Thorax (1970), **25**, 129.

Post-mortem size and structure of the human carotid body

Its relation to pulmonary disease and cardiac hypertrophy

DONALD HEATH, CHRISTOPHER EDWARDS, and PETER HARRIS

Department of Pathology, University of Liverpool, and the Institute of Cardiology, University of London

The weights of the carotid bodies have been measured in an unselected group of 40 successive cases coming for routine necropsy. Although the mean weight of the carotid bodies was higher in those subjects with emphysema or Pickwickian syndrome, this difference did not reach a level of statistical significance. Nevertheless, in a group of two patients with cor pulmonale and one with the Pickwickian syndrome the mean weight of the carotid bodies was significantly higher than that of the rest. Such observations would be consistent with enlargement of the glomic tissue in response to chronic hypoxia. In addition to these observations there was a significant correlation between the size of the carotid bodies and the weights of the left and right ventricles separately, while there was a substantially higher correlation between the weights of the carotid bodies and the combined weights of the two ventricles. Should such a correlation be determined by a causative link between these two measurements, a new metabolic role of glomic tissue will have to be considered. The histology of the carotid bodies was studied and differential cell counts were carried out in 21 cases. Enlargement of the carotid bodies was not accompanied by any change in the differential cell count, except that in one case with 'cor pulmonale' and large carotid bodies areas of hyperplasia showed a predominance of the dark variety of type 1 (chief) cell.

Knowledge of the pathology of the carotid body is virtually confined to the neoplasm of glomic tissue, the chemodectoma. Our understanding of the part played by the carotid body in generalized systemic disease is meagre. Its only known physiological function is to respond to a low partial pressure of oxygen in the arterial blood. Recently, Arias-Stella (1969) has reported an increase in the size of the carotid body in people dwelling at high altitude, which would suggest that it responds by enlargement to a prolonged physiological stimulus. The present investigation was undertaken to see whether the carotid body was enlarged in patients dying with respiratory disease. While the results neither confirm nor deny this hypothesis they suggest that there is an unexpected relation between cardiac hypertrophy and the size of the carotid bodies. The present study also comprises observations on the histology of the human carotid body.

METHODS

CASES STUDIED Observations were made on 40 successive cases coming to necropsy in the Department of Pathology at the University of Liverpool (Table I). Their ages ranged from 14 to 95 years, with a mean age of 69·4 years (SD 16·1). Twenty-two were male and 18 were female. There were nine cases of emphysema, of whom two had been diagnosed as 'cor pulmonale' during life, and one case of the Pickwickian syndrome.

Weighing of carotid bodies In every case the bifurcations of both common carotid arteries were resected. Usually the specimen consisted of 4 cm. of common carotid artery, the bifurcation of this vessel and 3 cm. of both the internal carotid and external carotid arteries. The carotid body was dissected out on the fresh specimen immediately after it had been obtained at necropsy. Using pointed scissors and fine forceps, surrounding connective tissues were dissected away. The fibrous adventitial

TABLE I

CARDIAC AND CAROTID BODY WEIGHTS IN 40 SUBJECTS
(The cases are listed in order of ascending combined weight of carotid bodies)

Case No.	Age	Sex	Diagnosis	Cardiac Weights (g.)		Carotid Body Weights (mg.)		
				L.V.	R.V.	R.	L.	C.
1	80	M	Cholangitis	152	61	1·9	2·8	4·7
2	86	F	Panacinar emphysema	130	60	3·2	2·8	6·0
3	77	F	Pulmonary oedema	135	60	3·3	3·0	6·3
4	66	M	Systemic hypertension	235	110	4·8	2·0	6·8
5	77	M	Lobar pneumonia	165	60	5·4	3·4	8·8
6	72	M	Systemic hypertension	220	50	3·9	4·9	8·8
7	63	F	Carcinoma colon	158	55	6·5	2·7	9·2
8	72	M	Aortic aneurysm	197	83	5·8	3·8	9·6
9	35	F	Renal cortical necrosis	170	85	3·4	6·4	9·8
10	87	F	Panacinar emphysema	142	40	4·1	5·7	9·8
11	60	F	Coronary disease	139	54	3·7	7·0	10·7
12	63	F	Multiple injuries	115	35	5·2	6·3	11·5
13	21	F	Goodpasture's syndrome	180	82	7·9	3·7	11·6
14	80	M	Coronary disease	188	60	5·1	6·6	11·7
15	87	M	Systemic hypertension	300	50	9·1	3·2	12·3
16	14	F	Phaeochromocytoma	160	42	5·9	8·3	14·2
17	68	M	Bronchial carcinoma	140	70	6·9	7·3	14·2
18	70	M	Coronary thrombosis	270	115	6·9	7·3	14·2
19	67	M	Cerebral haemorrhage	138	44	6·7	8·8	15·5
20	71	M	Systemic hypertension	215	47	10·0	5·6	15·6
21	84	F	Aortic stenosis	175	60	9·3	6·8	16·1
22	77	M	Coronary thrombosis	250	90	8·8	7·3	16·1
23	68	F	Polycystic disease of kidneys	175	50	8·9	8·0	16·9
24	69	M	Systemic hypertension	190	50	9·0	8·5	17·5
25	65	F	Amputation for gangrene	190	55	9·8	9·3	19·1
26	75	M	Panacinar emphysema	125	54	12·0	11·9	23·9
27	67	M	Myocardial infarction	290	79	18·9	7·5	26·4
28	85	F	Emphysema	145	55	18·2	11·8	30·0
29	78	M	Panacinar emphysema	210	70	20·3	12·2	32·5
30	65	F	Coronary thrombosis	140	65	20·5	13·0	33·5
31	84	F	Systemic hypertension	310	80	18·7	16·5	35·2
32	78	F	Aortic stenosis	285	95	15·4	23·8	39·2
33	95	F	Aortic stenosis	300	65	20·6	20·1	40·7
34	62	M	Panacinar emphysema	245	80	21·9	22·0	43·9
35	60	M	Pickwickian syndrome	240	90	15·8	29·7	45·5
36	80	F	Cerebral haemorrhage	279	64	26·0	24·1	50·1
37	58	M	Systemic hypertension	235	65	27·9	27·9	55·8
38	72	M	Cor pulmonale. P.A. emphysema	226	198	45·3	15·6	60·9
39	69	M	Cor pulmonale. P.A. emphysema	200	133	41·6	27·2	68·8
40	70	M	Chronic bronchitis and emphysema	290	80	37·4	47·4	84·8

L.V.= Left ventricle; R.V.= right ventricle; R= right carotid body; L.= left carotid body; C.= combined weight of carotid bodies.

sheath was then gently teased away to reveal the carotid body at or near the bifurcation on its posterior aspect. Dissection was easy and occupied only 5 to 10 minutes. The specimen was photographed (Figs 1 to 4) and the pedunculated carotid body was nipped off with scissors. Minute amounts of adipose and fibrous tissue were then dissected off the carotid body, which was lightly blotted, to remove any excess tissue fluid, and weighed. After being weighed the carotid body was immersed in 4% formaldehyde for fixation and subsequent histological study. The histological investigation confirmed that the tissue removed consisted of glomic tissue without extraneous tissues.

