HUMAN FACTORS IN DESIGN OF MILITARY AIRCRAFTS' OXYGEN SUPPLY EQUIPMENT

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INTRODUCTION

In view of the vital importance of oxygen supply equipment to protect pilots from hypoxia, and the interaction among the aircrafts' performance, the protective function and human ability, the oxygen supply equipment must be designed on the basis of human factors. It is a big challenge for the physiologists to optimize them to the tactical need.

This paper is to discuss some human factors in the development of oxygen supply system in order to make the views of engineers and physiologists identical both in theory and practice.

1. The altitude intrapulmonary absolute pressure of oxygen supply

The altitude intrapulmonary absolute pressure of oxygen supply is associated with three parts as mentioned above, we should not neglect any party. Although the development of the pressur ecabin has virtually eliminated many of the problems associated with high altitude flights, the possibility of a structural failure resulting in a rapid, accidental loss of cabin pressure has introduced a new potential hazard for the pilots. The pressure oxygen supplied is the only means to protect against hypoxia at high altitudes. However, positive pressure breathing raising the intrapumonary pressure, reduces the effective blood volume and induces respiratory disturbances. This effect could only be reduced by application of counter pressure to both the upper and lower limbs with the capstan pressure suit. However, inorder to make the pilots' personal equipment simplified, the "vest-anti-G-suit"was used as a means of providing short duration of protection against the effects of loss of cabin prssurization at high altitudes. Since its protection is limited, pressure breathing of higher absolute intrapumonary pressure brings about the disturbances upon the cardiopulmonary system. The interaction between the hypoxia and the circulatory load imposed by positive prssure breathing was studied in order to determine the most satisfactory compromise between the positive pressure breathing and absolute intrapulmonary pressure for adequate protection at altitude 18,000 m.

In our study, various degrees of hypoxia and positive pressure brathing were achieved by three different absolute intrapumonary pressure tests both on ground and at high altitude 18,000 m. Eight healthy young men participated the experiments. It was found that the tolerance time , the reason to suspend the test and the physiological indices were all mainly determined by the intrapulmonary pressures applied . The subjects had had cardiac and pulmonary disturbances with the decrease in cardiac output and effective circulatory blood volume when a " vest-anti-G-suit"was used under higher absolute pressure group(tables1, 2). Since blood was moved from thorax into limibs and alveoli are expanded, the alveolar ventilation perfusion ratio is changed, and the oxygenation saturation also changed. All the effects were lighter as long as the absolute pressure was lower than 15.3KPa, owing to the alleviation of positive pressure stress.

To sum up, with a simplified counterpressure protection, PPB is the principal factor compromising the human tolerance. The higher the positive pressure, the more obvious the circulatory stagnant hypoxia is. Based on these data, an intrapumonary pressure of 15.3kPa is recommended.

2. The onset time of emergency pressuring oxygen following the decompression

The onset time of emergency pressuring oxygen following decompressionis required within 5 seconds at above 12km. It is wanted to make it quicker than 1.0 second following cabin failure. However, weather it is the quicker, the better is needed studying. Various onset time(from 0.4s to 15s) of emergency pressuring oxygen following the decompression were observed in the altitude rapid decompression test. The results showed that the onset time of emergency pressuring oxygen following decompression has some influence upon the cardiopulmonary system. With various onset time of emergency pressuring breathing with oxygen following decompression some different reactions of the subjects have been observed at same altitude, with the same

table 1 The tolerant time and the cause of ceased test

absolute pressure (kPa)	subjects (n)	tolerant time (second)	the cause of ceased test(n)			
			A	B	C	D
16.0	8	277.0±38.0	7	2	1	1
15.3	5	264.0±26.0	1	1	1	3
14.7	12	207.0±11.2	0	0	2	5
15.3*	5	175.0± 4.0	0	2	0	2
14.7*	3	135.0±25.5	0	0	2	3

* the results of the altitude chamber experiment.
A dyspnea, B doubled HR, C SaO_2 <70%, D EEG slow wave.

