## SHOCK

PATHOLOGY METABOLISM SHOCK CELL TREATMENT

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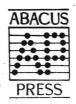
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#### PUBLISHER'S NOTE

This book was originally published in Romania in 1973 under the title Socul, with a Foreword by Mr Theodore Burghele, the President of the Academy of the Socialist Republic of Romania. The authors have been working as a team, under the guidance of the Professor of Surgery of the Bucharest University School of Medicine, Iuliu Suteu, for a number of years and this volume is a summary of their discoveries and the conclusions they have reached on the intricate problem of shock states. For the English edition, the original text has been completely revised and up-dated and additional illustrations have been provided.

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Shock has been recognized as a clinical syndrome for almost 2000 years, but today it is still a controversial phenomenon in the medical world. In the historical and geographical universe man is an infinitesimal entity subject to the action of a complexity of endo- and exogenous factors — the 'environmental pressure'. Contemporary life contains a great variety of harmful factors, of varied physical, chemical and/or biological origin that are the potential generators of critical situations, of true states of shock, from the viewpoint of medical pathology.

Conditioned by the outstanding technical progress in all fields, these deleterious factors increase in numbers and intensity at an unprecedented rate; their action, however, may be avoided, limited, prevented or removed. Although vast programmes of prevention have been drawn up, the world statistics communicated at the first International Symposium on accidents causing shock, held in Washington in 1970, gave impressive figures referring to trauma. Today it is known that one of ten inhabitants of the earth suffers yearly an injury that will necessitate several days hospitalisation, that two-thirds of the cases are youths, and that the care of a casualty with multiple injuries — in shock — demands a surgical intervention that often exceeds the complexity of cardiac surgery and transplant surgery; nevertheless, the organisation of centres of investigation and treatment of cases of severe trauma and shock is only in its initial stage in many countries. Among the present ecologic accidents is the 'trauma epidemic' which, unfortunately, occupies an important place.

Resulting from the multiple conflicts between human beings and the harmful factors of their environment, the world morbidity rate for trauma is greater than the morbidity rate for cardiovascular diseases, and four times that for cancer.

Apart from patients suffering from severe shock with acute functional imbalance, many are brought to operation or to intensive care units suffering from chronic diseases which result in severe functional and morphologic disturbances, and which justify the present notion of 'chronic shock'.

As a rule, in the clinic, a diagnosis of shock seems to be comparatively simple: cold or moist skin, arterial blood pressure below 80 mmHg, cardiac

index less than 2500 ml/min/m², urinary output less than 25 ml/hour and arterial standard bicarbonate below 2 mEq. But these criteria may become rigid and, therefore, useless if not followed up functionally. In point of fact, understanding of the complex phenomenology of shock implies a deeprooted knowledge of the pathophysiology of shock and its fluctuating course.

When shock was conceived as exclusively haemodynamic, systemic arterial pressure served as a guide for the clinical stages and the therapeutic approach; later, when shock was understood at rheologic level, the criteria for assessing the stage of shock became the lowering of the effective circulating blood volume and perfusion of the microcirculation. The notion of 'shock viscus' was coined (shock liver, kidney, intestine, lung, etc.). In 1935, Alexis Carrel stated that 'Man is the hardiest of all animals, . . . however, our organs are fragile. They are damaged by the slightest shock. They disintegrate as soon as blood circulation stops'.

The next stage in our understanding of the phenomenology of shock 'descended' to the intimacy of the cellular enzymatic chains; for a long time lactic acidosis came to be considered the most reliable prognostic marker of the severity of shock.

Today we have gone beyond this stage. The pathobiochemical and pathobiophysical events in the course of shock are analysed at molecular and atomic level, where the very logic of the cybernetic and thermodynamic organization of the human body is actually disturbed. Therefore, considering the cell as the lesional site where the main endogenous antishock struggle takes place in the macrosystem, in writing this book we considered that the notion of shock cell offers the best basis for studying the disturbances produced by shock down to molecular level, particularly as Science in the last decade has furnished an avalanche of data useful for our understanding of the biologic occurrences. The thermodynamic approach to shock was possible by separate consideration and then integration in the behaviour of the energy subsystems in the shock cell. The characteristics of the shock cell also allow for systematization of the governing and control relationships between the cellular space and the extracellular space.

