FULL PAPER *Clinical Pathology*

Radiographic Evaluation of Obesity-Caused Oppression of the Thoracic Cavity in Beagles

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ABSTRACT. Thoracic radiographs of fifteen beagles with mild-to-moderate obesity revealed that oppression of the thoracic cavity increased with increasing degree of obesity. Oppression of the thoracic cavity was evaluated based on the length, depth, width and area of the thoracic cavity. To obtain thoracic radiographs at the terminal inspiration and expiration phases, thoracic fluororadiographs were recorded with a digital video camera. Bodyweight and the depth of the back fat layer at the seventh lumbar vertebra (DB, measured by ultrasonography) were used as indicators of the degree of obesity. The length of the thoracic cavity tended to become shorter and the depth and width of the thoracic cavity tended to increase as bodyweight increased and as DB increased. On the other hand, the area of the thoracic cavity was not clearly related to bodyweight or DB. These results suggest that oppression of the thoracic cavity due to the cranial shift of the diaphragm is compensated for by increases in the depth and width of the thoracic cavity in beagles with mil d-to-moderate obesity.

KEY WORDS: beagle, diaphragm shift, obesity, radiographic evaluation, thoracic cavity oppression.

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Obesity in dogs is associated with complicating diseases such as cardiac and pulmonary insufficiency and spondylopathy [4, 6, 7, 14, 15, 21, 23]. Recently, several practical methods for evaluation of obesity in dogs have been reported, such as body condition score (BCS) [4, 10, 11], subcutaneous fat thickness [1, 17, 22] and blood leptin concentration [8, 9, 19].

Obesity in pet dogs is caused by over-feeding by their owners and by low levels of exercise. To prevent the problems associated with obesity, the amount of food must be limited. However, it is often difficult for the owner to realize that over-feeding is bad for their pet. This is partly because giving food to a pet is an important factor in the human-animal bond as a source of pleasure for the owner. The lifestyle of the owner may also be a cause of over-feeding because overweight owners are more likely to have overweight pets [3, 16]. Moreover, obesity-related dysfunctions gradually progress with increasing body fat, and are usually not noticed until they are at an advanced stage.

For these reasons, veterinarians need persuasive evidence to make pet owners realize that excessive feeding advances the onset of the complicating diseases in the subclinical stage. Although several studies have warned about the risk of obesity, there is little evidence linking increasing fat deposition with the onset of complicating diseases. We have noticed that extremely obese small-sized dogs have enlarged cardiac silhouettes and reduction of the lung area due to a considerable cranial shift of the diaphragm as viewed in thoracic radiographs. The cranial shift suggests oppression of the thoracic cavity, restriction of respiratory movement with increased endoceliac fat and the continuous acceleration of hypoxemia.

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In this study, we tested and confirmed our hypothesis that oppression of the thoracic cavity increases with increasing degree of obesity in the subclinical stage, by measuring the cranial shift of the diaphragm and reduction of the expansion area of the thoracic cavity in beagles using thoracic radiographs.

MATERIALS AND METHODS

Fifteen six-year-old, clinically normal male beagles with various degrees of obesity were used. BCS of these dogs on the nine-point scoring system [11] ranged from BCS 5 to BCS 8. Withers height was 38.0 ± 1.4 cm and body length was 42.4 ± 2.1 cm, the physique of the dogs did not so much differ. Measurements were made for a total of three times in early April, mid May and late June. Bodyweight and the depth of the back fat layer at the seventh lumbar vertebra (DB) were measured as indicators of the degree of obesity (Table 1). Simultaneously, chest girth and abdomen girth were measured. DB was measured by ultrasonography [17]. Images of the thoracic cavity at the terminal inspiration and expiration phases were obtained as follows. Dogs were anesthetized with medetomidine hydrochloride (10 μg/kg, IV) and examined at right lateral (RL) and dorsoventral (DV) positions under constant breathing conditions with a fluoroscope (TU-130XF, Hitachi, Tokyo). The anesthetic depth [13] was plane 1 of stage III. Because the thoracic size was larger than the frame size of the fluoroscope, the cranial, middle and caudal parts of the thorax were examined separately for both the RL and DV positions (Fig. 1). Fluoroscope images were continuously recorded with a digital video camera for a period of three respiratory cycles. For each cycle, the frames corresponding to the largest sizes for the three sections of the thoracic cavity were combined