Weighing of cardiac ventricles The heart was dissected according to the method of Fulton, Hutchinson. and Morgan Jones (1952) to obtain the weights of the right and left ventricles. Using this technique pericardial fat was trimmed from the cardiac muscle ; the valves and atria were also removed. The free wall of the right ventricle was dissected away from the interventricular septum and weighed separately. The interventricular septum and left ventricle were not dissected further and were weighed together. Fulton *et al.* (1952) state that the normal right ventricular weight does not exceed 65 g. and that the normal weight of the 'left ventricle' as defined above does not exceed 190 g.

Histological examination Histological examination was carried out in 21 of the 40 cases. These included the case of Pickwickian syndrome and six cases of emphysema, of which two had been diagnosed 'cor pulmonale'. After being weighed, the carotid bodies were fixed in 10% buffered formalin for 24 hours. Each carotid body was then embedded individually in paraffin wax and serial sections, 5 μ thick, were cut at 200 μ intervals throughout each block. Each section was stained with haematoxylin and eosin to demonstrate the general morphology.

An eyepiece micrometer was calibrated using a stage micrometer marked in divisions of 0·1 and 0·01 mm. The length and breadth of the section shown by micrometry on serial sections to be the largest from each block was measured and recorded, and

the depth of each specimen was calculated from the number of sections required to cut the specimen out of the block. The parenchymal cells of chemoreceptor tissue were arranged in *clusters* and aggregates of these clusters were arranged in *lobules* around the central artery. The length and breadth of these lobules were estimated by measuring the largest dimensions in a number of sections. The diameter of the clusters was estimated in a similar manner. No allowance was made for shrinkage during fixation and processing.

In each carotid body 500 cells were counted using 5 to 10 high-power fields chosen at random, except that obvious areas of fibrous tissue were avoided. The percentages of the cell types, which are defined below, were noted.

RESULTS

NAKED-EYE APPEARANCES The normal carotid body was found to be a pedunculated ovoid mass lying just behind the bifurcation of the common carotid artery in close apposition to the carotid sinus (Figs 1 and 2). It was commonly situated in a central position (Figs 1a and 2a), but sometimes it was situated just to one side of the bifurcation, usually in the wall of the external carotid artery (Figs 1b and 2b). Usually it was reddish brown in colour, but in both cases of 'cor pulmonale' and one other case of chronic bronchitis and emphysema the carotid body was deeply congested and mauve in colour (Fig. 3). Some of the large carotid bodies were bilobed (Fig. 4a).

CAROTID BODY WEIGHT The weight of the individual carotid bodies ranged from 1·9 to 47·4 mg. The average weight of the right carotid body was 12·9 mg. (SD 10·6). The average weight of the left carotid body was 11·3 mg. (SD 9·6). There was a high degree of correlation between the weight of the right and of the left carotid body ($r=0{\cdot}78$; $P<0{\cdot}01$) (Fig. 5). The weight of the two carotid bodies together ranged from 4·7 mg. to 84·8 mg. (Fig. 6). The mean combined weight was 24·2 mg. (SD 19·0). In the group of patients with emphysema or the Pickwickian syndrome the weight of the combined carotid bodies ranged between 6·0 and 84·8 mg. The mean weight of the combined carotid bodies in this group was 40·6 mg. (SD 24·1). There was no statistically significant difference in the weight of the carotid bodies between the group of cases with emphysema and the rest ($t=1{\cdot}03$; $P>0{\cdot}05$). However, among this group the two patients who had a post-mortem diagnosis of cor pulmonale had combined carotid body

