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Chronic Cor Pulmonale

Weight and Intraventricular Volume of the Right Ventricle in Chronic Pulmonary Diseases

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Summary. 1. The weights and intraventricular volumes of the right ventricles have been determined from 198 hearts from patients with pulmonary diseases.

2. For diagnosis of concentric or eccentric cardiac hypertrophy the relative volume (the degree of dilatation) of a ventricle, i.e., intraventricular volume in ml/100 g ventricular muscle mass, serves as the most important criterion.

3. In cases of chronic cor pulmonale the hypertrophic right ventricle is more likely to be concentric than normal right ventricles, which have a postnatal physiological structural dilatation.

4. The adaptative hypertrophic growth of the right ventricle is associated with an increasing absolute intraventricular volume but the relative volume (the degree of dilatation) decreases.

5. As a result of pathological pressure load in chronic cor pulmonale the right ventricle develops a shape which resembles the normal left ventricle. This "left-ventricularisation" of a hypertrophied right ventricle is more pronounced in younger individuals.

6. In extreme systemic arterial hypertension the blood pressure rises to twice the normal and the weight of the hypertrophied left ventricle is 3–4 times above normal. In cor pulmonale blood pressure values can be 4–5 times and the weights of the hypertrophied right ventricle 5–6 times greater than normal.

7. Chronic failure of the hypertrophic left ventricle is combined with an increasing relative ventricular volume (i.e. eccentric structural dilatation). Failure of the hypertrophic right ventricle can develop although the relative ventricular volume of the degree of dilatation is less than in the normal (concentric hypertrophy compared with the normal right ventricle).

8. Calculations indicate that chronic cor pulmonale would require a weight of right ventricle muscle exceeding 10 times normal in order to maintain a long-term compensation.

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9. Despite the good coronary blood supply of the right ventricle, with its excellent adaptative growth, hypertrophy accompanied by a decrease in the degree of dilatation is insufficient to compensate for the pressure load in chronic cor pulmonale and the primary physiological structural dilatation.

Key words: Chronic cor pulmonale – Weight – Intraventricular volume – Hypertrophy – Right heart failure.

Zusammenfassung. 1. Muskuläres Gewicht und absoluter Inhalt des rechten Ventrikels wurden an 198 totenstarren Herzen von 134 Männern und 64 Frauen mit chronischen Lungenerkrankungen bestimmt.

2. Der relative Inhalt eines Ventrikels (absoluter Inhalt in ml/100 g Ventrikelmuskulatur = Dilatationsgrad) gestattet eine genaue Unterscheidung von konzentrischer und exzentrischer Hypertrophie.

3. Der hypertrophierte rechte Ventrikel ist bei chronischem Cor pulmonale konzentrischer als der normale rechte Ventrikel mit physiologischer postnataler Gefügedilatation.

4. Die adaptive Gewichtsvermehrung des druckbelasteten rechten Ventrikels geht mit einer Vergrößerung des absoluten Inhaltes,aber einer Verminderung des relativen Inhaltes einher.

5. Mit zunehmender Hypertrophie nähert sich bei chronischem Cor pulmonale das Aussehen des rechten Ventrikels dem linken (Links-Ventrikularisation). Das gilt besonders für die Herzen von Jugendlichen.

6. Bei chronischem Cor pulmonale kann die Druckerhöhung das 4–5fache der Norm erreichen. Die Gewichte des hypertrophierten rechten Ventrikels können das 5–6fache der Norm betragen. Im großen Kreislauf steigt dagegen der Blutdruck nur um etwa das Doppelte, und die Gewichte des linken Ventrikels erreichen nur Werte, die maximal um das 3–4fache höher als in der Norm liegen.

7. Die chronische Insuffizienz des hypertrophierten linken Ventrikels geht mit einer exzentrischen Hypertrophie (Vergrößerung des relativen Inhaltes) einher. Die chronische Insuffizienz des hypertrophierten rechten Ventrikels bei chronischem Cor pulmonale entwickelt sich im Stadium der konzentrischen Rechtshypertrophie (Verminderung des relativen Inhaltes).

8. Berechnungen zeigen, daß die Gewichtsvermehrung des rechten Ventrikels bei hochgradiger pulmonaler Hypertonie mehr als das 10fache der Norm ausmachen müßte, um eine langdauernde volle Kompensation zu erreichen.