Table 2 The cardiovascular response in pressure breathing test

absolute pressure (kPa)	subjects (n)	Hr (beats/min)	caediac output	
			SV	CO
before test		72.4±2.5	92.5±4.9	6.19±0.4
16.0	8	117.6±8.8	54.9±5.4	5.61±0.5
15.3	5	107.4±3.4	71.5±2.2	7.20±0.9
14.7	12	108.6±7.3	69.8±2.6	6.68±0.8
15.3*	5	131.3±4.7	54.8±4.3	6.64±0.5
14.7*	3	119.0±7.8	54.5±3.5	6.22±0.8

* the results of the altitude chanber experiment.

Table3. The physiological reactions in 16km decompression experiments

Compression starting time(s)	Max. HR* (beats/min)	Min. Sao_2 (%)	EEG (Hz)	Respiritory rate(min^{-1})	Final results
15.0	130	77.5	8～10	13.5	success
1.5	133	75.0	8～11	12.0	success
0.4	130*	90.0	2～ 3	...	success

HR=heart rate; EEG=electroencephalogram; Sao_2=arterial oxygen saturation.
* theheart rate declined suddenly from 130/min to 70/min followed by arrhythmia 30s after ascending to 16km. # some slow waves(2～3Hz) appeared at 3s afteremergency descending.

Table 4 The effects in PAO_2 in the fulminating hypoxia tests(mmHg)

group (N=8)	before test	breathing rich O_2 1min	3min	5min	exposed to low oxygen(0.8%) 1s	5s	10s	15s	20s	25s	30s
1	137	119	119	119	119	118	91	26	21	21	59
2	139	133	135	136	138	137	79	50	25	25	72
3	141	167	168	167	168	167	86	34	34	35	98
4	137	172	175	174	173	173	122	37	37	36	86
5	141	231	236	238	239	238	164	86	53	68	163
6	143	254	264	265	265	204	173	62	61	83	162
F	2.30	146.33	151.93	153.20	134.08	142.3	2.9	7.5	9.6	10.5	6.49
P		*	*	*	*	*	*	*	*	*	*

* significant differences $P < 0.05$.

Table5 SaO_2 changes in the fulminating hypoxia tests

group (n=8)	before test	breathing rich oxygen gases 1min	3min	5min	exposed to 0.8% hypoxia gas 1s	5s	10s	15s	20s	25s	30s	min
1	97	96	96	95	94	93	88	83	85	90	92	81.5
2	97	97	97	97	96	95	91	87	87	91	93	85.7
3	98	97	98	98	97	96	93	90	92	95	96	89.7
4	97	98	98	98	98	98	97	96	96	97	97	94.7
5	97	98	99	99	98	98	97	97	97	97	98	96.1
6	98	98	99	99	98	99	98	98	98	98	98	97.5
F	0.5	7.1	15.5	38.5	13.5	12	13	25	25	21	22	23
P		*	*	*	*	*	*	*	*	*	*	*

Table 6 The changes of finger SaO_2 in the fulminating hypoxia tests(%)

group (N=8)	before test	breathing rich O_2 1min	3min	5min	exposed to low oxygen(0.8%) 1s	5s	10s	15s	20s	25s	30s
1	98	97	96	96	95	93	89	88	87	87	77
2	97	97	97	98	98	98	96	89	88	85	91
3	97	98	98	98	98	98	96	93	89	93	91
4	98	99	99	99	99	99	98	97	98	97	98
5	98	99	99	99	98	99	98	97	95	97	98
6	98	99	99	99	99	99	99	99	98	98	97
F	2.3	146.3	151.9	153.2	134.1	142.3	2.9	7.5	9.6	10.5	10.7
P		*	*	*	*	*	*	*	*	*	*