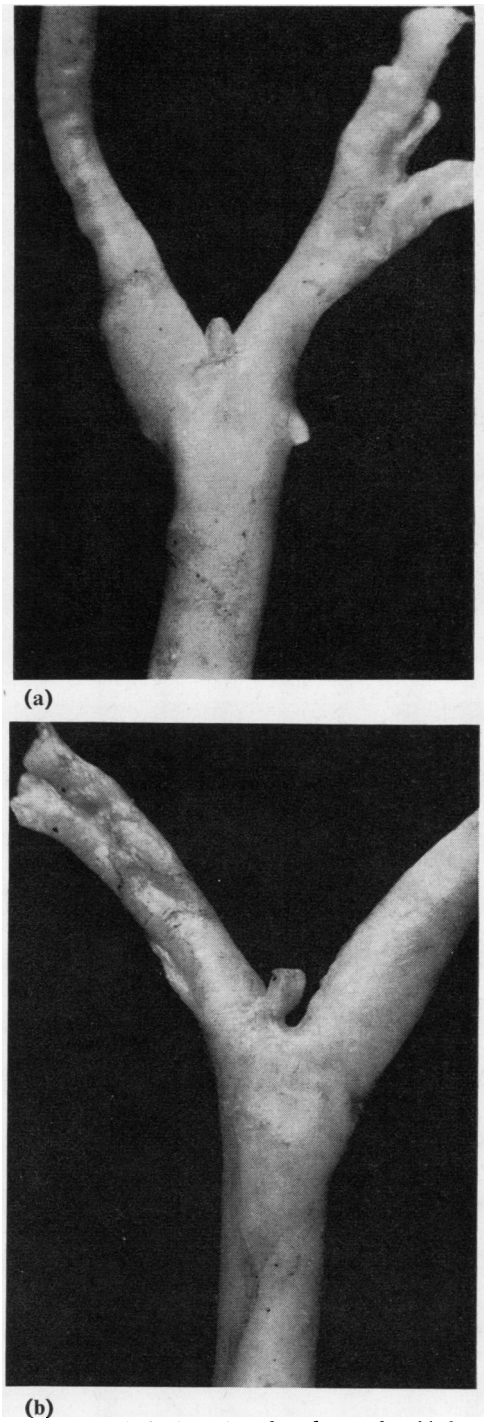
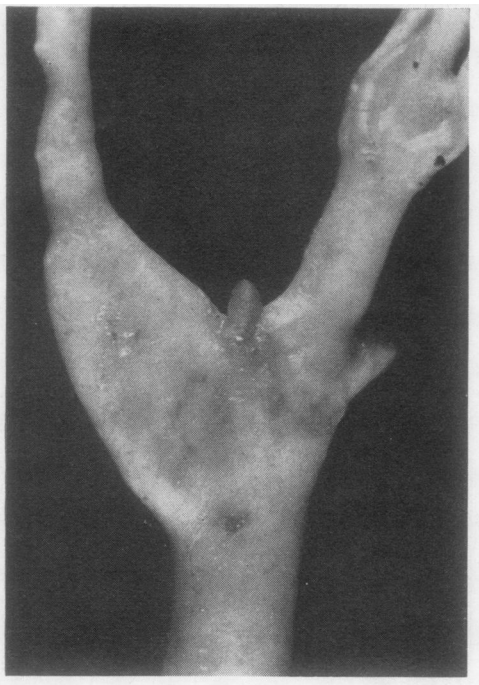
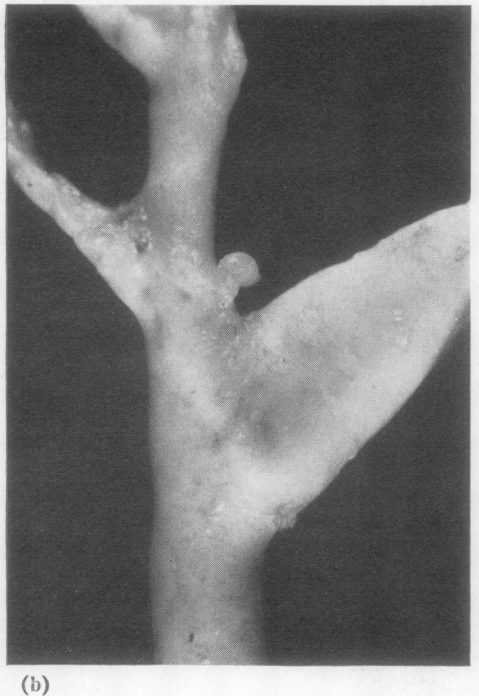


FIG. 1. *Carotid bodies (combined weight 11·6 mg.) from young woman aged 21 years (Case 13); (a) left, (b) right. (Both* × 3.)

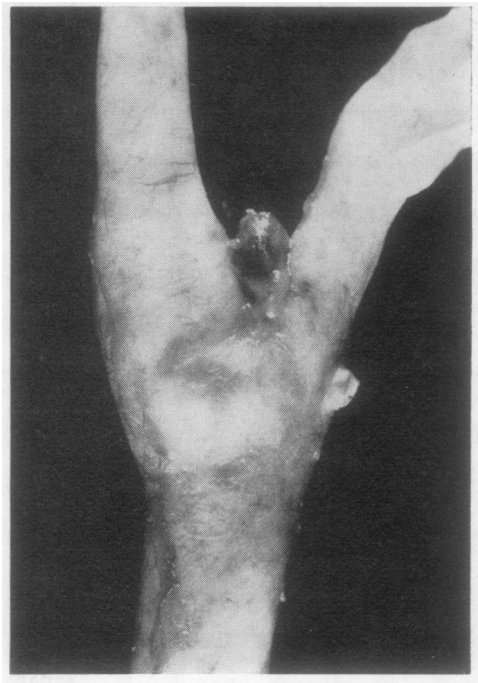


(a)

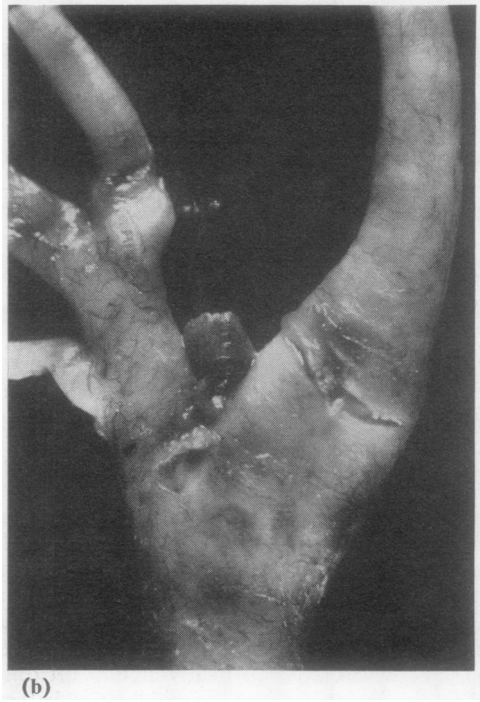


(b)

FIG. 2. *Carotid bodies (combined weight 14·2 mg.) from a man aged 68 years (Case 17); (a) left, (b) right. (Both × 3.)*



(a)



(b)

FIG. 3. *Carotid bodies (combined weight 43·9 mg.) from a man aged 62 years who had panacinar emphysema and left ventricular hypertrophy (Case 34); (a) left, (b) right. (Both × 3.)*