9. Das Ausmaß der konzentrischen Hypertrophie bei chronischem Cor pulmonale reicht trotz guter Blutversorgung und einem sehr guten adaptativen Wachstum nicht aus, um die primäre postnatale physiologische Gefügedilatation und die zunehmende Druckbelastung zu kompensieren.

Introduction

The definition of pulmonary heart hypertrophy given by Kirch (1924) corresponds to chronic cor pulmonale as later defined by the WHO (1961): "Hyper-

trophy of the right ventricle resulting from diseases affecting the function and/or the structure of the lung, except when these pulmonary alterations are the result of diseases that primarily affect the left side of the heart or of congenital heart disease."

The incidence of chronic cor pulmonale is about 1 to 4,5% in large series of autopsies (Griggs et al., 1939; Scott and Garvin, 1941; Zimmermann and Ryan, 1951; McKeown, 1952; Gelfand, 1955; Kirch, 1955; Goerttler, 1965). However, the frequency of chronic pulmonary disease in industrial areas differs from that in rural areas, and the occurrence of chronic cor pulmonale therefore displays considerable variability. It may represent as high a proportion as 50% of all cardiac disease (Walzer and Frost, 1954; Denolin, 1955; Matthes et al., 1960; WHO, 1961).

Hypertrophy of the right ventricle in chronic cor pulmonale may be either compensated or accompanied by right heart failure. The pathological alterations due to adaptation of the right ventricle in chronic cor pulmonale are described by Kirch (1924, 1933, 1955), Schepers (1957), Goerttler (1965), Hort (1965) and Giese (1966). However, there have been no systematic investigations of weights or volumes of right ventricles in chronic cor pulmonale.

Material and Methods

We investigated 198 cases (134 male and 64 female) of chronic pulmonary disease in cases of chronic pulmonary emphysema, interstitial pulmonary fibrosis, chronic pneumonia, chronic tuberculosis of the lung, pneumoconiosis, recurrent pulmonary embolism, kyphoscoliosis and primary pulmonary sclerosis. The right ventricles were examined in rigor mortis.

Our study was made on hearts from autopsies performed not later than 24 h post mortem. We excluded those with congenital heart disease, eccentric left ventricular hypertrophy with left sided heart failure, myocarditis, myocardial infarction or excessive lipomatosis.

In addition to the macroscopic determination of rigor mortis we measured the mean distance between Z-bands (= mean length of sarcomeres) in frozen sections. The mean length of sarcomeres of the right ventricle measured in rigor mortis is about 1.6 μ (Linzbach and Linzbach, 1951; Linzbach, 1960; Linzbach, 1967). Contraction in rigor mortis is more marked than in systole where the average sarcomere length is estimated to be about 1.65 to 1.7 μ. Measurements of the length of sarcomeres were made at different sample points in the right ventricles of 600 cases. In this investigation we excluded all hearts with a mean Z-band distance of more than 1.6 μ.

Chronic hypertrophic left-sided heart failure was diagnosed microscopically by demonstrating the changes seen in chronic pulmonary congestion with induration of the lungs, pulmonary hemosiderosis and pulmonary sclerosis. Chronic hypertrophic right-sided failure was confirmed by hepatic changes including atrophy of liver cells in the neighbourhood of central veins, congestive bridging and hepatic fibrosis.

After removing blood clots from the ventricles we filled them carefully with cellulose paper soaked in formaldehyde. We tried to avoid all pressure distortions in order to preserve the original shape. The hearts were then fixed for 5–7 days in a 5% solution of formaldehyde.

In our 10 control cases the loss of weight after fixation of the right ventricular muscle mass, excluding the epicardial fatty tissue, amounted to 2–3%. We measured right ventricular volume, after fixation, by filling with water up to the base of the tricuspid valves. We then carefully removed fatty tissue, the trunks of blood vessels and the extramural branches of coronary arteries, dissected the free portion of the right ventricle at the junction of the right ventricle wall with the septum and weighed the free portion.

The degree of dilatation, i.e., the relative volume of the right ventricle in rigor mortis, has been defined as intraventricular volume in ml/100 g ventricular

muscle mass (Linzbach, 1967). This variable alone determines whether a harmonic, a concentric or an eccentric hypertrophy of a ventricle is present. Depending on the degree of dilatation three forms of ventricular hypertrophy can be distinguished.

1. Harmonic Hypertrophy

The relative ventricular volume corresponds more or less to the norm. The absolute ventricular volume and the ventricular muscle mass increase in about equal proportions. The typical example of harmonic hypertrophy is the athlete's heart.