Most of the studies of the last few years deal with the cellular metabolic response to shock, and monitoring of the biochemical parameters most reliable for assessing the severity of shock. Monitoring has resulted in determination of flow parameters whe variations permit the institution of a prompt replacement of the fluid lost. It has become obvious that the hydroelectrolytic reaction to shock does not consist only in a simple loss of intravascular effective circulating fluid; studies have likewise been carried out on the role of the interstitium and lymphatics in shock, on crystalloids that are now being substituted for colloids for making up volume losses, especially in the first phases of shock. The first adverse responses to dextrans, mannitol, diuretics, etc. are recorded, and the much discussed electrolytic solutions have finally proved extremely useful, permitting re-equilibration of the interstitial space and decrease in the number of perfusions and, therefore, of the untoward post-transfusional reactions.

The reactions of the human body to shock are still imperfect and incomplete, a true shock-inducing agent always being a surprisingly intense pathogenic stimulus to which our homeostatic systems have not yet become fully adapted, and are not yet 'phylogenetically' aware. Therefore, the immediate endogenic measures the body takes, known at present as the reactional syndrome, are restricted by the inherent sacrifices this demands. Otherwise stated, the human body is at first panicstricken and tries to save itself by reacting over-hastily and, as a result, a certain disorder ensues which may sometimes in itself be fatal.

Prolonged selective vasoconstriction may give rise to irreparable damage in vital areas, among which the lung, today considered to be in man the most sensitive target organ of hypotension, hypovolaemia and septic states. On the other hand, enzymatic hyperglycolysis endeavours to supply energy to the shock cell, loading it, however, with protons up to the dangerous level of

hydrolase autodigestion.

Almost all the occurrences with an immediate adaptive trend that appear in shock are of disproportionate intensity and duration, impeding the therapy that has at times to attenuate or stimulate these same occurrences,

depending on the state of shock.

The alpha effect of catecholamines is at first useful both haemodynamically and metabolically (proven by selective blocking with bretilium tossilate of norepinephrine release which has never improved but, on the contrary, aggravated shock), but an excess of alpha stimulation is harmful if it persists in certain areas. Thus,  $\alpha$ -lytic drugs have gained favour in the therapy of shock but they do not yet occupy an important place because excessive dilatation of the microcirculation, which they bring about, may be dangerous and difficult to control.

In order to emphasize the uncertainty of the therapeutical measures taken today it is sufficient to recall that some authors recommend  $\beta$ -stimulating drugs (especially in cardiogenic and bacteraemic shock) and others use  $\beta$ -blocking agents (especially for the pulmonary protection they offer). The favourable action of catecholamine precursors in the late phases of shock is likewise well known, while  $\alpha$ -stimulants are administered with great care in bacteraemic shock because of their antiphlogistic effect.

Although of bewildering aetiologies, the numerous types of shock encountered in the clinic have a common pathophysiological centre whose segments fall into a fundamental and characteristic biological pattern.

It is almost unanimously recognized today that shock has a multiphase, oscillating development, but the moment of transition from one phase to another is difficult to establish clinically. These phases are, however, of great practical importance since they at times impose paradoxical changes in the treatment applied which must follow closely in the wake of the progressive waves of shock and exercise a continuous intracellular action sustaining the enzymatic and membraneous apparatus.

The oscillating course of shock is also unpredictable, differing entirely from the usual systemic reaction of the after-effects of an injury; hence,

in its dynamics, shock often reaches the borderline of irreversibility over

which it will pass in the absence of adequate treatment.

It is not yet known for certain, especially at clinical level, which are the limits of the reversible-irreversible; it appears certain, however, that this point is decided at the level of the shock cell, long before the terminal clinical signs appear. In the intimacy of cellular metabolism certain transformations occur a long while before they become clinically apparent; therefore the clinician must be permanently aware of the state of shock at enzymatic level and that of the functional reflexes of nucleic acids, so that the intracellular introduction of each therapeutical gesture must be borne in mind. The clinic thus acquires a deeper perspective, without losing its fascinating facet, an insight into the intracellular source of life, the old term of 'clinical picture' taking on three dimensions and becoming a 'clinical relief'.

The surgeon of today is almost always aware of the pathophysiological axis of shock, and faces, together with the anaesthetist-resuscitator, problems of tissular hypoperfusion and disturbances in the homeostasis of the human

body at every step.

'The modern surgeon must be acquainted with surgical pathophysiology', asserts Prof. Th. Burghele, recalling at the same time the classical condition announced by René Leriche: '... for the sake of quality the surgeon of today must rise from the rank of anatomist to the dignity of physiologist'.