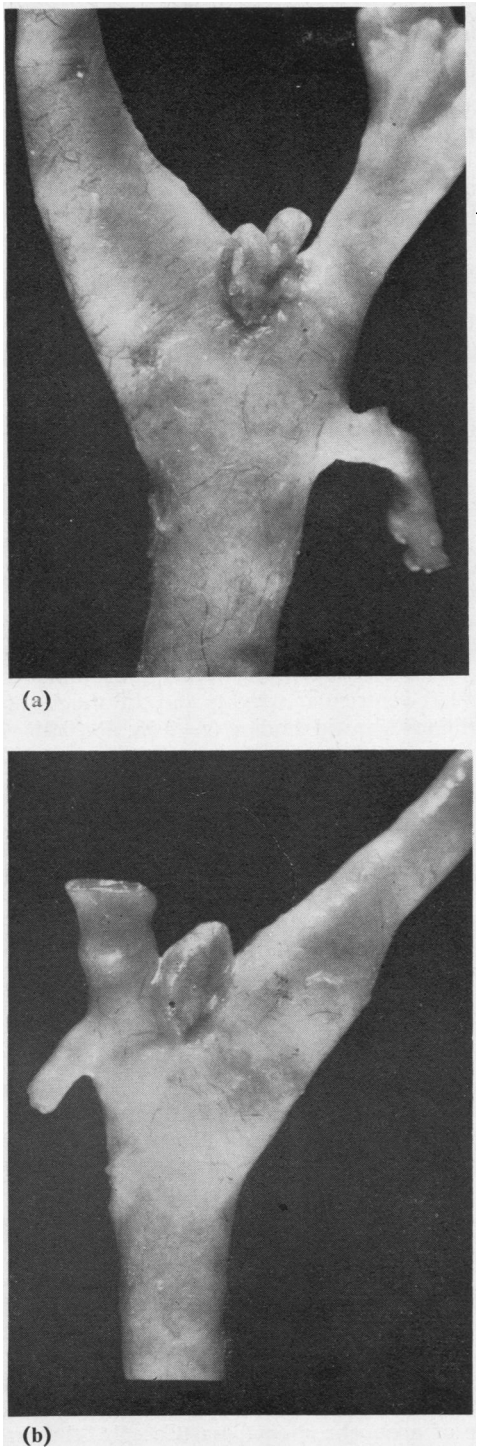


FIG. 4. *Carotid bodies (combined weight 55·8 mg.) from a man aged 58 years who had left ventricular hypertrophy due to systemic hypertension (Case 37); (a) left bilobed carotid body, (b) right. (Both* × 3.)

weights of 60·9 and 68·8 mg., while the combined weight of the carotid bodies in the case of Pickwickian syndrome was 45·5 mg. In this small group, selected because of the high likelihood of chronic hypoxia having occurred during life, the mean weight of the carotid bodies (58·4 mg.) was significantly higher than that (21·4 mg.) of the rest ($t=3·47$; $P<0·01$).

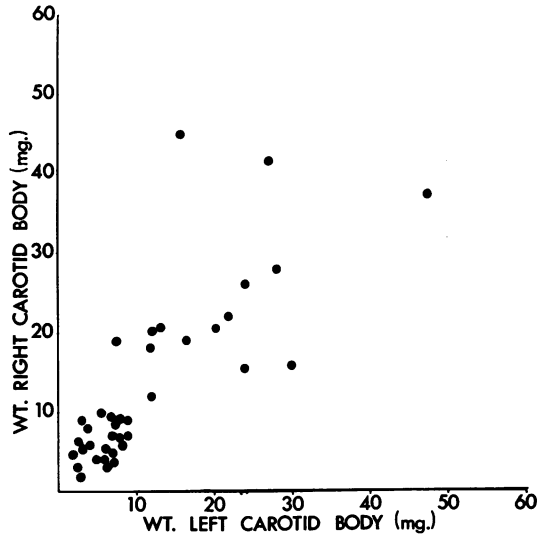


FIG. 5. *Relation between the weights of the right and left carotid body.*

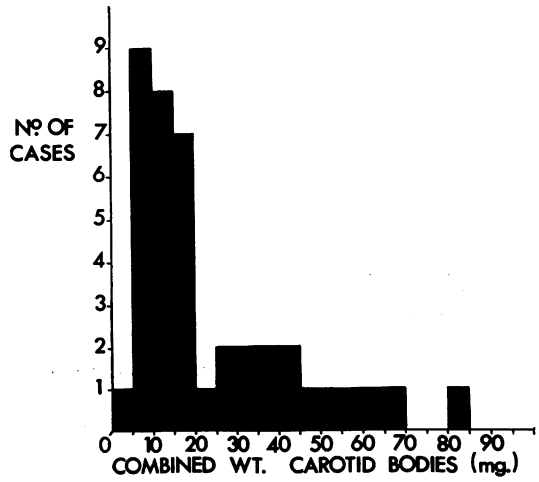


FIG. 6. *Histogram showing distribution of combined weights of both carotid bodies.*

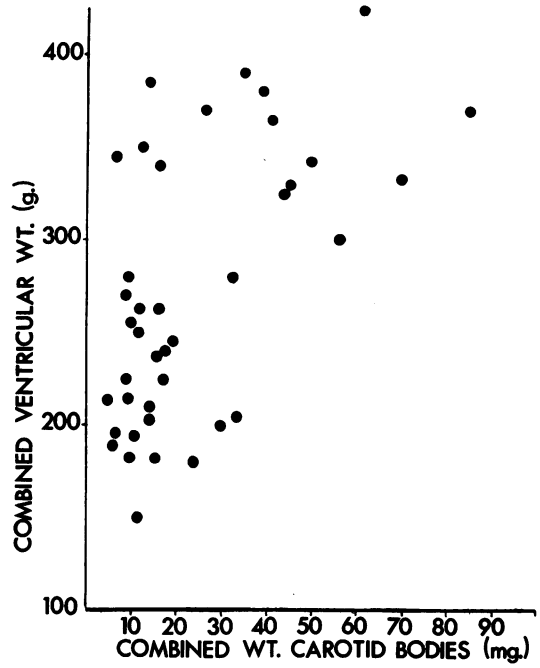


FIG. 7. *Relation between combined ventricular weight and combined carotid body weight in the cases studied.*

TABLE II

DIMENSIONS OF THE CAROTID BODY

	Maximal[1] Diameter (mm.)	Minimal[1] Diameter (mm.)	Depth (mm.)
Whole series (42 carotid bodies from 21 subjects)			
Mean	3·3	2·2	1·7
Standard deviation	1·0	0·6	0·5
Range	1·8–6·2	1·2–3·5	0·7–2·8
Individual cases			
Cor pulmonale (Case 38) ..	5·1	2·8	2·3
Cor pulmonale (Case 39) ..	4·9	2·3	2·0
Pickwickian syndrome (Case 35)	4·0	3·1	2·2

[1] Dimensions at maximal cross-sectional area, assessed by micrometry from serial sections.

The weight of the left ventricle and septum ranged between 115 and 310 g. (mean 201·1 g.; SD 56·9 g.). There were 17 cases with a left ventricular weight in excess of 200 g. which we regard as unequivocally hypertrophied. The weight of the free wall of the right ventricle ranged between 35 and 198 g. (mean 71·0 g.; SD 29·1 g.). There were 10 cases in which the weight of the free wall of the right ventricle exceeded 80 g. which we regard as evidence of unequivocal hypertrophy. Of these only three occurred in the group of subjects with emphysema or the Pickwickian syndrome. The mean right ventricular weight in this group of subjects was 86·0 g. (SD 44·6). The right ventricular mass of the group with emphysema and the Pickwickian syndrome did not differ significantly from the rest of the subjects. The ratio of left ventricular to right ventricular weight ranged from 1·1 to 4·6 (mean 3·0; SD 0·93). The mean ratio in the group of subjects with emphysema or the Pickwickian syndrome was 2·6 (SD 0·78), which was not significantly different from the rest of the subjects. The two subjects with the diagnosis of cor pulmonale, however, had a left ventricular to right ventricular ratio of 1·1 and 1·5.