Table 7 The changes of the heart rate (beats/min)

group (N=8)	before test	breathing enriched O_2 1min	3min	5min	exposed to low O_2(0.8%) 10s	20s	30s
1	65	70	71	69	72	75	82
2	65	68	67	68	70	72	77
3	69	67	68	69	72	73	77
4	66	66	68	67	70	68	68
5	65	65	67	65	65	66	66
6	68	65	63	65	66	66	65
F	0.46	0.40	0.95	0.48	1.56	1.91	5.98*

oxygen equipment and same kind of pressure vest (table3). Although the quicker onset of pressuring oxygen was advantageous for pretection of hypoxia, it might turn favourable factors into unfavourable ones when it was too fast. The results explain that the lung tissues subject to two involves of pressures in decommpression, that is, two pressure waves, the peak value owing to the gas within breathing equipment expanded in altitude decommpression, and pressure breathing of oxygen system after decompression, come to the thorax in succession within 1 second. Both of them appearing in the twinkling of an eye have two unfavourable influences. First, it stands in the way of expiration; second, it makes the alveoli overextend and then the nervous vagus are overexcited; as a result, some agitations in heart were soon suppressed, and the heart block appeared. Therefore, the onset time of pressure breathing after decommpression should be within the range from 1.3 to 5.0 seconds.

3. The concentration of oxygen supply before the decompression

The concentration of oxygen supply before the decompression must be sufficient to prevent the severe hypoxia after decompression. But the concentration of molecular sieve oxygen generating system(MSOGS) can not be reached pure oxygen because argon is not removed from air. Whether hypoxia protection during rapid decompression may be provided or not is unknown.

For the purpose of our study, an equipment for the experiments had been made which can provide two gas resources: one is gases with rich oxygen(50%, 60%, 70%, 80%, 90%, 100%;) that are simulated attitude 7000m at which the subjects breath them, another is low-oxygen gas(0.8%O_2) that is identical with breathing air at the altitude 16Km when the aircraft cabin pressurization is lost. Eight subjects inspired the oxygen concentration from 50% to 100% for 5 min and then suddenly exposed to simulated 16km for 15 sec by controlling the switch that switched out the rich oxygen and switch on the mixture gas($O_2$0.8%, N_2 99.2%)during the experiment.

The results showed that as long as the concentration of oxygen inspired was above 70% befor exposed, the oxygen partial pressure of expired gas in mask was above 4.0KPa(30mmHg)and no hypoxia symptoms had been found during the test. Our results demonstrated that the initial alveolar oxygen partial pressure has a significant influence upon the final alveolar oxygen partial pressure, the oxyhemoglobin saturation, the heart rate and the hypoxia symptoms when the subjects were exposed to the less oxygen (0.8%) environment(table4~7). When the oxygen concentrations higher than 70% imitated the initial before exposed to hypoxia situation, the subjects had no symptoms of hypoxia, the ear oxyhemoglobin saturation were higher than 90% from the start to the end, the heart rates were less than 70 beats per min. The results seem that as long as the initial oxygen concentration is above 70% before the sudden loss of cabin pressure it can protect aircrew member against severe transient hypoxia following rapidde-compression before pressure oxygen supply. If the concentration is below 60%, the aircrew will suffer from transient hypoxia. Therefore, the requirement of the concentration of aircraft oxygen system in the maximum ceiling at normal cabin altitude should be set at 70% in order to avoid hypoxia under emergency situation.

The preliminary experiment showed 70-80% oxygen concentration may not only prevent from severe transient hypoxia, but also avoid damaging the pilots' lungs due to using pure oxygen in flying.

In summary, the absolute pressure, the time of emergency pressuring oxygen and the oxygen concen tration breathing used in the " vest-anti-G-suit" should be fully considered in the oxygen supply engineering design. They should be adjusted neither too high nor too low, and the human factors should be in basic start point in human engineering interface.

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