The chapters deal with different aspects in the study of shock and may therefore seem to be unbalanced. The themes contained in previous well-known works may only be outlined or merely mentioned in some chapters, or dealt with in detail in others and then taken up again in further chapters where the subject of shock was deemed to be insufficiently treated or needing to be brought up to date.

The reader will not find in this book precise instructions but only principles concerning the investigation and therapy of each type of shock. The great variety of clinical aspects calls for individual consideration of each particular case; hence we have avoided any attempt at a schematical repre-

sentation.

This is not a book on clinical practice and treatment of shock but a discussion on the pathophysiology of shock, based on the biological perspective of this phenomenon and with additional new data on biochemical, hormonal, genetic, cybernetic, rheodynamic and thermodynamic aspects with an attempt at bringing into relief some of the facets that until recently have been neglected.

To those who will perhaps regret that certain facts or ideas have not been sufficiently emphasized we answer by quoting Montesquieu: 'When treating a subject it is not necessary to exhaust it; it is sometimes sufficient

to draw attention to it'.

Long-term investigation of a complex biological phenomenon such as shock calls for team work and presupposes the cooperation of numerous

specialists. The authors of this volume undertook the role of initiating the experimental models and of promoting their implementation; they also made the decision to combine experimental findings with clinical experience and, especially, to take on the responsibility of gathering together suitable material about shock, which by its very nature is composed of diverse elements.

The studies were carried out in Bucharest in the Department of Experimental Medicine of the Central Military Hospital (Dr T. Giurgiu) and the Department of Experimental Nuclear Medicine of the Fundeni Clinical Hospital (Dr O. Cavulea). Histologic examinations were performed and interpreted in the Laboratory of Pathologic Morphology of the Central Military Hospital (Dr I. Strîmbeanu). The biochemical constants and plasma enzymes were determined in the Laboratory of Biochemistry of the same hospital (pharmacist Dr C. Apreotesei) and haematologic studies in the Center of Haematology of the Armed Forces (Dr C. Iercan and Dr R. Perlea) and the Laboratory of Haematology of the Central Military Hospital (Dr C. Satmari).

We wish to express our gratitude to all who have participated and contributed so generously in the multiple themes required in the study of shock; we likewise acknowledge our grateful thanks to doctors D. Singer, P. Ionescu, D. Bărboi, T. Safta, A. Verdeş and a numerous group of enthusiasts without whose practical aid and advice our endeavours would have been

inadequate.

To our colleagues who by their work and skill down the years have made of our team a closely knit working unit, we owe more than a debt, for this volume belongs to them, too, to an equal extent.

Our most cordial thanks are due to Prof. Ion Teodorescu-Exarcu for

his outstanding help, comments and advice.

We cannot say how deeply indebted we are and how much we value the work of our colleagues in the field of medicine, whatever their specialty, who meet at the patients bedsides to allay their daily suffering by understanding, self-denial and professional integrity.

The editors of the Military Publishing House have always been solicitous and have constantly shown a keen interest in medical publications; thanks to them transforming a rough manuscript into a published book became a

pleasant work of collaboration.

THE AUTHORS

Bucharest, December, 1976.