There was no correlation between the age of the subject and the combined weight of the carotid bodies ($r=0·17$; $P>0·05$). There was a positive correlation between the weight of the left ventricle and the weight of the combined carotid bodies ($r=0·50$; $P<0·01$). There was also a positive correlation between the weight of the right ventricle and the weight of the combined carotid bodies ($r=0·48$; $P<0·01$). There was a higher degree of correlation between the combined right and left ventricular weights and the weight of the combined carotid bodies ($r=0·58$; $P<0·01$) (Fig. 7). A multiple regression equation was derived from least squares:

$$\text{Carotid bodies} = 0·174\ RV + 0·0643\ LV$$

This, however, gave a correlation co-efficient of 0·59 which was not substantially higher than that between the combined ventricular weights and the total carotid body weight. There was no correlation between the ratio of left to right ventricular weight and the weight of the carotid bodies ($r=0·0204$).

HISTOLOGY The carotid bodies were ovoid in shape. Each consisted of a number of lobules arranged around a central fibrous tissue core which contained the main artery of supply. Each lobule was separated from its neighbour by a variable amount of vascular fibrous tissue. In the lobules the parenchymal cells of the organ were arranged in spherical clusters or 'zellballen' surrounded by a cellular fibrous tissue stroma.

The sizes of the carotid bodies and their cell lobules in the 21 cases subjected to histological study are indicated in Table II. The heavier carotid bodies were larger and had bigger lobules. Examples are illustrated in Figures 11 to 15. In a case of phaeochromocytoma (Case 16) the carotid bodies together weighed 14·2 mg. and the average size of the lobules was 500 × 150 μ (Fig. 13). By contrast, in a case of hypoxic cor pulmonale complicating panacinar emphysema (Case 38) the

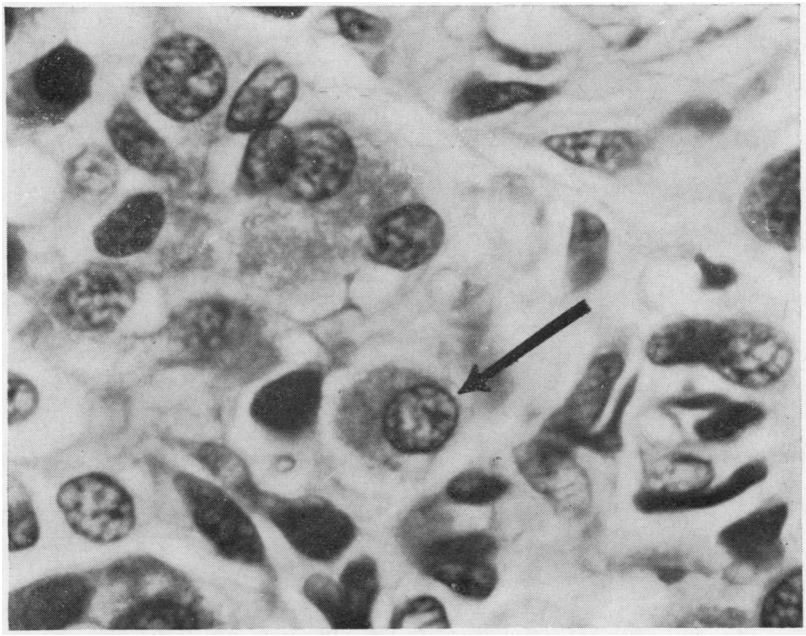


FIG. 8. *A 'light cell' with an ill-defined cytoplasmic outline is seen in the lower centre of the field (arrow). Note the open chromatin pattern of the nucleus. To the left and above this there is a further collection of light cells. (Haematoxylin and eosin.* × 1,500.)

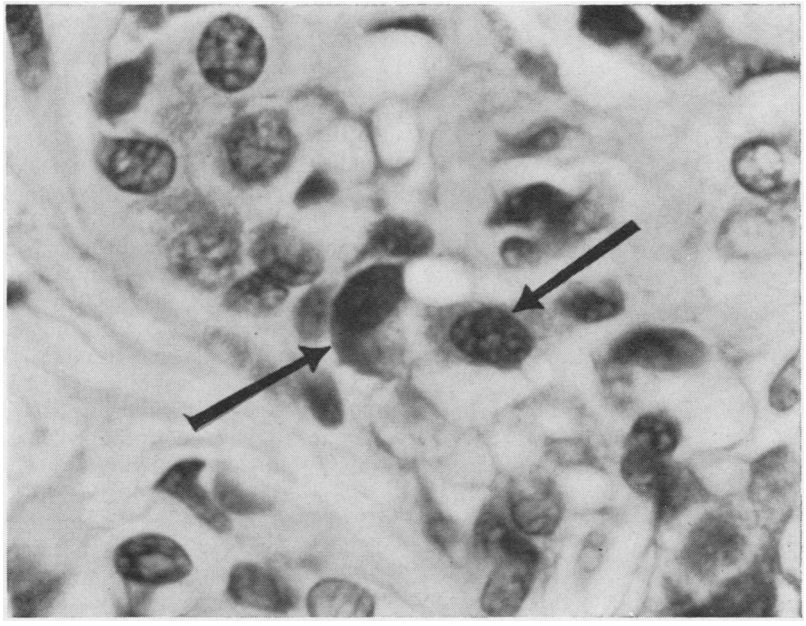


FIG. 9. *Two 'dark cells' (arrows) are present in the centre of the field. The nuclear chromatin pattern is denser than in the light cell and the cytoplasm is more deeply staining. Note the marked vacuolation in the cytoplasm. (H. and E.* × 1,500.)

