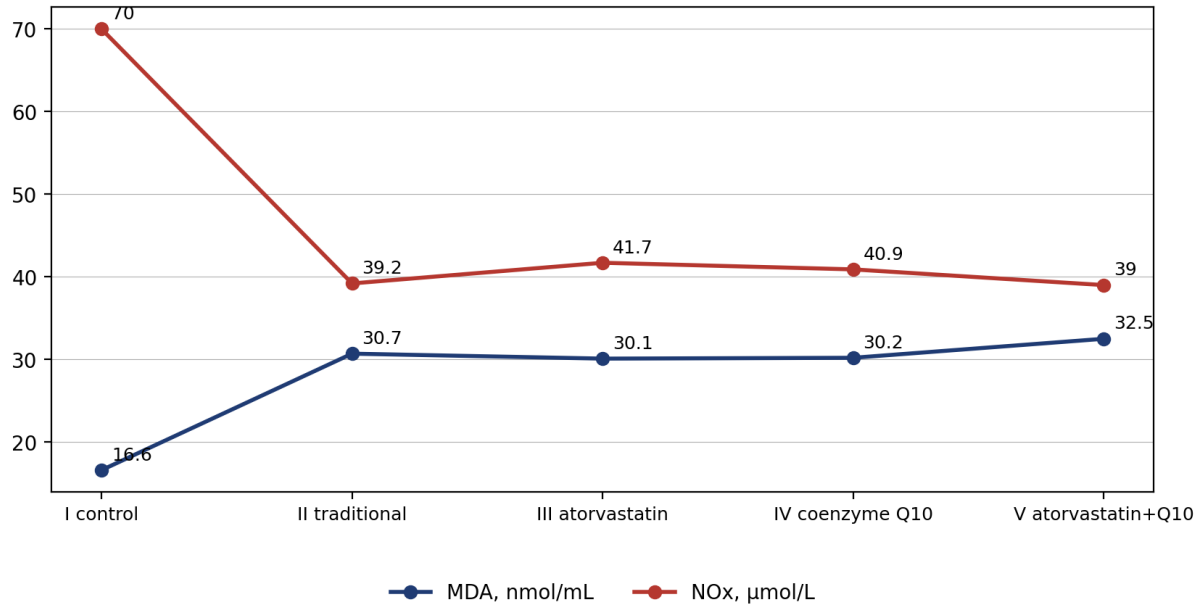


Fig. 1.7. Mean group values of MDA and NOx.

In the control group, MDA (16.6 nmol/mL) and NOx (70.0 μmol/L) appeared together. In groups II–V the picture reversed as in a mirror: at MDA levels of 30.1–32.5 nmol/mL, NOx fell to 39.0–41.7 μmol/L. The prooxidant shift is seen running alongside the decline in stable nitric oxide metabolites.

Conclusions

At baseline, patients with stable angina show clinical manifestations of the disease, exertional ischemia, and metabolic disturbances.

Increased lipid peroxidation is accompanied by reduced NO-dependent vascular regulation. This baseline characterization serves as the basis for the subsequent analysis of therapy's effect on the lipid, inflammatory, and metabolic contours of the disease.

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