2. Concentric Hypertrophy

The relative ventricular volume is less than normal. It follows that the increase in muscle mass is greater than the increase in absolute volume. This mechanism corresponds to the state of compensated pressure hypertrophy.

3. Eccentric Hypertrophy

The relative ventricular volume is greater than normal, and the increase in absolute volume is greater than the increase in muscle mass. This phenomenon can be diagnosed in cases of chronic structural dilatation (Gefügedilatation) of a ventricle, e.g., hypertrophy following myocarditis, volume hypertrophy, or decompensated pressure hypertrophy.

Control values were provided by the weights and volumes of the right ventriles of 34 males and 48 females, without chronic pulmonary diseases, who died between 1–18 h after traffic accidents or brain surgery (Table 1).

Our data were evaluated by calculating regression lines and correlation coefficients. In addition we computed the mean values, standard deviations, variance and relative and absolute frequency distributions.

Table 1

	Total $n=62$	Male $n=34$	Female $n=28$
Weight of the right ventricle	(55 ± 18) g	(62 ± 20) g	(47 ± 12) g
Absolute volume of the right ventricle	(44 ± 18) ml	(49 ± 19) ml	(36 ± 14) ml
Relative volume of the right ventricle	(82 ± 31) ml/100 g	(82 ± 28) ml/100 g	(81 ± 36) ml/100 g

Conventionally, good correlation obtains for correlation coefficients greater than or equal to 0.7.

We used the allometric growth formula:

$$y = k \cdot x^{\alpha} \tag{1}$$

as a mathematical model. With this formula the relative growth of linear, two dimensional and three dimensional quantities in any organ in any arbitrary combination can be determined. In a double logarithmic plot the formula:

$$\log y = \log k + \alpha \cdot \log x \tag{2}$$

is a straight line. y stands for a dimension of any part of an organ, x stands for a dimension of an entire organ. k is a constant, defined as the initial growth index. The exponent α is the allometric constant of growth, whose value corresponds to the tangent of the angle of ascent of the linear growth curve. The value of the growth constant indicates whether a part of an organ grows at the same rate (isometry, i.e., harmonic growth), more rapidly (positive allometry), or more slowly (negative allometry) than the entire organ.

All mathematical analyses were carried out on an 1108 Univac computer of the "Gesellschaft für Wissenschaftliche Datenverarbeitung mbH, Göttingen".

Results

1. Weights and Absolute Volume of the Right Ventricle

Figure 1 shows in a double logarithmic plot that the absolute volume of the right ventricle increases with increasing weight of the free portion of the right ventricular muscle mass. The regression line is $\log y = 0.234 + 0.768 \log x$, the correlation coefficient is $r = 0.704$.

The absolute volume, however, increases at a slower rate (negative allometry) than does the weight of the free portion of the right ventricular muscle mass.

In harmonic growth of two quantities having the same dimension the allometric constant $\alpha = 1$. Assuming that abscissa and ordinate are graduated in identical units then $\alpha = 1$ corresponds, in a double logarithmic plot, to a straight line with a angle of ascent whose tangent is 1, i.e., $\alpha = 45°$. The observed angle of ascent for the regression line of our data has a tangent of 0.768, however, corresponding to an α of 34.5°. The difference between the rise of the regression line and the rise of the line for harmonic growth is significant.

2. Weight and Relative Volume (Degree of Dilatation) of the Right Ventricle

To show how the values of the relative volume in cases of chronic cor pulmonale compare with the normal mean value of the relative volume, i.e., the normal degree of dilatation of the right ventricle (82 ml/100 g), a straight line has been drawn in the diagram, in addition to the regression line. It can be stated that