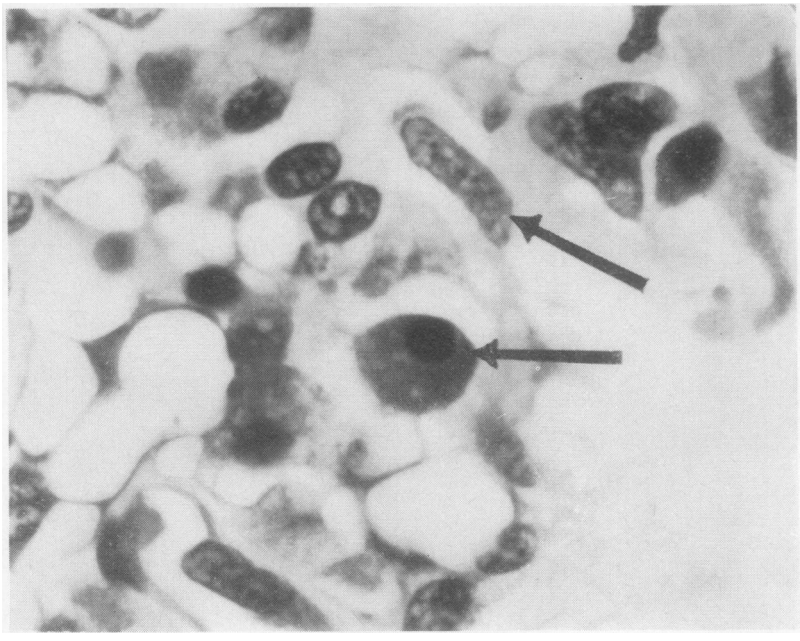


FIG. 10. *A pyknotic cell is situated in the centre of this field (lower arrow). The dark granular cytoplasm and eccentric pyknotic nucleus are well seen. Above the pyknotic cell is a type II cell with an elongated nucleus (upper arrow). (H. and E.* × 1,500.)

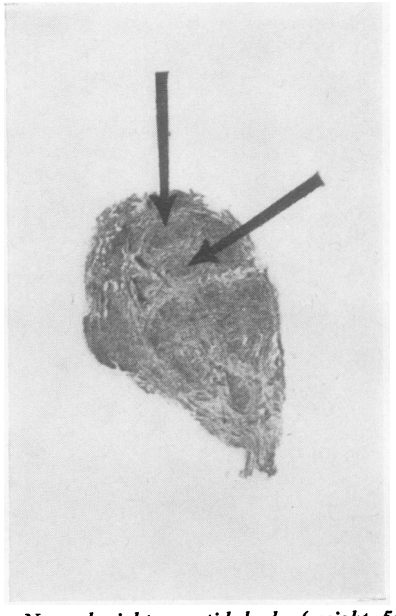


FIG. 11. *Normal right carotid body (weight 5·9 mg.) from a girl of* 14 *years who had a phaeochromocytoma (Case* 16). *This carotid body measured* 2·5 × 1·9 × 1·3 *mm. It consists of lobules of glomic tissue (arrows) separated by fibrous tissue.* (× 12.)

carotid bodies together weighed 60·9 mg. and the average size of the lobules was 1,000 × 1,000 μ (Fig. 14) while there was a concomitant increase in overall size (Figs 11 and 12). The cell clusters were uniform in size in all the cases studied, their diameter being about 100 μ.

The parenchymal cells of the carotid body were of two types, the type I or 'chief' cell, and the type II or 'sustentacular' cell. Type I cells were found to exist in three distinct forms, which may be termed the light cell, the dark cell, and the pyknotic cell. The light cell was the most abundant. It was about 13 μ in diameter and had an ill-defined cytoplasmic outline. The cytoplasm was pale and eosinophilic and showed a variable number of large vacuoles up to 7 μ in diameter. Its nucleus had an open chromatin pattern and was 7 μ in diameter (Fig. 8).

The dark type I cell also had a diameter of about 13 μ, but the cytoplasmic outline was better defined, and the cytoplasm stained more deeply with eosin. Cytoplasmic vacuolation similar to that seen in the light cell also occurred in this type of cell. The nucleus was ovoid in shape and measured 7 μ × 4·5 μ. In contrast to the light cell the chromatin pattern of the nucleus had a denser and more granular pattern (Fig. 9).

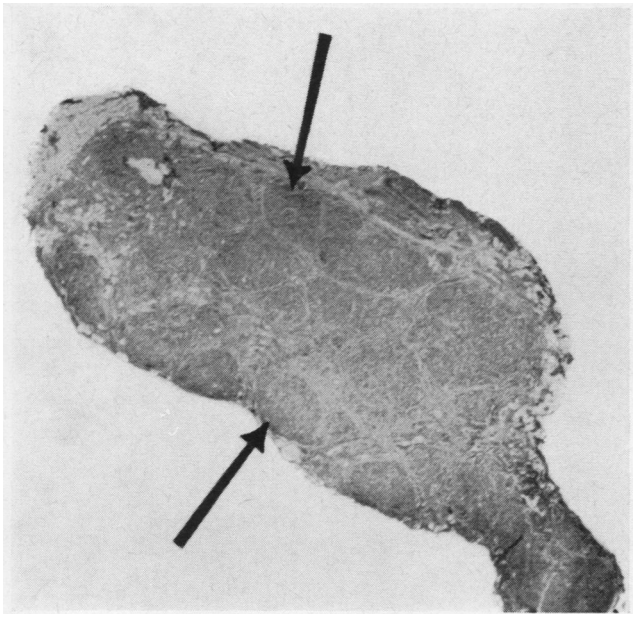


FIG. 12. *Enlarged right carotid body (weight 45·3 mg.) from a man of 72 years who died from hypoxic cor pulmonale complicating panacinar emphysema (Case 38). This carotid body measured 6·2 × 3·3 × 2·5 mm. The lobules of glomic tissue are enlarged compared to those of the normal carotid body shown in Fig. 11 (× 12.)*