### ABBREVIATIONS

AB	actual bicarbonate	DIC	disseminated intravascular
ACTH	adrenocorticotrophic	,	coagulation
· 'g	hormone	DNA	deoxyribonucleic acid
ADH	antidiuretic hermone	DOCA	deoxy-11-corticosterone
ADP	adenosine diphosphoric	1.1 DDC	acetate
AND	acid	1,3-DPG	1,3-diphosphoglycerate
AMP	adenosine monophosphoric	1,2-DPG .	1,2-diphosphoglycerate
ARAS	ascending reticular	ECBY	effective circulating blood volume
	activating system	ECG	electrocardiogram
ARF	acute renal failure	EM	
ATP	adenosine triphosphoric	EMK	Embden-Meyerhof cycle
ATP-ase	adenosine triphosphatase	ENIK	Embden-Meyerhof-Krebs cycle
AV shunt	arteriolo-venular shunt		cycle
AV Shunt	arteriolo-venular shufft	FA	acid phosphorylase
BB	buffer bases	FAC	acid phosphatase
BE	base excess	FAD	flavinadeninedinucleotide
BP	blood pressure	FFA	free fatty acids
CI	cardiac index	FFAC	free fatty acids cycle (Lynen)
cAMP	cyclic AMP (3', 5', AMP)	FSH	follicular stimulating
Cit a, b, c, etc.	cytochrome a, b, c, etc.		hormone
CITOX	cytochrome-oxidase	FSRH	follicular stimulating
CNS	central nervous system		releasing hormone
CO	cardiac output	FT	total phosphorylase
CoA	coenzyme A	FWC	free water clearance
CO <sub>2 total</sub>	total carbon dioxide	F-6-P	fructose-6-phosphate
COMT	catechol-ortho-methyl- transferase	F-1,6-DP(FDP)	fructose-1,6-diphosphate
CRF	corticotrophic releasing	GABA	gamma amino-butyric acid
	factor (see CRH)	GFR	glomerular filtration rate
CRH	corticotrophic releasing	GH (see STH)	growth hormone
0.00 1	hormone	GOT	glutamic oxalacetic
	and a series	GOI	Bluttillic Ondiacette
C <sub>2</sub> SC <sub>0</sub> A	active acetate	GOT	transaminase
CSF	cerebrospinal fluid	GPT	transaminase glutamic pyruvic
_ ~	decine stati rate describito	GPT	transaminase glutamic pyruvic transaminase
CSF	cerebrospinal fluid central venous pressure dihydroxyacetone	GPT GRH	transaminase glutamic pyruvic transaminase growth releasing hormone
CSF CVP	cerebrospinal fluid central venous pressure	GPT	transaminase glutamic pyruvic transaminase

GSSG	dehydrogenated glutathione	PIOR	postinjury oscillating reaction
G-1-P	glucose-1-phosphate (Cori	PISS	postinjury systemic
G-6-P	ester) glucose-6-phosphate	PEP	syndrome phosphoenolpyruvate
G-0-P	(Robison ester)	PFK	phosphofructokinase
G-6-Pase (G-6-PA)	glucose-6-phosphatase	PGDH	phosphoglyceraldehyde-
G-6-PDH	glucose-6-phosphate-		dehydrogenase
	dehydrogenase	PVP	polyvinyl pyrrolidon
НЬ	haemoglobin	$PCO_2$	partial carbon dioxide
HbO <sub>a</sub>	oxyhaemoglobin	P.CO	pressure
HES	hydroxyethyl starch	$P_aCO_2$	partial (arterial) carbon- dioxide pressure
Ht	haematocrite	$P_aO_2$	partial (arterial) oxygen pressure
ICP	intracranial pressure	$P_vO_2$	partial (venous) oxygen
IDH	isocitric-dehydrogenase		pressure
IgM (IgG, IgE, etc.)	immunoglobulin M, G, E, etc.	$P_vCO_2$	partial (venous) carbon di- oxide pressure
ICSH	islet cell stimulating	$P_AO_2$	partial (alveolar) oxygen pressure
	,	$P_ACO_2$	partial (alveolar) carbon
$\mathbf{K}_{\mathbf{i}}$	intracellular potassium		dioxide pressure
$K_e$	extracellular potassium	$pH_a$	actual pH
LDH	lactic-dehydrogenase	RBC	red blood cell
LH	luteinizing hormone	RBF	renal blood flow
LPEP	leucinaminopeptidase	RES	reticuloendothelial system
LRH	luteinizing releasing	RF(RH)	releasing factor (releasing hormone)
LTH	luteotrophic hormone	RI	respiratory index
		RNA	ribonucleic acid
MAO	monoamino-oxidase	SB	standard bicarbonate
MDF	myocardial depressor factor	SDH	succinic-dehydrogenase
MDH	malic-dehydrogenase	SFEZA	sulphatase
7	,	STH (see GH)	somatotrophic hormone
NAD	nicotinamide adenine dinucleotide	77	and the Marketon and the
NADP	nicotinamide adenine	T <sub>3</sub>	triiodothyronine thyroxine
,	dinucleotide phosphate	T <sub>4</sub> THAM (TRIS)	trihydroxymethylamino-
Nai	intracellular sodium	THAD (TRIS)	·methane
Nae	extracellular sodium	TPR	total peripheral resistance
OAA	oxaloacetic acid	TRF	thyrotropic releasing factor
O	oxidative phosphorylation	TRH	thyrotropic releasing
OPC	oxidative phosphorylation		hormone
	chain	TSH '	thyrostimulating hormone
PAOR	postaggressive oscillating reaction	UO	urinary output
PASR	postaggressive systemic	VDM	vasodilatator material
	reaction	VEM	vasoexcitator material

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