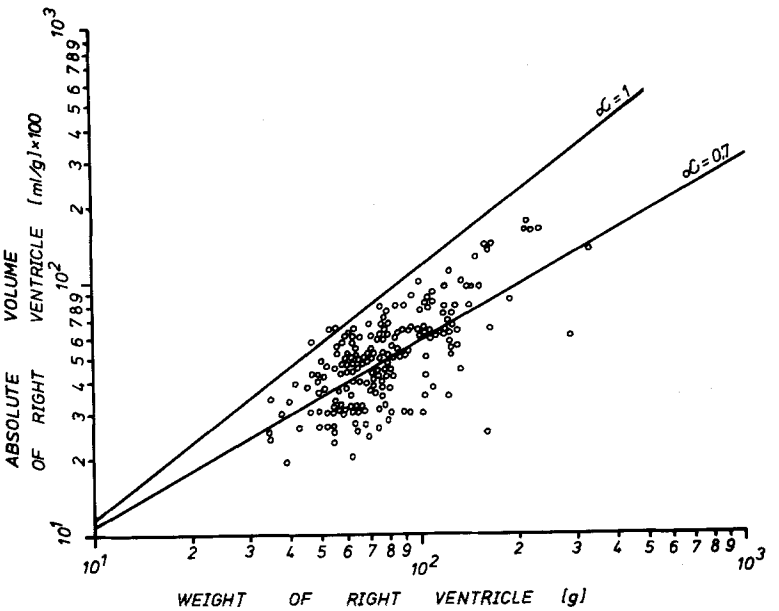

Fig. 1. Ordinate (logarithmic): The absolute volume of the right ventricle. Abscissa (logarithmic): The free portion of the right ventricular muscle mass. Data from 198 human hearts in cases of chronic pulmonary diseases. The angle of ascent of the upper straight line, α, is less than 45° because the units are different for the abscissas and ordinates

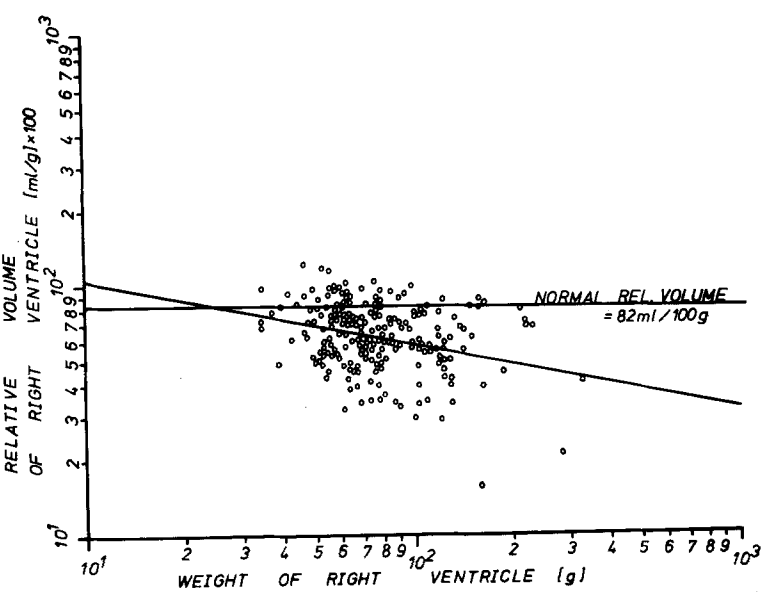

Fig. 2. Ordinate (logarithmic): The relative volume of the right ventricle. Abscissa (logarithmic): The weight of the free portion of the right ventricular muscle mass. Data from 198 human hearts in cases of chronic pulmonary diseases. The straight line drawn in addition to the regression line gives the mean value of the normal relative volume of the right ventricle (82 ml/100 g)

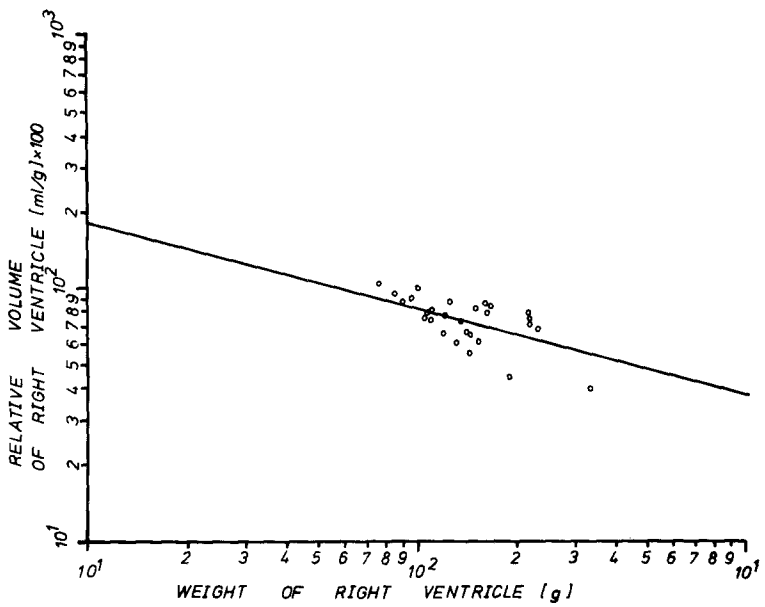

Fig. 3. Ordinate (logarithmic): The relative volume of the right ventricle. Abscissa (logarithmic): The weight of the free portion of the right ventricle muscle mass. Data from 28 human hearts in cases of chronic pulmonary diseases with absolute volumes of the right ventricle of 80 ml or more

the degree of dilatation in severe hypertrophy of the right ventricle is always less than or equal to the normal value of 82 ml/100 g. The regression line is $\log y = 2.234 - 0.232 \log x$ (Fig. 2). The correlation coefficient r is only -0.287 (data from 198 hearts).

