CHANGES IN CARDIAC OUTPUT IN THE ACUTE PERIOD OF CLOSED CHEST INJURY

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Experiments on dogs showed that a combination of external blood loss (20 ml/kg) and injury to the thoracic cage causes a clear decrease in pumping function of the heart, terminating in some cases in acute cardiac failure and death of the animals. If the adaptive reactions of the heart and of the body as a whole are maintained, after a period of temporary depression of the myocardium, gradual normalization of the pumping function took place with restoration of homeostasis, even without any special treatment.

KEY WORDS: closed chest injury; cardiac output; cardiac failure; disturbance of gas exchange.

The mechanisms of death after severe closed chest injuries, which are so frequently fatal, have not been adequately studied. In 50-70% of patients affected, this type of trauma causes multiple fractures of the ribs, accompanied by a varied degree of blood loss [1, 4, 8]. However, the influence of these factors on the circulation and respiration calls for more precise explanation. Exposure of the body simultaneously to a combination of pathogenic factors of varied intensity is an obstacle to investigations of this type under clinical conditions. Accordingly the authors decided to undertake an experimental study with the aim of determining the character of the effect of artificially reproduced components of chest trauma, of standard intensity, on cardiorespiratory function: blood ions, disturbance of the integrity of the thoracic cage, and also a combination of both [5].

EXPERIMENTAL METHOD

Experiments were carried out on 43 dogs weighing 6-23 kg in which the arterial pressure (BP), pulmonary arterial pressure (PAP), and heart rate (HR) were recorded through implanted catheters. The cardiac output was determined by the thermodilution method, and the cardiac and stroke indices (CI and SI), the mean central circulation time (MCCT), and total pulmonary resistance (TPR) were calculated; the circulating blood volume (CBV) was determined by the dye dilution method with Evans' blue. The pulmonary ventilation was estimated by spirography (SG-1 spirograph), with measurement of the respiratory volume (RV), respiration rate (RR), and respiratory minute volume (RMV). The efficiency of the pulmonary gas exchange was investigated by measuring the partial pressure of oxygen and carbon dioxide in the arterial blood (p_aO_2 and p_aCO_2) and the acid-base balance was determined by measuring the pH and BE of the blood by means of an Astrup micro-gas analyzer.

Altogether three series of experiments were carried out, in which the traumatic factors (TF_1, TF_2, TF_3) were external blood loss amounting to 20 ml/kg (series 1, 13 dogs), displacement of a fragment of the thoracic cage prepared beforehand (series 2, 14 dogs), and a combination of these procedures (series 3, 16 dogs). The data were recorded initially and at intervals thereafter: hourly for the first 4 h after trauma, then once a day for the next 7 days. The results were subjected to statistical analysis by Student's method. The significance of differences (P) was determined by comparing each value of the parameters with its initial value, and the results are given in the tables with a level of probability of $P<0.05$.

EXPERIMENTAL RESULTS AND DISCUSSION

The most significant feature of TF_1 was a decrease in the volume indices of the circula-

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TABLE 1. Changes in Indices of Hemodynamics, External Respiration, and Acid–Base Balance during Measured Blood Loss and Injury to Thoracic Cage (M±m)