Table 1. Body measurements of 15 beagles at three measurement times

Body measurements	Early April	Mid May	Late June
Bodyweight			
Mean ± SD (kg)	13.5 ± 3.4	14.7 ± 3.4	14.9 ± 3.3
Range (kg)	9.0–19.4	10.0–20.2	11.0–20.6
Back fat layer depth			
Mean ± SD (mm)	3.5 ± 2.6	4.1 ± 2.9	6.2 ± 3.7
Range (mm)	0.4–9.7	0.6–11.1	0.9–15.2
Chest girth			
Mean ± SD (cm)	53.1 ± 5.8	53.7 ± 5.5	54.1 ± 5.4
Range (cm)	45.1–63.3	46.4–63.4	46.7–63.1
Abdomen girth			
Mean ± SD (cm)	47.6 ± 8.5	48.4 ± 8.7	48.6 ± 9.0
Range (cm)	36.0–60.7	36.4–64.3	36.7–65.9

on a computer to obtain a view of the whole thoracic cavity in the terminal inspiration phase. Thus, three images of the expanded thoracic cavity were obtained, one for each of the three respiratory cycles. This procedure was repeated for the terminal expiration phase, using the frames corresponding to the smallest size.

To evaluate the oppression to the thoracic cavity, the length, depth, width and area of the thoracic cavity were measured for each of the three images for each phase and then averaged. Measurements of the thoracic cavity were made as follows. The most cranial point of the thoracic cavity in the radiograph was defined as the posterior border of the spinous process of the seventh cervical vertebra, and the most caudal point was defined as the most posterior point of the diaphragm. In the RL view (Fig. 1A, B), the thoracic cavity was defined as the part enclosed by the lines of the thoracic vertebrae and sterna and diaphragm, and in the DV view (Fig. 1C), it was defined as the part enclosed by the lines of the costal edge and the diaphragm. The length of the thoracic cavity was measured in the RL view along two lines (LA and LP) starting from the cranial point of the thoracic cavity (Fig. 1A). LA was to the most anterior border of the diaphragm and LP was to the most posterior border of the diaphragm. The depth of the thoracic cavity was measured in the RL view along three lines (D3, D6 and D9), starting from the anterior ventral point of the third, sixth and ninth thoracic vertebrae, and vertically extending to the sterna (Fig. 1B). The width of the thoracic cavity was measured in the DV view along three lines (W3, W6 and W9), starting from the anterior border of the base of the spinous process of the third, sixth and ninth thoracic vertebrae, and horizontally extending to costal edge (Fig. 1C). The area of the thoracic cavity was measured in both the RL and DV view. The length, width and depth of the thoracic cavity were calculated by dividing the number of pixels along the lines by the number of pixels per unit length. Similarly, the area of the thoracic cavity was calculated by dividing the number of pixels in the enclosed area by the number of pixels per unit area.

