

CONTENTS

1. INTRODUCTION	9
1.1 Historical notes	9
1.2 The aim of the work	11
1.3 List of abbreviations used	11
2. VENTRICULAR FUNCTION IN ACUTE MYOCARDIAL INFARCTION	17
2.1 Control of cardiac contraction	17
2.2 Control of blood circulation	22
2.3 Metabolic control of muscle contraction	25
2.4 Oxygen consumption of the myocardium and coronary blood flow	27
2.5 Myocardial ischemia, local and general response	30
2.6 The consequences of ischemia	33
2.7 The infarction phases	34
2.8 Pathogenesis of ventricular dysfunction	35
2.9 Clinical heart failure	39
3. HEMODYNAMIC INVESTIGATION	41
3.1 Invasive methods	41
3.1.1 Venous access	41
3.1.2 Measurement of intracardiac pressures	42
3.1.3 Measurement of cardiac output by thermodilution method	46
3.1.4 Measurement of blood pressure	48
3.1.5 Complications of cannulation and measurement	48
3.2 Other invasive methods	50
3.2.1 The examination of blood gases and the acid-base balance	50
3.2.2 Measurement of cardiac output by the Fick method	50
3.2.3 Auxiliary methods of measuring cardiac output	53
3.3 The parameters measured and their evaluation	54
3.3.1 Pressure values	54
3.3.2 Flows	61
3.3.3 Derived parameters	63
3.4 Non-invasive investigation	64
3.4.1 Clinical examination	64
3.4.2 X-ray examination	64
3.4.3 Systolic time intervals	67
3.4.4 Echocardiographic examination	68

4. ANALYSIS OF THE HEMODYNAMICS OF ACUTE MYOCARDIAL INFARCTION	71
4.1 Hemodynamic groups in AMI and differentiated therapy	71
4.2 Changes to the heart and their validity	77
4.2.1 Gallop	78
4.2.2 Heart size	80
4.2.3 Systolic time intervals	82
4.3 Changes to the lungs and their validity	86
4.3.1 Ventilation	87
4.3.2 Respiration	88
4.3.3 Rales	91
4.3.4 X-ray picture of pulmonary venostasis	93
4.4 The clinico-hemodynamic correlation of AMI groups	96
4.4.1 Killip classes and hemodynamics	96
4.4.2 The effect of the infarction site on the hemodynamics	101
4.4.3 The hemodynamics of vegetative reactions	104
4.4.4 The hemodynamics of arrhythmias	106
4.5 Structural complications in AMI	106
4.5.1 External myocardial ruptures	106
4.5.2 Internal ruptures	109
4.5.3 Pericarditis and pericardial tamponade	115
4.5.4 Infarct extension	116
4.5.5 Cardiac aneurysm	119
4.5.6 Pulmonary embolization and pulmonary infiltrates	120
5. DIFFERENTIATED THERAPY	121
5.1 Beta-lytics	122
5.2 Volume expansion	126
5.3 Furosemide	128
5.4 Positive inotropic drugs	130
5.4.1 Cardiac glycosides	130
5.4.2 Dobutamine	132
5.4.3 Vasopressors	133
5.5 Vasodilating drugs	133
5.5.1 Nitrates	136
5.5.2 Phentolamine	143
5.5.3 Calcium flux inhibitors	143
5.5.4 Prazosin	145
5.5.5 Captopril	146
6. PROGNOSIS	149
6.1 Clinical prognosis	150
6.2 Prognostic indices	156
6.3 Hemodynamic prognosis	156
6.4 Hemodynamic prognostic indices	159
7. SYNOPSIS OF HEMODYNAMIC MONITORING	161
7.1 Its significance	161
7.2 Indications	161
7.3 The therapeutic rationale of heart failure	163
7.4 Can hemodynamically differentiated treatment limit cardiac necrosis?	165
7.5 Prospects for the future	165
8. SUMMARY	167
9. SOUHRN	169
10. PERSPECTIVE	173
11. REFERENCES	175