Parameter	Injury to thoracic cage in dogs							Blood loss 20 ml/kg			
	initial value (n = 14)	animals which died (n = 8)			animals which survived (n = 6)			initial value (n = 13)	1st hour	2nd-4th hours	1st-2nd days
		1st-4th hour	1st-2nd days	3rd-7th days (death)	1st-4th hour	1st-2nd days	3rd-7th days				
CI, ml/min · kg	124±20,01	102±13,08	108±13,41	116±15,21	86±38,11	103±11,23	124±54,31	143±8,71	116±4,11	147±4,21	141±1,05
HR, beats/min	145±10,01	117±15,12	120±10,71	117±15,31	162±8,42	145±7,21	159±6,52	129±7,91	164±14,31	164±11,85	139±11,21
SI, ml/kg	1,06±0,09	0,70*±0,09	0,77*±0,05	0,78±0,14	0,82±0,18	0,88±0,04	0,83±0,05	1,31±0,12	0,63*±0,09	0,89±0,03	1,08±0,04
CBV ml/kg	94±12,00	153*±8,52	84±16,92	—	82±18,12	72±11,21	—	76±5,86	56*±2,23	75±13,71	65±9,65
Systolic BP, mm Hg	152±4,95	152±3,59	116*±16,9	146±10,7	143±6,32	150±7,31	149±9,42	149±5,47	154±11,39	132±7,82	138±11,42
TPR, dynes · sec · cm^{-5}	699±68,56	903±80,52	683±103,93	779±117,12	—	957±63,18	—	597±114,11	906±182,21	774±156,14	652±61,32
MCCT, sec	5,1±0,18	5,3±0,52	5,4±0,34	6,6±2,28	5,7±0,86	5,0±0,33	7,0±1,20	4,8±0,64	5,7±0,82	7,0±0,88	6,5±0,73
BR, breaths/min	22±3,12	29±2,71	22±3,29	34±5,34	51*±2,24	20±3,00	19±1,89	23±3,18	33±5,44	38±10,61	18±5,41
RV, ml/kg	11±1,02	10±1,12	14*±0,42	13±2,82	12±1,75	16*±1,56	14±0,82	12±1,69	13±1,23	12±3,23	15±2,56
RMV, ml/min·kg	281±31,70	280±21,13	279±24,29	452*±72,86	632*±50,01	324±50,01	266±41,20	276±35,52	430±56,21	456±138,10	270±50,01
pH	7,44±0,02	7,38±0,02	7,31±0,07	7,38±0,03	7,43±0,01	7,43±0,03	7,36*±0,01	7,36*±0,01	7,31*±0,02	7,32*±0,02	7,38±0,04
BE meq/liter	−1,9±0,91	−6,8*±0,51	−9,5*±2,00	−7,7*±1,14	−3,5±1,16	−2,5±0,87	−5,3±0,81	−3,6±1,45	−8,2±1,77	−6,0±1,00	−0,5±0,43
PaO_2, mm Hg	85±2,80	88±4,35	70*±2,75	54*±1,97	74±1,57	83±2,21	81±3,42	85±2,17	79±2,62	77±4,04	84±2,05
$PaCO_2$, mm Hg	37±2,41	37±4,42	33±3,33	26±5,28	34±2,10	34±1,12	32±1,52	34±3,86	—	31±2,52	25*±1,78

Legend. Here and in Table 2, values differing statistically significantly from initial value marked by asterisk (P<0.05). Number of animals given in parentheses.

TABLE 2. Changes in Indices of Hemodynamics, External Respiration, and Acid–Base Balance after a Combination of Measured Blood Loss and Injury to Thoracic Cage (M±m)

Parameter	Initial value (n = 16)	Animals which died (n = 10)				Animals which survived (n = 6)		
		1st-4th hour	1st-2nd day	Day of death		1st-4th day	1st-2nd day	3rd-7th days
				after 2-6 h	after 1 hour			
CI, ml/min · kg	151±12,52	105*±17,01	151±13,10	93*±15,21	61*±7,23	120±11,31	165±10,33	180±10,42
HR, beats/min	125±15,00	150±7,95	173*±10,31	164±15,22	158±17,80	194*±13,01	158±7,76	162*±13,41
SI, ml/kg	1,32±0,06	0,72*±0,16	0,85*±0,09	0,68*±0,05	0,45*±0,04	0,63*±0,07	0,94*±0,13	1,00±0,22
CBV, ml/kg	84±5,16	46±19,01	57*±8,00	91±5,61	69±6,82	77±5,02	75±6,24	75±15,43
Systolic BP, mm Hg	153±5,25	130*±5,12	132±8,21	128±8,81	93*±18,00	135±5,42	134±8,43	136±11,32
TPR, dynes · sec · cm^{-5}	531±40,10	630±103,11	639±119,2	715±107,2	1436±142,21	640±110,11	470±50,98	505±25,54
MCCT, sec	5,2±0,27	7,1*±0,52	5,3±0,65	9,0*±1,81	11,1*±0,66	5,5±1,89	4,6±0,68	4,2*±0,21
BR, breaths/min	23±3,21	38*±3,66	16*±1,03	45*±5,61	35±10,72	31±1,95	35±4,23	37±7,65
RV, ml/kg	14±0,81	11,7*±0,61	14,4±1,23	10,7*±0,45	15,5±0,35	14,3±1,16	11,8*±0,46	11,4±1,05
RMV, ml·min·kg	346±56,60	400±54,71	241±30,82	474±117,11	542±157,21	430±12,25	396±34,12	422±54,82
pH	7,36±0,01	7,24*±0,03	7,36±0,02	7,35±0,02	7,29*±0,01	7,38±0,06	7,36±0,02	7,36±0,18
BE, meq/liter	−3,10±0,59	−10*±0,89	−8,2*±0,61	−8,8*±0,77	−15*±0,99	−8,3±2,35	−7,74*±1,43	−6,6±2,15
p_aO_2, mm Hg	87±2,78	78±6,80	84±3,39	71*±4,57	86±7,63	75±7,13	82±4,07	87±4,92
$Pa\ CO_2$, mm Hg	35±2,91	32±3,23	28±3,36	23*±3,00	27±2,44	36±4,87	35±5,11	35±4,09

