Electrocardiograms of Deceptive Form in Ventricular Hypertrophy

BY CHARLES E. KOSSMANN, M.D.

(Dr. Kossmann: Early in your work in the clinics and on the wards you heard the word "incidence" used repeatedly. Briefly it describes who gets a disease where, when, and how often. It is customary to talk of the frequency component of incidence of a disease usually in terms of a percentage of a susceptible population, or of a death rate. The incidence of a specific phenomenon is usually quoted as a percentage of the cases that will display it. This "incidence" is valuable information to have as can easily be demonstrated. For example, when a right bundle-branch block is observed in an electrocardiogram of a patient born and bred in New York, Chagas' disease of the heart need not be given a second thought because that disease is unknown here. On the other hand if we were physicians in certain parts of Brazil, it would be seriously considered in the differential diagnosis. In each instance a knowledge of incidence of a disease and of a phenomenon in it leads to a correct diagnosis.

But incidence, especially of phenomena, also misleads the clinician. This seems to be especially true if the frequency of a particular phenomenon in a disease is fairly high—let us say 70 per cent. There is a great natural tendency to underestimate the significance of the other 30 per cent in which the phenomenon is known to be absent. Further, the occasional or frequent occurrence of it in another disease is often lost sight of completely.

In the two cases about to be presented, the phenomenon in question was electrocardiographic, and overweighting of it, on the basis of known incidence, in the over-all appraisals resulted in some confusion, at least in the beginning, regarding the exact anatomic cardiac diagnosis in each.

Mr. Lehrer will present the first case.

Clinical Clerk Lehrer: Patient A. A. was a divorced, unemployed shoemaker at the time of his death in congestive heart failure at the age of 63 years.

He had "always" had a mildly productive morning cough but the only significant past respiratory disease was influenza in 1918 when he was 29 years old. As a shoemaker he was exposed to dust while sanding leather by hand for a period of four years. He also had used sandstone for grinding carpenter's tools for some 20 years. He smoked a pack of cigarettes a day.)
At 41 he had a penile chancre which was treated for six months with intravenous and intramuscular injections in another hospital. About this time he was first admitted to Bellevue Hospital for swelling of the ankles. Treatment consisted of rest in bed and a salt free diet. The swelling subsided but recurred with physical activity. He was discharged after two weeks.

At 50 an appendectomy was performed under nitrous oxide and ether anesthesia at Bellevue Hospital. A notation was made of exertional dyspnea and edema for some time. An electrocardiogram showed normal sinus rhythm with no deviation of the electrical axis of QRS; it was not abnormal in any respect. A Wassermann test was negative.

At 58 he came to the medical clinic complaining of exertional dyspnea, edema, headache, dizziness, and weakness. Evidence of heart failure was present in the form of rales at both lung bases, an enlarged liver, and edema. Clubbing of the fingers was noted. An electrocardiogram displayed normal sinus rhythm with left axis deviation of QRS but was not otherwise beyond normal limits. X-ray showed questionable cardiac enlargement and some irregular shadows in the bases interpreted as congestion. He was treated for a few months with digitalis and diuretics without improvement. He discontinued taking these drugs in August 1947.

His complaints worsened so that he sought readmission to the hospital in December 1948. He showed little evidence of congestive failure other than edema and rales in the lower parts of the lungs which could be made to disappear with coughing. Additional findings were an inequality of the radial pulses with a blood pressure on the right of 122/70, and on the left of 90/80, minimal sclerotic changes in the retinal vessels, and an increased anteroposterior diameter of the chest. The patient was quite obese (weight 185 pounds, height 62 inches). As on previous examinations the heart sounds were distant, the second pulmonic sound was questionably louder than the second aortic sound, and the electrocardiogram displayed left deviation of the QRS axis (fig. 1). The venous pressure was 95 mm. H2O. His response to digitalis and mercurials was poor. The impression was that most of his difficulty was pul-

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**FIG. 1.** Electrocardiograms of patient A. A. recorded over a period of approximately three years. On Feb. 10, 1949, only leads I, II and III were recorded. On the other dates there were, in addition, the augmented unipolar extremity leads (aV_R, aV_L, aV_F) and the unipolar precordial leads V_1, V_2, V_3, V_4, V_5, and V_6 made with the string sensitivity at half normal (1 mv. = 0.5 cm.). To be noted particularly are the changes in T wave over a period of time in leads aV_F, V_1 and V_3. Time lines occur every 0.04 second.
ELECTROCARDIOGRAMS OF DECEPTIVE FORM

monary, that obesity contributed to his dyspnea, and that he had an anomaly of the arteries to the left arm.