The correlation between the relative volume, i.e., the degree of dilatation of the right ventricle and the weight of the muscle mass is shown in Figure 3 for right ventricles with an absolute volume of 80 ml or more. The regression line is $\log y = 2.596 - 0.337 \log x$. The correlation coefficient is $r = -0.701$.

Thus, we observed a decrease in the relative volume as a function of muscle mass. This decrease was particularly pronounced ($r = -0.7$) in hypertrophied right ventricles having large absolute volumes. This means that in chronic cor pulmonale with increasing weight of the right ventricle concentric hypertrophy gets more and more marked, in spite of a large absolute intraventricular volume compared with controls.

3. The Onset of Chronic Right Heart Failure

Table 2 shows the mean values and standard deviations of our measurements ordered in weight groups for the right ventricle, for all data, and separately for males and females. It can be seen from the last column that the microscopic diagnosis of chronic liver congestion implies chronic right heart failure.

Sinusoidal dilatation and pressure atrophy of the intervening liver cells near the central vein is indicated by a + sign.

Table 2

	Number of the right ventricles	Mean age	Mean weight of the right ventricle (g)	Mean absolute volume of the right ventricle (ml)	Mean relative volume of the right ventricle (degree of dilatation) (ml/100 g)	Number of cases displaying chronic liver congestion		
						+	++	+++
Total number of cases ♂+♀	198	62±17	86±42	55±28	64±21	14	24	17
Weight classes								
< 50 g	15	65±23	42± 5	33±10	74±27	–	–	–
50– 74 g	82	63±17	62± 7	42±12	66±12	3	1	–
75– 99 g	48	63±17	82± 6	53±16	64±18	6	6	1
100–150 g	39	63±12	118±13	68±20	58±17	4	13	8
>150 g	14	49±19	201±55	118±45	61±24	1	4	8
Total number of cases ♂	134	55±16	90±45	58±29	65±20	10	12	13
Weight classes								
< 50 g	4	72±15	44± 5	31±10	71±19	–	–	–
50– 74 g	57	61±18	62± 7	42±12	66±22	2	–	–
75– 99 g	35	58±17	83± 7	55±15	67±18	3	2	1
100–150 g	27	63±12	118±14	72±20	61±17	4	6	7
>150 g	11	45±19	205±60	120±48	61±27	1	4	5
Total number of cases ♀	64	67±17	78±36	48±22	62±22	4	12	4
Weight classes								
< 50 g	11	63±25	42± 5	34±10	75±30	–	–	–
50– 74 g	25	67±18	61± 7	41±10	66±21	1	1	–
75– 99 g	13	77± 7	80± 4	45±14	55±16	3	4	–
100–150 g	12	63±14	118±10	59±18	50±15	–	7	1
>150 g	3	66± 5	188±34	113±40	59±14	–	–	3

Chronic passive liver congestion with congestive bridges is shown by a ++ sign and cardiac sclerosis of the liver by +++.

From the data presented in Table 2 it follows that 27% of the cases in the weight group between 75 g to 99 g have already developed right heart failure. The incidence of right heart failure increases considerably when the free portion of a right ventricle muscle weighs 100 g or more. In a group of 39 right ventricles with a weight of the free part between 100 and 150 g as many as 25 (64%) had signs of chronic right heart failure in the liver. In 13 (93%) of 14 right ventricles with a ventricular weight surpassing 150 g, evidence of chronic passive congestion of the liver was found. In one single case, involving the right ventricle of a 14 year old boy suffering from primary pulmonary sclerosis, no signs of chronic passive liver congestion were noted. The weight of the free muscular