tion, accompanied by an increase in HR, TPR, MCCT, and respiratory function, and by a tendency toward arterial hypoxemia and metabolic acidosis (Table 1). However, after only 2-4 h an improvement was observed in most of these parameters, and they returned completely to normal after 24 h.

Under the conditions of TF_2 (Table 1) against the background of considerable and progressive arterial hypoxemia a tendency was noted for SI to fall, for MCCT to be slowed, and for RR and RMV to rise; these changes were most marked toward the time of death of the animals, which took place on average after 99.5 h. In the group of surviving animals of this series (six dogs) the values of p_aO_2 showed a reversed trend, and there was also a tendency for CI to exceed its initial level, chiefly on account of an increase in HR.

Under TF_3 conditions (Table 2) a marked decrease was observed in SI, CI, BP, and CBV, accompanied by an increase in HR, RR, RMV, TPR, MCCT, and BE. Later, despite restoration of CBV, this trend of changes in the parameters persisted and ended with death of the animals on average after 27.5 h. In the group of surviving animals (six dogs), a temporary decrease in the volume indices of the circulation and stimulation of respiratory function was followed by a period of gradual normalization of most of the indices measured. The most significant feature was the reverse trend in SI and CI, recovery of which was accompanied by a fall in HR.

In all series of experiments, the changes in cardiac output were most significant. However, the nature of these disturbances differed in each series of experiments. Whereas under TF_1 conditions the decrease in cardiac output was transient and due mainly to the deficiency of CBV, in TF_2 and, in particular, in TF_3 signs of acutely increasing heart failure appeared more and more conspicuously, with the appearance first of all of a decrease in SI, then a decrease in CI, and an increase in MCCT. The degree of severity of the disturbances in cardiac function arising in the different series corresponded to differences in the mean length of survival of the animals: maximal in the 1st and minimal in the 3rd series of experiments. An essential role in the genesis of heart failure in the 2nd series could have been played by the marked arterial hypoxemia. However, in a combination of TF_1 and TF_2 heart failure developed in the absence not only of a deficiency of CBV, but also of hypoxemia, thus indicating a special character of development of depression of the myocardium under these conditions. At a certain stage the increase in TPR and in metabolic acidosis which, according to some evidence [3, 6, 7], can depress cardiac function, may have made a contribution to the development of cardiac failure. The appearance of profound arterial hypocapnia, impeding dissociation of oxyhemoglobin in the tissues [2] must also be reflected in the working capacity of the myocardium under extremal conditions.

Meanwhile analysis of the results reveals several mechanisms of compensation of disturbances of cardiac activity, among which may be included strengthening of respiratory function and an increase in CBV, leading to elevation of p_aO_2, improvement of coordination of ventilation–perfusion relations, an increase in HR, and so on. Marked depression of cardiac function and activation of an extensive series of protective and adaptive reactions distinguished the pathological state under TF_3 conditions in both essence and form from those in the control experiments. The degree of preservation of protective reactions in the body as a whole and, in particular, in the heart determined temporary stabilization of the state of the animals which died and complete recovery of homeostasis in those which survived.

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