Influence of Hypothalamic Hyperphagia on Tolerance of Lung to Explosive Decompression

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A study was made of the effect of hypothalamic hyperphagia on the tolerance of lung to explosive decompression in male Long-Evans rats. The control and hypothalamic hyperphagic rats were explosively decompressed together from 1 atm to an ambient pressure of 30 mm Hg in 0.04s. The hypothalamic-lesioned rats gained from 252 g average weight to 460 g, a 82% gain. The respective figures for the controls were from 248 g to 336 g and 36%. It was also observed that a considerable amount of fat was accumulated between pleura and lungs in experimental animals. The average accumulation of fat between pleura and lungs in experimental rats was 3.23 g, while the value of the control group was only 0.42 g. The difference was statistically significant. Such an increase of fat accumulation in the thoracic cage could decrease the tidal volume. The severity of decompression-induced pulmonary hemorrhages might thus be decreased. On the other hand, it also seems possible that the soft fat cushion between pleura and lungs might damp the bruising of the pulmonary tissue against the resistant thoracic wall to a certain extent, thus resulting in a decreased susceptibility to decompression-induced lung damage. Besides, the mortality in obese rats undergoing explosive decompression was also significantly lower than that of the controls.

It is generally accepted that the most common lesions seen in experimental animals subjected to explosive decompression are pulmonary hemorrhages. Previous work from this laboratory has shown that several experimental conditions—namely, bilateral cervical vagotomy, occlusion of carotid arteries, intravenous administration of epinephrine, and prolonged starvation or semi-starvation—could influence the frequency of occurrence and the severity of decompression-induced pulmonary hemorrhages (2,3,5). The present study is to ascertain whether hypothalamic obesity would affect the tolerance of lung to explosive decompression in rats.

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Materials and Methods

Matched pairs of Long-Evans male rats 18 pairs as nearly alike in initial body weight as possible were used; one of each pair served as a control and the other as an experimental animal. They were housed in individual cages. The experimental animals received lesions in the area of ventromedial nuclei of the hypothalamus (using 2 ma of d.c. current for 20 s) with the aid of a stereo-taxic instrument. All controls received sham operations. During the postoperative period (12 weeks), daily food intake and weekly body weight were measured. After the experimental animal had gained the desired amount of body weight, the obese rat was then explosively decompressed (from 760 to 30 mm Hg in 0.04 s) together with the control rat. Decompression was accomplished by perforating a sheet of X-ray film separating an animal chamber at 1 atm from a large vacuum chamber using the method described previously (2). The rate of decompression was measured from the trace of a Statham model PM±15-350 transducer recorded on the Grass polygraph. All rats were recompressed immediately following the decompression. The lungs were then carefully examined for gross evidence of hemorrhage within a few minutes. The severity of decompression-induced pulmonary hemorrhage was graded according to the following scale: 0 = no hemorrhage; + = slight hemorrhage (a few petechial hemorrhages); ++ = moderate hemorrhage (hemorrhagic area was less than 25% of the lungs); +++ = severe hemorrhage (hemorrhagic area was 25-50% of the lungs) and ++++ = very severe hemorrhage (hemorrhagic area was greater than 50% of the lungs).

At the end of each acute experiment, the carcass was weighed and dried in an oven at 105°C in a pre-weighed beaker. After constant weight was obtained, petroleum ether was added in order to extract the body fat from the carcass; 5-7 changes of petroleum ether were needed before the supernatant appeared free from fat. The beaker containing the carcass was then dried in the same oven to a constant weight again. From the difference of the two constant weights the weight of body fat was obtained. Body fat expressed as percentage of total body weight (fat %) was calculated from the fat weight and the total body weight at sacrifice.

Results

The results are shown in Table 1. The experimental rats weighed an average of 252 g at the start, hypothalamic hyperphagia increased this to 460 g; the average weight gain was 82%. The control animals, otherwise, gained from 248 g average weight to 336 g, only a 36% gain. The average percentage of body fat content of the experimental rats was also significantly higher than that of the control group. Fig. 1 shows the obesity of a hypothalamic hyperphagic rat. It was also observed that a considerable amount of fat was accumulated between pleura and lungs in experimental rats. The average of such fat accumulation of the obese and control rats was 3.23 g and 0.42 g respectively. As mentioned above, the severity of decompression-

Table 1 The Occurrence of Pulmonary Hemorrhage in Control and Hypothalamic Hyperphagic Rats Following a Single Explosive Decompression from 1 atm to an Ambient Pressure of 30 mm Hg in 0.04 s.