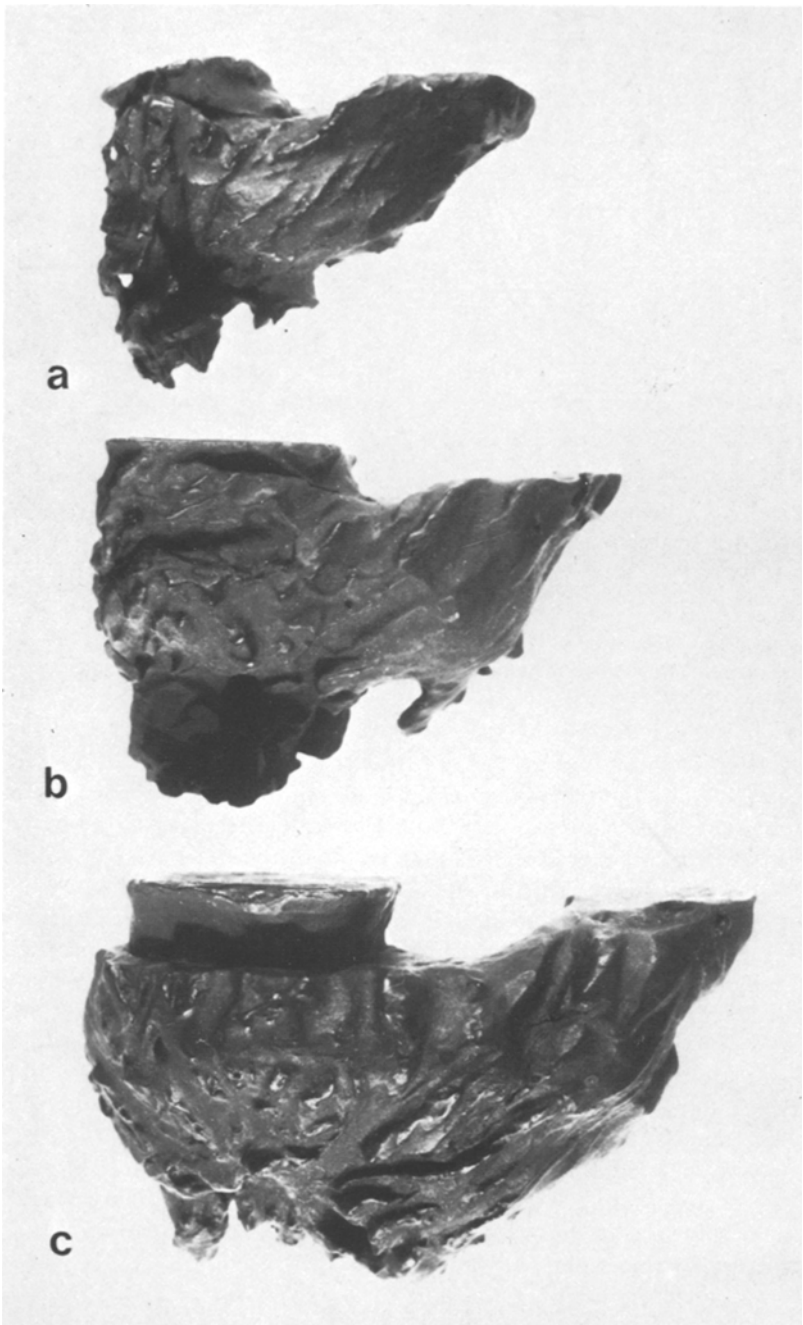

Fig. 4a–c. Lateral view of plaster moulds of right ventricles. The outflow tract is at the right, the inflow tract on the left. The weights of the free muscle of the right ventricle and the absolute volumes are: **a** 45 g, 40 ml; **b** 85 g, 56 ml; **c** 180 g, 118 ml

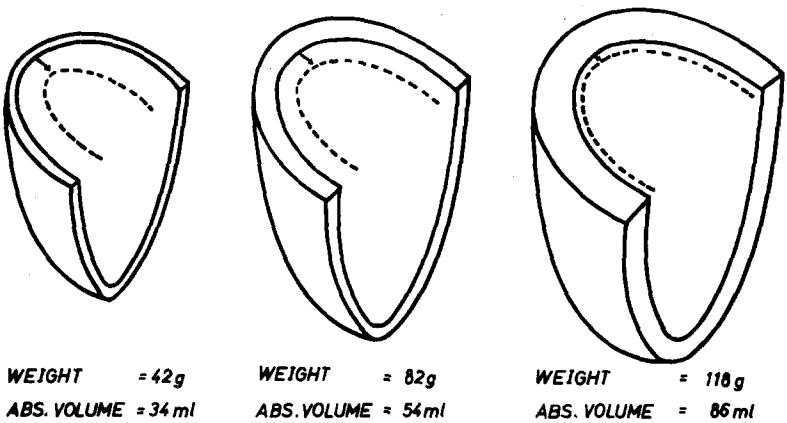

Fig. 5. Scale representation of right ventricles for different stages of hypertrophy in diastole

portion of the right ventricle had the remarkable value of 288 g, the relative volume was 21 ml/100 g.