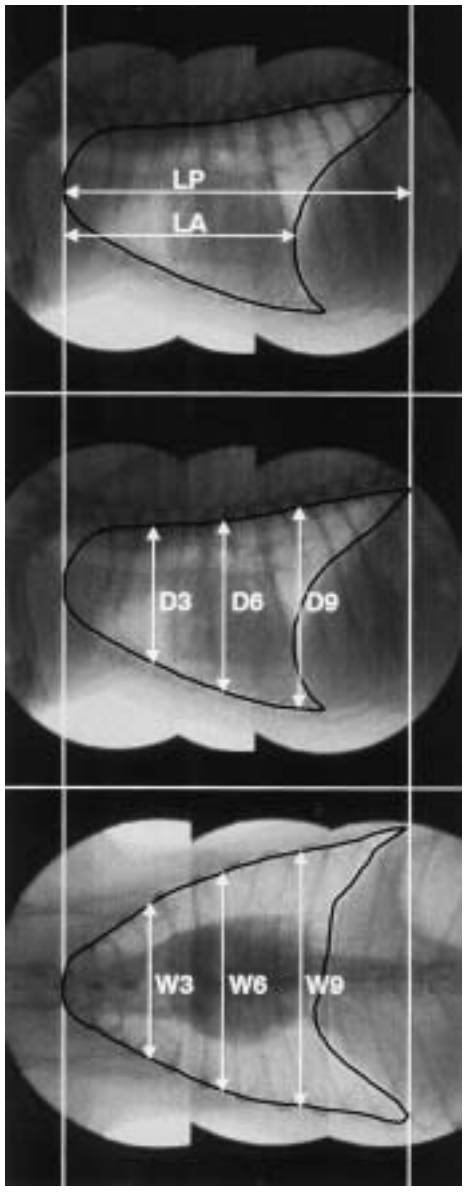

Fig. 1. Representative thoracic fluororadiographs of a beagle in right lateral views (A, B) and in a dorsoventral view (C). A and B are the same radiograph. Each radiograph was obtained by combining three adjacent radiographs made of the cranial, middle and caudal part of the thorax. The most cranial and the most caudal points of the thoracic cavity are indicated by white lines. The thoracic cavity is defined as the part enclosed with the black line, and its area including the heart is taken as the area of the thoracic cavity. The double-headed arrows indicate the measurement points used to determine the length (A), depth (B) and width (C) of the thoracic cavity. The length was measured from the cranial point of the thoracic cavity to the most anterior border of the diaphragm (LA) and to the most posterior border of the diaphragm (LP). The depth was measured at the anterior borders of the body of the third (D3), sixth (D6) and ninth thoracic vertebrae (D9). The width was measured at the anterior border of the spinous process of the third (W3), sixth (W6) and ninth thoracic vertebrae (W9).

RESULTS

BCS in each dog did not changed among three measurement times. DB correlated positively with bodyweight (r=0.67, P<0.001) (Fig. 2). The chest girth and abdomen girth correlated positively with bodyweight (r=0.95 and 0.90, respectively, P<0.001) and DB (r=0.60 and 0.58, respectively, P<0.001).

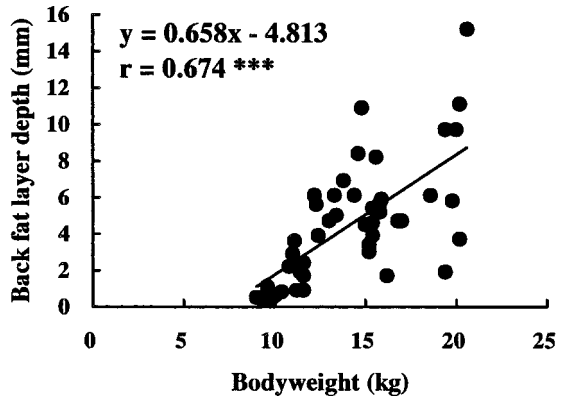

Fig. 2. Relationships between bodyweight and the depth of the back fat layer. ***: P<0.001.

The data obtained for all 15 dogs at each of the three measurement times were pooled for each breathing phase (n = 45).

The relationships between the length of the thoracic cavity and bodyweight and DB are plotted in Fig. 3. The two measures of the length of the thoracic cavity (LA and LP, Fig. 3A, B) showed significant negative correlations with bodyweight (r = –0.44 to –0.57, P<0.01) in both the inspiration and expiration phases. DB showed slight negative correlations with the lengths at LP (Fig. 3D) for the inspiration and expiration phases (r = –0.28 and –0.33, respectively, P<0.10), while it showed significant negative correlations with the lengths at LA (Fig. 3C) (r = –0.52 and –0.52, respectively, P<0.001). The length of the thoracic cavity showed significant negative correlations with chest girth (r = –0.50 to –0.57, P<0.001) in both the inspiration and expiration phases, and also showed significant negative correla-