From January 1949 to the time of his death on Feb. 20, 1952 he was observed in the Cardiac Clinic, and had no less than six additional admissions to the hospital. One of these was for a febrile episode treated successfully with systemic and aerosol antibiotics. The others were for progressive diminution of pulmonary and probably cardiac reserve. Digitalis therapy was reinstated on several occasions to a point of easily reached toxicity after which the patient would refuse to take the drug. The thera-

Table 1.—Hemodynamic Data on Patient A.A. Collected on March 24, 1949

<table>
<thead>
<tr>
<th>Normal Values</th>
<th>Observed</th>
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<tbody>
<tr>
<td></td>
<td>Rest</td>
</tr>
<tr>
<td>Pulmonary artery (mm. Hg)</td>
<td>18–34</td>
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<tr>
<td>Right ventricle (mm. Hg)</td>
<td>18–34</td>
</tr>
<tr>
<td>Right atrium (mm. Hg)</td>
<td>1–3</td>
</tr>
<tr>
<td>Artery (mm. Hg)</td>
<td>120 ± 20</td>
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<tr>
<td>Heart rate (per min.)</td>
<td>65–85</td>
</tr>
<tr>
<td>Ventilation volume (L./min.)</td>
<td>6–8</td>
</tr>
<tr>
<td>Oxygen consumed (ml./M3/min.)</td>
<td>110–140</td>
</tr>
<tr>
<td>Arterial oxygen (vols. %)</td>
<td>17–20</td>
</tr>
<tr>
<td>Arterial saturation (%)</td>
<td>93–96%</td>
</tr>
<tr>
<td>Mixed venous oxygen (vols. %)</td>
<td>12–15</td>
</tr>
<tr>
<td>A-V oxygen diff. (vols. %)</td>
<td>4–6</td>
</tr>
<tr>
<td>Cardiac output (L./min.)</td>
<td>5–6</td>
</tr>
<tr>
<td>Cardiac index (L./M3/min.)</td>
<td>2.7–3.4</td>
</tr>
<tr>
<td>Syst. periph. resist. (mm. Hg/L./min.)</td>
<td>14–22</td>
</tr>
</tbody>
</table>

* Mean pressures in parentheses.

peutic response was never impressive. On one admission cardiac catheterization was done (March 24, 1949) with the results shown in table 1. Briefly there was a slight elevation of pressure in the lesser circuit and a normal cardiac output. Both were increased by exercise. The oxygen saturation of the blood fell after effort.

An important complaint beginning in October 1949 was epigastric and subxiphoid pain on effort, not relieved by nitroglycerin. From that point on tachypnea, tachycardia, and cyanosis were progressive. There was a gradual loss in weight of about 40 pounds. Electrocardiograms showed an inversion of the T wave in leads from the right side of the precordium beginning in January 1951 but no change in the axis of QRS (fig. 1). Chest x-ray films did not change remarkably. The pulmonic second sound was now persistently louder than the aortic second. The venous pressure rose to 152 mm. Hg but decholin and ether circulation times were normal (15 seconds and 8 seconds, respectively, on Jan. 20, 1951). He was finally transferred to Goldwater Memorial Hospital for chronic hospital care on Oct. 10, 1951.

At the chronic disease hospital extensive pulmonar

ary functional studies were done while the patient received 206.5 mg. of adrenocorticotropic hormone between Nov. 27, 1951 and Jan. 16, 1952. Results of these are summarized in table 2. By way of summary, the adrenocorticotropic hormone at first caused euphoria and relief of the epigastric pain, along with some improvement in pulmonary function. However, he finally became irritable and unmanageable. Complications included a pneumonitis on January 22 which responded to penicillin, a herpes zoster in the distribution of the right seventh intercostal nerve on January 30, and a mycotic stomatitis. Another febrile episode with leukocytosis and dehydration occurred on February 17. He was improving slightly when he died suddenly on Feb. 20, 1952.

Between 1947 and 1952 his hemoglobin was 14.5 to 17 Gm., and the red blood cells from 4.74 to 7.0 million per cubic millimeter. The hematocrits during the last weeks of life were approximately 54. Urinalyses, blood chemistries, and serum electrolytes were not unusual except for some hyponatremia and hyperkalemia terminally.
### Table 2.—Pulmonary Function (Patient A. A.)