4. The Changes in Shape of the Right Ventricle in the Course of Chronic Cor Pulmonale

Preparations of plaster moulds of the right ventricles at different stages of hypertrophy (Fig. 4) show that the inflow and the outflow tract can be distinguished clearly. The limit between inflow and outflow tract is marked by a deep impression of the anterior right papillary muscle. The inflow tract has a somewhat conical shape, but it is plumper than the outflow tract which is more like the elongated frustrum of a cone. A small indentation at the base of the right ventricle separates the inflow and outflow tracts. This indentation becomes more shallow in hypertrophy. In cases of severe chronic cor pulmonale bulging of the lateral ventricular walls originates in the vicinity of the anterior papillary muscle. Comparison of the plaster moulds of the right ventricles of severe cases of chronic cor pulmonale with moulds of normal right ventricles shows that there is no great difference in general configuration.

The form changes of our right ventricles for the weight groups up to 50 g (left); 75–99 g (middle) and 100–150 g (right) are shown to scale in Figure 5. In hypertrophy of the right ventricle the rate of increase of the muscle mass lies above the corresponding increase in absolute volume. The figure, which shows the right ventricles in diastole, also shows the decrease in internal radius during systole for a stroke volume of 60 ml.

Discussion

Kirch (1924, 1933, 1955) has shown that the structural adaptation of the right ventricle in chronic cor pulmonale begins with an elongation of the outflow

tract. In the later stages of chronic cor pulmonale the increase of muscle mass is accompanied by an additional increase of the width of the right ventricle which Kirch called eccentric hypertrophy. In these late stages the occurrence of right heart failure increases more and more. Giese (1966) differentiates four fundamental alterations in the development of chronic cor pulmonale.

1. Hypertrophy of the wall of the right ventricle.
2. Elongation of the longitudinal axis of the heart.
3. Increase in width of the right ventricle with an increase of the transverse diameter.
4. Leftward rotation of the heart with attendant cardiac displacement.

Both investigators are in agreement that in the presence of eccentric hypertrophy of the right ventricle the probability of right heart failure increases. Our results indicate that during the development of chronic cor pulmonale augmentation of the absolute ventricular volume takes place as the muscle mass increases. But it is impossible to decide whether hypertrophy is concentric or eccentric, when compared with normal ventricles, given only the values of the absolute volumes of the ventricles.

Cohnheim, as early as 1882, and Moritz and Mohr (1933) pointed out that the ratio of ventricular volume to ventricular weight is the most important criterion for the definition of the special types of heart hypertrophy. In this sense Linzbach (1967) has defined the degree of dilatation of a ventricle in rigor mortis as the relative volume, i.e., ventricular volume in ml/100 g ventricular muscle mass. In cases of concentric hypertrophy the relative volume of the ventricle is less than the norm. The absolute volume, however, may exceed the norm due to the rapid rise in ventricular weight.

In eccentric hypertrophy the values of the relative volume lie above the norm. From this it follows that in eccentric hypertrophy the augmentation of the absolute ventricular volume is greater than the increase in weight of the ventricle muscle mass.

Analysing the findings of our investigations we see that: The relative volume of the right ventricle in chronic cor pulmonale decreases. This behaviour is especially pronouced in severe chronic cor pulmonale. Stated differently; in the course of chronic cor pulmonale the right ventricle becomes more and more concentric compared to normal, and may come closely to resemble the left. This "left-ventricularisation" is more marked in adolescents than in adults, and is most impressive in two of our cases, one a 14 year old boy and another of 18 years both suffering of primary pulmonary sclerosis. The most marked cases of so-called "left ventricularisation" are, however, observed in congenital cardiac malformations.

The initial left ventricular response to an increase in pressure load in the systemic circulation is concentric pressure hypertrophy, where the relative volume in rigor mortis or the degree of dilatation is less than in the normal heart. Systolic failure of the left ventricle only develops in late stages, when concentric pressure hypertrophy changes to an eccentric hypertrophy (Gefügedilatation or structural dilatation) as a result of increasing coronary insufficiency (Linzbach, 1967).