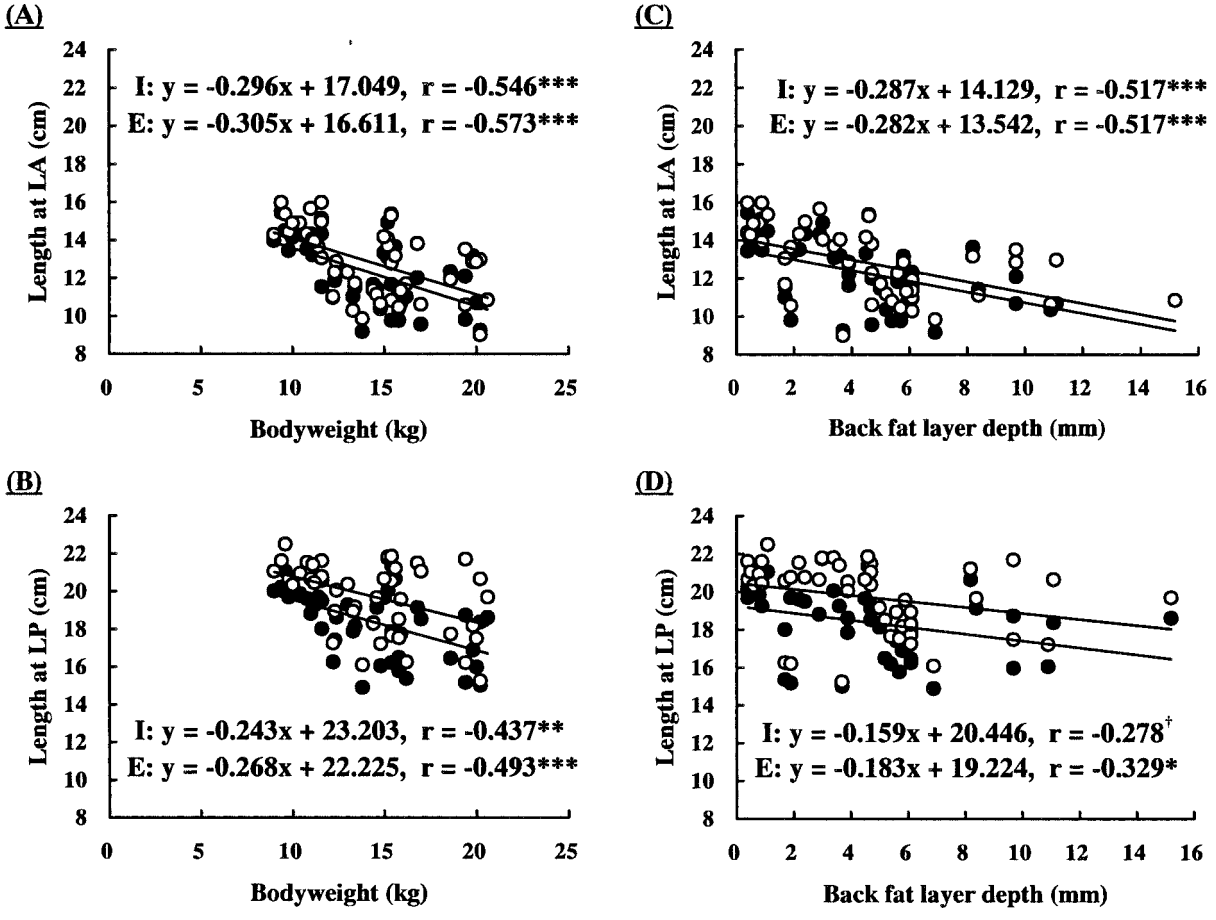

Fig. 3. Relationships between the length of the thoracic cavity and bodyweight (A: at LA, B: at LP) and the depth of back fat layer (C: at LA, D: at LP). Open circles indicate the inspiration phase (I) and closed circles indicate the expiration phase (E). †: P<0.10, *: P<0.05, **: P<0.01, ***: P<0.001.

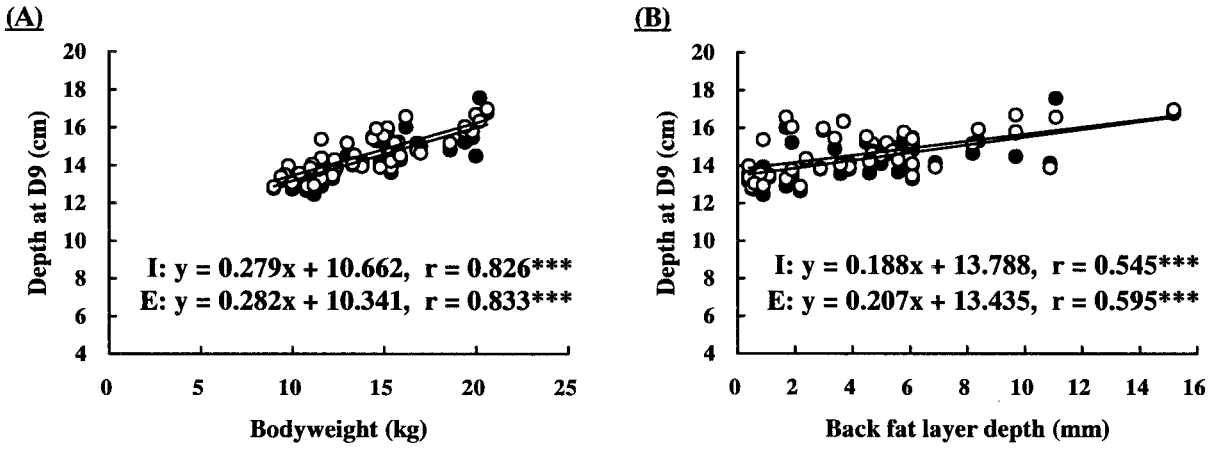

Fig. 4. Relationships between the depth of the thoracic cavity at D9 and bodyweight (A) and the depth of back fat layer (B). See Fig. 3 for an explanation of panels and symbols. ***: P<0.001.