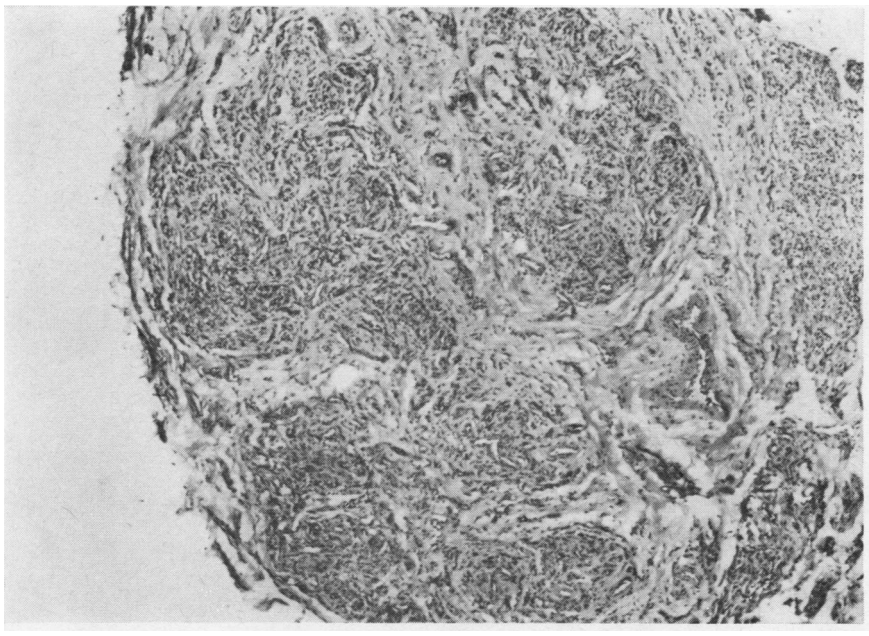


FIG. 13. *Section of normal right carotid body (weight 5·9 mg.) from Case 16 illustrated in Fig. 11. Lobules of glomic tissue are separated by fibrous tissue. The average dimensions of lobules in this carotid body were 500 × 250 μ. (H. and E. × 60.)*

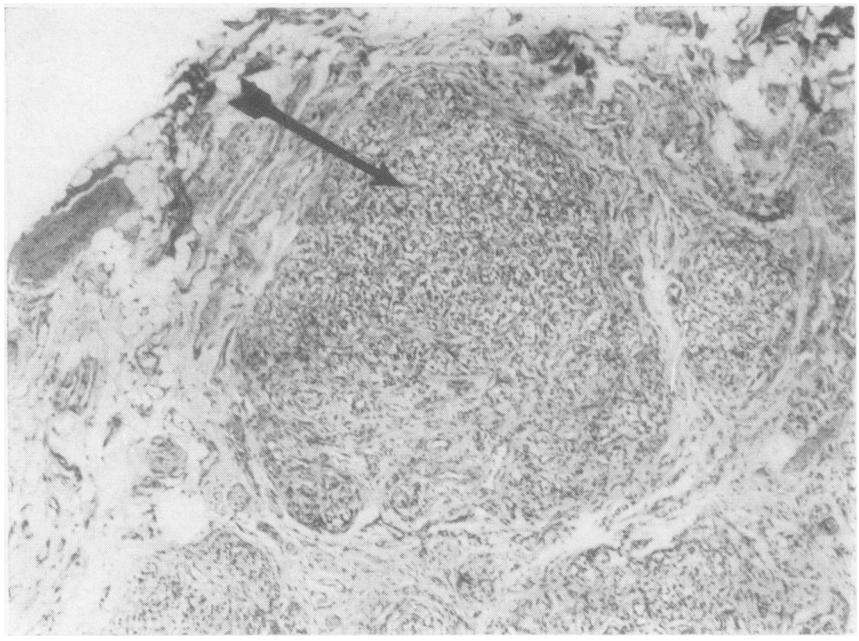


FIG. 14. *Sections of enlarged right carotid body (weight 45·3 mg.) from Case 38 illustrated in Fig. 12. The enlarged lobule of glomic tissue is about 1,000 μ in diameter. Compare its size with that of normal lobules shown in Fig. 13. Part of the enlarged lobule indicated by an arrow shows cellular hyperplasia. (H. and E. × 60.)*

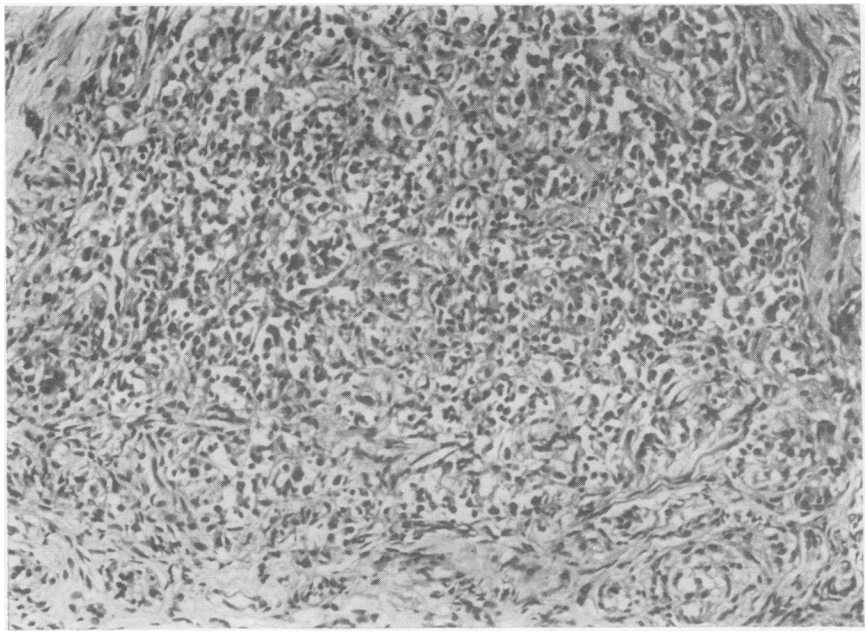


FIG. 15. *Part of Fig. 14 at higher magnification showing that the hyperplasia of the upper portion of the lobule involves the 'dark' variety of type I (chief) cells. (H. and E. × 150.)*

The third form of type I cell was the pyknotic cell, which had a sharp cytoplasmic outline and an overall diameter of about 10 μ. The cytoplasm was deeply eosinophilic and granular, and vacuolation was fairly common. The nucleus was deeply basophilic, apparently structureless and excentrically situated. Its diameter was about 4 μ (Fig. 10).

The type II cell (Fig. 10) had an elongated nucleus up to 13 μ long and up to 4 μ wide. The chromatin was arranged in small clumps which gave a pale, dusty appearance. In paraffin sections the cytoplasm was difficult to see, but it is worth noting here that on electron microscopy this type II cell can be shown to be wrapped around small aggregates of type I cells by means of cytoplasmic processes (Grimley and Glenner, 1968).

The significance of vacuolation in the variants of type I cells is not clear at present. When we examined the carotid body of rats within five minutes of death there was no vacuolation in the cytoplasm of this type of cell. After this period, however, cytoplasmic vacuolation increased and was pronounced in the carotid body of a rat that had died two hours previously. This suggests to us that vacuolation may be an expression of autolysis rather than of any metabolic activity in life. However, such vacuolation seems to occur too quickly after death to be attributable entirely to the usual processes of post-mortem change.