<table>
<thead>
<tr>
<th>Period</th>
<th>Date</th>
<th>Weight lbs</th>
<th>Vital Capacity ml (J206)*</th>
<th>Total Capacity ml (4635)*</th>
<th>Residual Air/Total Capacity % (% 41)*</th>
<th>Maximal Breathing Capacity (not less than 80)*</th>
<th>Oxygen Inhaled Air %</th>
<th>Pulmonary Ventilation L/min./M² (1.2-4.9)</th>
<th>Oxygen Consumption ml/min./M² (107-165)</th>
<th>R.Q.</th>
<th>pO₂ Alveolar Air mm. Hg</th>
<th>pO₂ Arterial Blood mm. Hg (90-110)</th>
<th>pO₂ Arterial Blood mm. Hg (90-100)</th>
<th>pCO₂ Arterial Air mm. Hg</th>
<th>Arterial Blood Oxygen Saturation %</th>
<th>Dead Space/Tidal Air % (30 or less)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control (10/11/51 to 11/26/51)</td>
<td>10/24/51</td>
<td>148</td>
<td>153</td>
<td>149</td>
<td>1785</td>
<td>3842</td>
<td>54</td>
<td>21</td>
<td>7.99</td>
<td>21</td>
<td>151</td>
<td>.75</td>
<td>154</td>
<td>90</td>
<td>46</td>
<td>48</td>
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<tr>
<td></td>
<td>11/5/51</td>
<td>151</td>
<td></td>
<td></td>
<td>103 to 123</td>
<td>21</td>
<td>7.60</td>
<td>139</td>
<td>.75</td>
<td>218</td>
<td>21</td>
<td>151</td>
<td>.75</td>
<td>215</td>
<td>160</td>
<td>100</td>
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<tr>
<td></td>
<td>11/5/51</td>
<td>151</td>
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<td>21</td>
<td>7.60</td>
<td>139</td>
<td>.75</td>
<td>218</td>
<td>21</td>
<td>151</td>
<td>.75</td>
<td>215</td>
<td>160</td>
<td>100</td>
<td>43</td>
</tr>
<tr>
<td></td>
<td>11/5/51</td>
<td>(Exercise)</td>
<td></td>
<td></td>
<td>103 to 123</td>
<td>21</td>
<td>7.60</td>
<td>139</td>
<td>.75</td>
<td>218</td>
<td>21</td>
<td>151</td>
<td>.75</td>
<td>215</td>
<td>160</td>
<td>100</td>
</tr>
<tr>
<td>Therapy with ACTH§ (11/27/51 to 1/16/52)</td>
<td>12/18/51</td>
<td>154</td>
<td>1000</td>
<td>3797</td>
<td>50</td>
<td>21</td>
<td>8.36</td>
<td>159</td>
<td>.80</td>
<td>153</td>
<td>21</td>
<td>151</td>
<td>.75</td>
<td>215</td>
<td>160</td>
<td>100</td>
</tr>
<tr>
<td></td>
<td>12/20/51</td>
<td>154</td>
<td></td>
<td></td>
<td>21</td>
<td>10.64</td>
<td>193</td>
<td>.81</td>
<td>149</td>
<td>21</td>
<td>151</td>
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<td>160</td>
<td>100</td>
<td>43</td>
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<td></td>
<td>1/10/52</td>
<td>158</td>
<td></td>
<td></td>
<td>102 to 127</td>
<td>21</td>
<td>10.64</td>
<td>193</td>
<td>.81</td>
<td>149</td>
<td>21</td>
<td>151</td>
<td>.75</td>
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<td>160</td>
<td>100</td>
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<tr>
<td></td>
<td>(Exercise)</td>
<td>1/10/52</td>
<td></td>
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<td>102 to 127</td>
<td>21</td>
<td>10.64</td>
<td>193</td>
<td>.81</td>
<td>149</td>
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<td>100</td>
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<tr>
<td></td>
<td>1/14/52</td>
<td>157</td>
<td>1744</td>
<td>3872</td>
<td>55</td>
<td>21</td>
<td>14.94</td>
<td>509</td>
<td>.89</td>
<td>148</td>
<td>21</td>
<td>151</td>
<td>.75</td>
<td>215</td>
<td>160</td>
<td>100</td>
</tr>
<tr>
<td>Control (1/17/52 to 2/15/52)</td>
<td>2/15/52</td>
<td>151</td>
<td></td>
<td></td>
<td>108 to 117</td>
<td>21</td>
<td>15.60</td>
<td>207</td>
<td>.81</td>
<td>152</td>
<td>21</td>
<td>151</td>
<td>.75</td>
<td>215</td>
<td>160</td>
<td>100</td>
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* Predicted value. † Normal range at rest. ‡ Interpolated from standard oxygen dissociation curve. § ACTH kindly provided by Armour and Co. Each daily dose dissolved in 1000 ml. of 5 per cent glucose in distilled water was administered intravenously over a period of 8 to 12 hours. The dosage schedule was as follows: Nov. 27, 28—15 mg.; Nov. 29 to Dec. 3—10 mg.; Dec. 4 to 11—5 mg.; Dec. 12, 13—2.5 mg.; Jan. 14—2 mg.; Jan. 15, 16—1 mg. From Jan. 17 to 20 an infusion without ACTH was given. ¶ Approximately 30 step-ups in one minute on a stool six inches high. # Last test performed on Jan. 18.
DR. KOSSMANN: Dr. Brumlík, you fluoroscoped this patient on several occasions. Will you comment on the radiologic findings both on fluoroscopy and on teleroentgenography?