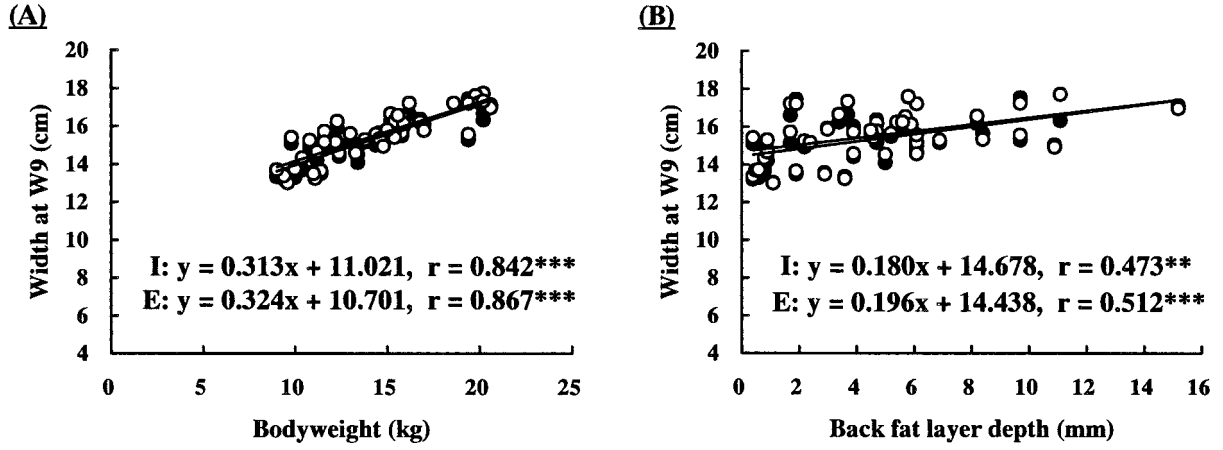

Fig. 5. Relationships between the width of the thoracic cavity at W9 and bodyweight (A) and the depth of back fat layer (B). See Fig. 3 for an explanation of panels and symbols. **: P<0.01, ***: P<0.001.

tions with chest girth and abdomen girth (r = –0.61 to –0.66, P<0.001) in both the inspiration and expiration phases.

The relationships between the depth of the thoracic cavity and bodyweight and DB are plotted in Fig. 4. The depth of the thoracic cavity at D9 (Fig. 4A) for the inspiration and expiration phases showed significant positive correlations with bodyweight (r=0.83 and 0.83, respectively, P<0.001). Similar trends were found at D3 and D6 (r=0.69 to 0.86, P<0.001). The depth of the thoracic cavity at D9 (Fig. 4B) for the inspiration and expiration phases showed significant positive correlations with DB (r=0.55 and 0.60, respectively, P<0.001). Similar trends were found at D3 and D6 (r=0.54 to 0.62, P<0.001).

The relationships between the width of the thoracic cavity and bodyweight and DB are plotted in Fig. 5. The width of the thoracic cavity at W9 (Fig. 5A) for the inspiration and expiration phases showed significant positive correlations with bodyweight (r=0.84 and 0.87, respectively, P<0.001). Similar trends were found at W3 and W6 (r=0.42 to 0.79, P<0.01). The width of the thoracic cavity at W9 (Fig. 5B) for the inspiration and expiration phases showed significant positive correlations with DB (r=0.47 and 0.51, respectively, P<0.01). Similar trends were found at W3 and W6 except for the width at W3 in the expiration phase (r=0.32 to 0.50, P<0.05).