Control									Experimental							
	Body weight				Fat in thoracic cage	Pulmonary hemorrhage				Body weight				Fat in thoracic cage	Pulmonary hemorrhage		
Rat No.	Initial wt. g	Final wt. g	Diff. %	Body fat %	g	Lt. lung	Rt. lung	Issue	Rat No.	Initial wt. g	Final wt. g	Diff. %	Body fat %	g	Lt. lung	Rt. lung	Issue
1	246	366	49	12.7	0.41	+	+++		2	252	482	91	51.4	2.72	+	+	
3	254	351	38	16.0	0.34	++	++		4	267	511	91	50.6	3.24	++	++	
5	240	319	33	14.5	0.46	++	++		6	244	409	68	44.1	3.81	++++	+++	D
7	224	318	42	15.0	0.45	++++	++++	D	8	226	367	62	40.7	2.58	+	0	
9	252	341	35	12.8	0.35	++	++++		10	259	509	97	46.5	3.69	+	+++	
11	241	330	37	11.3	0.43	++++	++++	D	12	248	451	82	48.0	2.91	++	++	
13	246	335	36	10.3	0.44	++++	+++	D	14	254	497	96	47.7	2.42	++	+	
15	241	329	37	12.4	0.40	++++	++++	D	16	243	468	93	43.2	2.78	+++	++	
17	229	313	37	9.9	0.42	++++	+++	D	18	231	360	56	48.0	3.13	+	+	
19	250	332	33	9.6	0.39	++++	++++	D	20	253	444	76	45.2	3.76	+++	++++	D
21	255	345	35	11.6	0.56	++++	++++	D	22	262	404	54	49.2	3.72	++	+++	D
23	251	310	24	8.7	0.34	++++	+++	D	24	259	464	79	45.4	3.51	++	++	
25	247	360	46	15.3	0.46	++++	++++	D	26	250	465	86	49.2	3.54	++++	++	D
27	266	354	33	11.0	0.41	++++	+++	D	28	269	493	83	50.3	3.95	+++	++	
29	251	320	28	14.3	0.43	+++	+++		30	254	466	84	48.4	3.81	+++	+	
31	249	322	29	13.6	0.43	++++	+++	D	32	256	494	93	51.2	3.61	+++	+++	D
33	258	342	33	10.8	0.37	+++	++		34	258	513	99	46.3	3.77	+++	+++	D
35	261	360	38	12.8	0.49	++++	++++	D	36	254	489	93	41.0	4.05	++	++	
Av.	248	336	36[a]	12.4[b]	0.42[c]	+++[d]	+++[e]			252	460	82[a]	47.0[b]	3.23[c]	++[d]	++[e]	
Number of rats died								12[f]									6[f]

0 = No hemorrhage
\+ = Slight hemorrhage
++ = Moderate hemorrhage
+++ = Severe hemorrhage
++++ = Very severe hemorrhage
D = Died following explosive decompression

p(a:a) < 0.001
p(b:b) < 0.01
p(c:c) < 0.01
p(d:d) < 0.01
p(e:e) < 0.005
p(f:f) < 0.05

Fig. 1. Hypothalamic lesions and obesity in the rat. Left: Obese rat (No.4), obesity due to bilateral destruction of the ventromedial nuclei of the hypothalamus. Initial body weight: 267 g. Final body weight (12 weeks after hypothalamic lesions) : 511 g. Body fat: 50.6%. Right: Control rat (No.3). Initial by weight: 254 g. Final body weight (12 weeks after sham operation): 351 g. Body fat: 16.0%.

induced hemorrhages was evaluated by the number of + signs; the severer the former, the more the latter. The average number of + signs induced from explosive decompression in control rats was +++, while the average for the experimental rats was only ++. Besides, 67% and 33% of the control and experimental animals respectively, died following explosive decompression. The differences were statistically significant.

Discussion

Because of the delicate nature of the pulmonary tissue, the lungs are potentially the most vulnerable part of the body during an explosive decompression. There are two possible mechanism which may play a role in producing decompression-induced pulmonary he-

morrhage. In the one case, sudden rapid expansion of the lungs, with stretching of the alveolar walls, might result in the actural tearing of these structures. Such a mechanism was postulated by Greeley and Drury (4), who suggested the possibility of preventing lung damage by appropriate taping or binding of the thorax of the rat. In the present study, a considerable amount of fat was found to be accumulated between pleura and lungs in experimental rats and the lungs of such obese rats were more tolerant to explosive decompression. When the fat accumulation in the thoracic cage is increased, the volume of thoracic cavity is relatively decreased. As a result, the tidal volume and alveolar ventilation may thus be decreased. Auchincloss et al. (1), working on obese man, also found that obese subjects revealed the presence of severe alveolar hypoventilation which was characterized by a marked reduction in tidal volume. A decrease in the volume of gas in the lugs of the experimental rat may, therefore, decrease the severity of decompression-induced pulmonary hemorrhage. The present findings seem to lend support to the mechanism as postulated by Greeley and Drury (4). On the other hand, Whitehorn et al. (6) found that binding of the thorax and abdomen of dogs enhanced the incidence and severity of the pulmonary hemorrhage following explosive decompression. They maintained that the pulmonary hemorrhage seen after explosive decompression was due primarily not to overexpansion of the alveoli, with tearing of their walls, but rather to the sudden application of pressure to these alveolar walls, driving them against the more rigid thoracic wall with a resultant bruising action. If such a mechanism is true, the decompression-induced pulmonary hemorrhage will also be less severe when there is fat accumulation between pleura and lungs. In other words, it seems possible that the soft fat cushion between pleura and lungs of the experimental rats might damp the bruising of the pulmonary tissue against the resistant thoracic wall to a certain extent, resulting in a decreased susceptibility to lung damage following explosive decompression.

There is a relationship between the severity of pulmonary hemorrhage and the mortality in rats undergoing explosive decompression. The severer the former, the greater the latter. Since the fat accumulation between pleura and lungs could decrease the susceptibility to decompression-induced lung damage, it would be reasonable to expect that the fat accumulation would decrease the mortality in obese rats following explosive decompression.

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