DR. BRUMLÍK: I first fluoroscoped this man on Feb. 10, 1949. In the frontal view the heart was transverse in position, and although there was a pleuropericardial fat-pad at the apex, nevertheless the heart seemed a little enlarged in its long diameter. In the right anterior oblique view there was a retrocardiac shadow interpreted as calcified glands, and the outflow tract of the right ventricle was bulging anteriorly. The lung markings were accentuated in both fields. On x-ray this had the form of nodulations with some coalescence at the right base. There was not much change in the radiographic picture described while the patient was observed in our clinic (fig. 2).

DR. KOSSMANN: Are there any questions regarding the course of the patient's disease or the findings?

CLINICAL CLERK: Is it likely that the edema for which this patient was admitted to the hospital at 41 was similar to the edema seen in the last few years of his life?

DR. BERGER: I think not. The edema was of two types. When we first saw this man in the clinic it was the nonpitting type often seen in short, obese people, probably on the basis of chronic lymphatic obstruction with excessive subcutaneous fat around the ankles and lower legs. The second, pitting type appeared much later in the course of the disease.

CLINICAL CLERK: What was the epigastric pain on effort ascribed to?

MR. LEHRER: No statement was available in any of the records on the possible cause.

DR. KOSSMANN: I can add a little to the answer. In seeking possible causes of the epigastric pain we were able to demonstrate by further workup a hiatus hernia of the stomach, an osteoarthritis of the dorsal spine, and a gall bladder which on two occasions filled poorly or not at all with dye. None of these disease states seemed adequate, however, to explain the symptom. Coronary insufficiency as a cause was considered, but against it was the failure of the pain to respond to nitroglycerin. Further, although it was worse on effort, the pain was sometimes persistent at rest for long periods of time, particularly toward the end. No convincing explanation was ever forthcoming. It is noteworthy, however, that patients with heart disease secondary to a chronic disease of the lungs will often suffer from lower substernal or epigastric pain. It has been given a variety of names including hypercyanotic angina and pulmonary hypertensive pain. It has been ascribed to dilatation of the pulmonary artery, to coronary insufficiency largely on the basis of arterial oxygen unsaturation, to congestion of the liver, to an unusually low level of the diaphragm with tension on the central and other ligaments, to peptic ulcer, and to other causes, most of them difficult to document or indict. The symptom, nevertheless, is real, and most often simulates anginal syndrome except for its long duration.

DR. EICHNA: Would you say that the hemodynamic data collected by you about three years before the patient’s death make it possible to say, without reference to any other data, that we were dealing with latent pulmonary arterial hypertension from intrinsic pulmonary disease?

DR. EICHNA: Probably yes. The data (table 1) indicate slightly higher than normal systolic and diastolic pressures in the pulmonary artery and in the right ventricle when the patient was...
at rest. These pressures, except the diastolic pressure in the right ventricle, were further increased by a degree of exercise which raised the cardiac output approximately two and one-half fold. Other figures indicate that the resistance of the pulmonary vascular bed was increased, but whether by intrinsic vascular disease or by the inability of the left ventricle to increase its output under stress cannot be stated definitely. However, the normal cardiac output at rest and its satisfactory increase with exercise suggest that myocardial failure was not responsible for the elevated pulmonary artery pressures. On the other hand, the somewhat elevated pulmonary artery pressure at rest and its sharp rise with an increase in blood flow (cardiac output), a rise which would not occur in the normal subject, certainly suggests primarily increased pulmonary vascular resistance as the cause. Whether this increased resistance was neurogenic in origin or due to organic pulmonary vascular disease depends on the ability to demonstrate a primary pulmonary disease which would affect the pulmonary vascular bed.