There were no significant correlations between the area of the thoracic cavity and bodyweight and DB either in the RL or the DV view in either the inspiration or expiration phases. However, the cardiac silhouette showed significant positive correlations with bodyweight and DB (r=0.60 to 0.79, P<0.001) in both the RL and DV view, and the ratio of the cardiac silhouette to the area of the thoracic cavity showed significant positive correlations with bodyweight and DB (r=0.54 to 0.84, P<0.001) in both the RL and DV

view in both the inspiration and expiration phase. In addition, the area of the thoracic cavity excluding the cardiac silhouette in the DV view showed significant negative correlations with bodyweight and DB except for with DB in the expiration phase ($r = -0.30$ to -0.42, $P<0.05$).

Thus, the length of the thoracic cavity tended to become shorter and the depth and width of the thoracic cavity tended to increase as bodyweight increased and as DB increased. On the other hand, the area of the thoracic cavity was not clearly related to bodyweight or DB. However, oppression of the lung by enlarged heart tended to become large as bodyweight increased and as DB increased.

DISCUSSION

Obesity increases the risk of circulatory disorders because of the increased perfusion requirements of an expanded fat mass. Hypercardia and cardiac insufficiency are caused by increases in the cardiac output and heart rate (1) to deal with the expanded blood perfusion area due to excessive body fat deposition and (2) to cope with the continuously high tissue oxygen requirement owing to the increased bodyweight [6, 23]. In addition, panting often appears with obesity.

In humans, many studies have shown that excess weight can lead to pulmonary dysfunction. A gain in bodyweight has been shown to decrease pulmonary functions such as forced vital capacity and forced expiratory volume in one second [2]. The accumulation of fat with obesity in and around the ribs, the diaphragm and the abdomen decrease the chest wall compliance, and the functional residual capacity falls below the closing volume [5,12,18], and hypoxemia occurs even in the sitting position [20]. In the supine position, hypoxemia becomes obvious due to oppression of the diaphragm by abdominal organs.

On the other hand, in dogs, several studies have investigated the relationship between obesity and pulmonary dysfunction. Obesity is one of the causes of dyspnea [21] and weight reduction with chronic bronchial or pulmonary disease can be of great benefit [7]. Moreover, pulmonary volume restriction, owing to intrathoracic fat deposits and the cranial displacement of the diaphragm by abdominal fat, can cause a decrease in lung capacity [23]. Despite the importance this problem, few studies have investigated it in detail. One reason for this is that methods for measuring pulmonary function require the subject to control his breathing and this can't be done with dogs.

As shown in this study, this problem could be solved by selecting the appropriate frames from a videotape of the thoracic cavity. The two-dimensional data that we obtained confirmed that the cranial shift of the diaphragm increases with increasing degree of obesity. Although endoceliac fat was not measured directly in this experiment, it is thought that the endoceliac fat increases with increasing degree of obesity, as the abdomen girth showed negative correlations with bodyweight and DB. And, it is guessed that the cranial shift of the diaphragm occurs due to the accumulation of the endoceliac fat, as the length of the thoracic cavity showed negative correlations with abdomen girth. In addition, the lung is more oppressed by enlarged heart with oppression of the thoracic cavity by abdominal fat, as the cardiac silhouette became larger with increasing degree of obesity. However, the area of the thoracic cavity was not clearly related to the degree of obesity because the depth and width of the thoracic cavity tended to increase with increasing degree of obesity. These results suggest that oppression of the thoracic cavity due to the cranial shift of the diaphragm is compensated for by increases in the depth and width of the thoracic cavity in beagles with mild-to-moderate obesity.

In conclusion, this thoracic radiographic method is useful for indirect evaluation of oppression of the thoracic cavity and provides clear evidence of the suppressive effects of obesity on the thoracic cavity in the subclinical stage.