The results of differential cell counts on the carotid bodies in 21 of the 40 cases are shown in Table III. In general the increase in size of the carotid bodies could not be attributable to hyperplasia of any one cell type. However, in one of the cases of 'cor pulmonale' hyperplastic nodules were seen in the right carotid body (Fig. 15). The nodules protruded beyond the rounded contours of the enlarged glomic lobules. A differential count of these hyperplastic nodules revealed a predominance of the dark variety of type I cells (Table III ; Fig. 15).

TABLE III

DIFFERENTIAL CELL COUNT OF CAROTID BODIES

	Type I (%)			Type II (%)
	Light	Dark	Pyknotic	
Whole series (41 carotid bodies from 21 subjects)				
Mean	39·7	11·2	5·9	43·2
Standard deviation ..	8·5	8·3	6·0	7·7
Individual cases				
Cor pulmonale (Case 39)	52	6	1	42
Cor pulmonale (Case 38)	40	12	0	48
(Hyperplastic area) (Case 38)	9	70	3	18
Pickwickian syndrome (Case 35)	30	7	13	50

DISCUSSION

Our experience confirms that the technique of dissection and weighing of the carotid bodies introduced by Arias-Stella (1969) is valid and useful. Histological studies confirmed the accuracy of the dissection. The high degree of correlation between the weights of the right and left carotid bodies defines the limits of experimental error and biological variation. This high degree of correlation also suggests that enlargement of the carotid bodies has a constitutional rather than a local origin.

The original purpose of the investigation was to determine whether the weight of the carotid bodies was greater in patients with diffuse pulmonary disease. To this question the investigation has not given a clear-cut answer. Perhaps this is inevitable since many patients with generalized pulmonary emphysema do not suffer from a signicant degree of arterial hypoxaemia. It may be noted, for instance, that the group of subjects with diffuse pulmonary disease included three females all of whom were over the age of 85, and the weights of the carotid bodies in these three subjects came amongst the four lowest weights recorded in this group. Conversely, the two subjects with clear-cut cor pulmonale had unusually high carotid body weights (60·9 and 68·8 mg.) while the subject with Pickwickian syndrome also had a high carotid body weight (45·5 mg.). The patient with the carotid body weight of 60·9 mg. was known to have a systemic arterial P_{O_2} of 36 mm. Hg and P_{CO_2} of 54 mm. Hg during life, and presumably the other two also suffered from chronic hypoxaemia. It is noteworthy that, in this small group, selected specifically on account of chronic hypoxaemia rather than diffuse pulmonary disease, the mean weight of the carotid bodies was significantly higher than that of the rest.

The observations of Arias-Stella (1969) also suggest that chronic hypoxia is a cause of enlargement of the carotid bodies. He studied the weight of the carotid bodies in two groups of subjects in Peru, both groups consisting of 25 persons of comparable ages. One group came from Cerro de Pasco, situated at an altitude of 14,300 feet above sea level in the Peruvian Andes. The second group came from Lima at sea level. Arias-Stella found that, in the low altitude group, often one or both carotid bodies were not identifiable. In contrast

to this the carotid bodies in the high altitude dwellers were readily found and proved to be heavier and larger. In some instances the carotid bodies from persons living at Cerro de Pasco were greater than 1·5 cm. in diameter. One obvious possible interpretation of his findings is that the enlargement of the carotid bodies at high altitude is associated with chronic hypoxia brought about by low barometric pressure.

An unexpected finding in this investigation was the significant correlation between the weight of the carotid bodies and the weights of the right and left ventricles. Possibly the relation with right ventricular weight might have been expected since none of these subjects had any anatomical abnormality affecting the work of the right ventricle, and right ventricular hypertrophy might therefore be reasonably related to the occurrence of pulmonary hypertension, which in turn would signify diffuse pulmonary disease. The relation between the weight of the carotid bodies and the weight of the left ventricle, however, is entirely inexplicable on such grounds. It does not seem likely that this enlargement of the carotid bodies was due to changes in the wall of the carotid arteries secondary to hypertension, since in two cases of aortic stenosis there was a notable enlargement of the carotid bodies (39·2 and 40·7 mg.) (Cases 32 and 33). Neither does it seem likely that the relation between the weight of the ventricles and the weight of the carotid bodies is simply a result of a mutual link with the weight of the whole body. Thus, although the weight of the ventricles in normal subjects is known to vary with the weight of the cadaver (Hudson, 1965), the range of ventricular weight (180–425 g.) over which the correlation with carotid body weight existed far exceeded the normal. Such considerations led us to wonder if an increase in the size of the carotid bodies was related in some fashion to hypertrophy of the myocardium, whether it be in the right or in the left ventricle. The rather higher degree of correlation between the combined weights of the ventricles and the weight of the carotid bodies seemed to support this suggestion. The meaning of such a correlation is at present obscure. But it is conceivable that glomic tissue might in some way be stimulated by substances produced by a hypertrophy in the myocardium or that glomic tissue possesses endocrine properties concerned with myocardial hypertrophy. It may be noted that Pearse (1969) includes the carotid body type I cell in the APUD series of cells for which, on cytological grounds, a hormonal function may be predicted.

On the other hand, it cannot be said that the hyperplasia of the glomic tissue was in general associated with a particular form of cell. The exception to this general rule was the patient with 'cor pulmonale' (Case 38), in whom hyperplastic nodules showed a predominance of the dark variety of type I cells. Arias-Stella (1969) stated that in his cases from high altitude sections of the carotid body showed no histological abnormality.

We conclude that, when either or both the ventricles are abnormally large, we expect there to be hyperplasia of the carotid bodies, though usually no specific cell type is concerned in the hyperplasia. The meaning of this observation remains to be revealed.

REFERENCES

Arias-Stella, J. (1969). Human carotid body at high altitudes. Item 150 in the 69th Programme and Abstracts of the American Association of Pathologists and Bacteriologists, San Francisco, California.

Fulton, R. M., Hutchinson, E. C., and Jones, A. Morgan (1952). Ventricular weight in cardiac hypertrophy. *Brit. Heart J.*, **14**, 413.

Grimley, P. M., and Glenner, G. C. (1968). Ultrastructure of the human carotid body. A perspective on the mode of chemoreception. *Circulation*, **37**, 648.

Hudson, R. (1965). *Cardiovascular Pathology*, vol. 1, p. 34. Arnold, London.

Pearse, A. G. E. (1969). The cytochemistry and ultrastructure of polypeptide-producing cells of the APUD series and the embryologic, physiologic and pathologic implications of the concept. *J. Histochem. Cytochem.*, **17**, 303.