In contrast to left heart failure, right sided failure results in chronic cor

pulmonale, although the hypertrophy is connected with a decrease of the relative volume, i.e., the degree of dilatation when compared with the normal right ventricle.

This difference in reaction of the left and right ventricles can be interpreted by considering the differences in shape and muscular mechanics of both ventricles in the normal heart.

The postnatal reversal of circulation with occlusion of the ductus arteriosus and subsequent decrease of the right systolic intraventricular pressure from 80 to 20 mmHg causes a physiological structural dilatation of the right ventricle (Linzbach, 1947; Boellaard, 1952; Hort, 1955; Kyrieleis, 1963). This dilatation is caused by a slippage of muscle fibres, causing a decrease in the number of muscle layers in the wall of the ventricle. This results in decreased mechanical efficiency of the muscle of the right ventricle. The disadvantages of this structural dilatation can be illustrated by the following:

The muscle mass of the normal right ventricle, including its septal component is about half that of the left ventricle. As the right ventricle produces only 1/5 of the pressure of the left for an equal stroke volume, the right ventricle performs, with one half of the muscle mass of the left but approximately the same tension per unit muscle cross section, only 1/5 of the left ventricle's work. Thus, due to structural dilatation the right ventricle requires about 2.5 times more muscle mass for the same work than does the normal left ventricle.

The blood supply of the myocardium of the right ventricle is better and more constant than that of the left during the cycle of cardiac activity (Schütz, 1958). Normal systolic pressure in the right ventricle is about 20 mmHg, thus the compression pressure in the wall of the right ventricle during heart action is always less than the mean capillary pressure of about 30 mmHg.

Physiological structural dilatation and a good coronary blood supply will determine the extent and the quality of the adaptative hypertrophy of the right ventricle in chronic cor pulmonale. Cardiac hypertrophy is caused by an increase in tension (t) per unit muscle cross section according to an approximation of the Laplace equation:

$$t = \frac{p \cdot r}{2w} \quad \frac{\text{dyn}}{\text{cm}^2}. \tag{3}$$

The tension (t) is proportional to the systolic pressure (p), the radius (r) and inversely proportional to the wall thickness (w).

The heart muscle, under increasing strain, tries to keep the tension/unit muscle cross section in the normal range as far as possible by the adaptative formation of muscle mass. In severe cases of systemic hypertension the blood pressure may exceed twice the normal. Pressure hypertrophy of the left ventricle may result in weights about 3 to $3^1/_2$ times the norm. In chronic cor pulmonale, however, we have observed right ventricular weights 5 to 6 times the norm (Figs. 1 and 2).

In severe cases of chronic cor pulmonale the systolic pressure in the right ventricle may reach 80 mmHg or more, exceeding normal pressure by 4 to 5 times. At these pressures if the work has to be performed with the same

tension/unit heart muscle cross section, then an enlargement of the cardiac muscle cross section to 4 or 5 times normal requires an 8 to 11 times increase in muscular weight. From this it follows that with normal weight of the free muscular portion of the right ventricle (50 g) a weight of 390 g to 550 g of muscle would be necessary.

In addition to this we have to consider that tension is not only directly proportional to the systolic pressure but also to the radius of the ventricle.

Hypertrophy of the right ventricle, already physiologically dilated in the normal individual, is accompanied by an increase in the absolute volume (Fig. 1). In other words: the systolic residual volume of the right ventricle increases considerably while values of the stroke volume in cases of chronic cor pulmonale remain the same (Fig. 5). The diminution of the ventricular width decreases in systole (Fig. 5) and the tension/unit muscle cross section is bound to increase if no corresponding formation of new muscle mass occurs.

The mass of right ventricular muscle needed for adaptation in chronic cor pulmonale is therefore dependant not only on the intraventricular systolic pressure but also on the absolute volume. Table 2 shows that the values for the absolute volume can amount to three times the norm.

The compression pressure in the wall also rises in chronic cor pulmonale because of the increased intraventricular pressure. Consequently the capillaries in the myocardium of the right ventricle no longer have a constant blood flow during heart action, since the mean intracapillary pressure may be surpassed with impaired blood supply to the right ventricle. This explains why, in chronic cor pulmonale, although the degree of dilatation decreases continually, the mechanical situation deteriorates, with the final failure of the right ventricle. In the end, the muscle mass can not eject a normal stroke volume in spite of well developed concentric hypertrophy.

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