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REFERENCES

1. Anderson, D. B. and Corbin, J. E. 1982. Estimating body fat in mature beagle bitches. *Lab. Anim. Sci.* **32**: 367–370.
2. Chen, Y., Horne, S. L. and Dosman, J. A. 1993. Body weight and weight gain related to pulmonary function decline in adults: a six year follow up study. *Thorax* **48**: 375–380.
3. Crane, S. W. 1991. Occurrence and management of obesity in companion animals. *J. Small Anim. Pract.* **32**: 275–282.
4. Edney, A. T. B. and Smith, P. M. 1986. Study of obesity in dogs visiting veterinary practices in United Kingdom. *Vet. Rec.* **118**: 391–396.
5. Emirgil, C. and Sobol, B. J. 1973. The effects of weight reduction on pulmonary function and the sensitivity of the respiratory center in obesity. *Am. Rev. Respir. Dis.* **108**: 831–842.
6. Ettinger, S. J. 2000. Dietary modifications in cardiac disease. pp. 262–269. *In*: Textbook of Veterinary Internal Medicine, 5th ed. (Ettinger, S. J. and Feldman, E. C. eds), W. B. Saunders, Philadelphia.
7. Hawkins, E. C. 2000. Pulmonary parenchymal disease. pp. 1061–1091. *In*: Textbook of Veterinary Internal Medicine, 5th ed. (Ettinger, S. J. and Feldman, E. C. eds), W. B. Saunders, Philadelphia.
8. Ishioka, K., Soliman, M. M., Sagawa, M., Nakadomo, F., Shibata, H., Honjoh, T., Hashimoto, A., Kitamura, H., Kimura, K. and Saito, M. 2002. Experimental and clinical studies on plasma leptin in obese dogs. *J. Vet. Med. Sci.* **64**: 349–353.
9. Kitagawa, H., Mizoguchi, H., Kitoh, K., Kuwahara, Y., Ohba, Y., Shimizu, Y., Ohtsuka, Y. and Sasaki, Y. 2000. Plasma leptin concentrations in obese dogs. *J. Jpn. Vet. Med. Assoc.* **53**: 311–314 (in Japanese with English summary).
10. Kronfeld, D. S., Doneghue, S. and Glickman, L. T. 1991. Body condition and energy intakes of dogs in a referral teaching hospital. *J. Nutr.* **121**: S157–S158.
11. Laflamme, D. 1997. Development and validation of a body condition score system for dogs. *Canine Pract.* **22**: 10–15.
12. Luce, J. M. 1980. Respiratory complications of obesity. *Chest* **78**: 626–631.
13. Lumb, W. V. and Jones, E. W. 1984. General anesthesia. pp. 199–211. *In*: Veterinary Anesthesia, 2nd ed. (Lumb, W. V. and Jones, E. W. eds), Lea & Febiger, Philadelphia.

14. Lund, E. M., Armstrong, P. J., Kirk, C. A., Kolar, L. M. and Klausner, J. S. 1999. Health status and population characteristics of dogs and cats examined at private veterinary practices in the United States. *J. Am. Vet. Med. Assoc.* **214**: 1336–1341.
15. MacEwen, E. G. 1992. Obesity. pp. 313–318. *In*: Current Veterinary Therapy, 11th ed. (Kirk, R. W. and Bonagura, W. B. eds.), W. B. Saunders, Philadelphia.
16. Manson, E. 1970. Obesity in pet dogs. *Vet. Rec.* **86**: 612–616.
17. Morooka, T., Niiyama, M., Uchida, E., Uemura, M., Miyoshi, K. and Saito, M. 2001. Measurement of the back fat layer in beagles for estimation of obesity using two-dimensional ultrasonography. *J. Small Anim. Pract.* **42**: 56–59.
18. Ray, C. S., Sue, D. Y., Bray, G., Hansen, J. E. and Wasserman, K. 1983. Effect of obesity on respiratory function. *Am. Rev. Respir. Dis.* **128**: 501–506.
19. Sagawa, M., Yoneda, S., Nakadomo, F., Honjoh, T., Ishioka, K. and Saito, M. 2002. Enzyme-linked immunosorbent assay of plasma leptin in dogs: a highly positive correlation to body fat content. *Am. J. Vet. Res.* **63**: 7–10.
20. Taguchi, O. 1995. Respiratory function disturbance in obesity. *Jpn. J. Clin. Med. (Suppl.)* **1995**: 343–348 (in Japanese).
21. Turnwald, G. H. 2000. Dyspnea and tachypnea. pp. 166–169. *In*: Textbook of Veterinary Internal Medicine, 5th ed. (Ettinger, S. J. and E. C. Feldman, E. C. eds), W. B. Saunders, Philadelphia.
22. Wilkinson, M. J. A. and McEwan, N. A. 1991. Use of ultrasound in the measurement of subcutaneous fat and prediction of total body fat in dogs. *J. Nutr.* **121**: S47–S50.
23. Wolfsheimer, K. J. 2000. Obesity. pp. 70–72. *In*: Textbook of Veterinary Internal Medicine, 5th ed. (Ettinger, S. J. and Feldman, E. C. eds), W. B. Saunders, Philadelphia.