DR. KOSSMANN: Dr. Galdston was kind enough to come this evening to tell us about the pulmonary functional studies he did on this patient in the last months of his life at Goldwater Memorial Hospital.

DR. GALDSTON: Before the administration of adrenocorticotropin hormone the patient exhibited a borderline normal total lung volume (table 2). The vital capacity was reduced. There was an abnormally large absolute and relative volume of residual air. These are common findings with pulmonary emphysema. The excellent maximal breathing capacity indicated that bronchiolar obstruction did not contribute to the emphysema.

The minute volume of respiration was greatly increased even when the patient was afebrile and when his resting rate of oxygen consumption was within the predicted range. These data, together with a normal gas exchange ratio (R.Q.) indicate that the hyperventilation was the result of physiologic and metabolic drives. The volume of each breath which did not partake in gas exchange, the dead space, was abnormally large. One might expect an alveolar oxygen tension of less than 90 mm. Hg and a greater degree of carbon dioxide tension in the alveolar air than 48 mm. Hg with such maldistribution of gas in the lungs. The increased minute volume of ventilation most likely accounted for the minimal derangements in the tension of these gases. The results of the simultaneously measured oxygen tensions of alveolar air and of arterial blood when breathing room air (21 per cent oxygen) and again when breathing 30 per cent oxygen indicate that there were many poorly ventilated alveoli with capillaries well circulated with mixed venous blood. Exchange of oxygen was poor in these areas which may be considered to be veno-arterial oxygen shunts. The data also suggest, though they do not permit definitive quantitation, that impediments to oxygen diffusion, such as thickened alveolar-capillary membranes or absolute reduction in the effective gas diffusing surface, contributed to the oxygen unsaturation of the arterial blood. The further fall in arterial blood oxygen saturation and rise in pCO₂ of the alveolar air during moderate exercise are commonly seen in chronic pulmonary disease complicated by pulmonary hypertension. In short the patient exhibited many derangements in pulmonary function compatible with advanced emphysema and fibrosis.

During the period of therapy with adrenocorticotropin hormone some noteworthy improvements in pulmonary function were observed. The distribution of air to the alveoli improved as did the transfer of gas across the alveolar capillary membrane. These were manifested by a decrease in the ratio of dead space to tidal air, a rise in pO₂ and a fall in pCO₂ of the alveolar air, and an increase in resting arterial blood oxygen saturation.

The complications which occurred after discontinuance of adrenocorticotropin hormone permitted only a limited number of studies. These indicated, in general, a regression of pulmonary function to pretreatment levels.

CLINICAL CLERK: Can it be stated when this patient’s heart disease began?

DR. BERGER: That question is difficult to answer because the stages of chronic cor pulmonale, which he was suspected of having, run
imperceptibly one into the other. Arbitrarily there is the precardiac phase, when pulmonary symptoms dominate; the cardiac phase without failure, when right ventricular hypertrophy is recognizable; and the cardiac phase with failure, which is the time when evidence of congestion behind the right ventricle begins to appear.

It was difficult to say just when this patient began to have failure of the right ventricle, and indeed there was, at times, a question whether there was any at all. The old lymphedema, present for 20 years, did not make the task easier. Obesity made palpation of the liver uncertain. There was X-ray evidence of enlargement of the right ventricle in 1949 when he was 60 years old. It was soon thereafter that symptoms were intensified. Sometime in 1950, the electrocardiogram began to show inversion of T waves in leads from the right side of the precordium. Actually distinct pitting edema and a palpable liver were regularly observed only in the last year of life.

Weighing all the facts it is probable that the clinical picture was dominated most of the time by the pulmonary dysfunction, and failure of the right ventricle was probably present for not much more than two years before death.

Dr. Kossmann: The electrocardiograms, especially during the first three years of observation, were disturbing because they displayed left deviation of the electrical axis of QRS with an angle alpha of approximately +6 degrees with a normal ventricular gradient. Although we knew the patient had pulmonary disease, a normal blood pressure, a loud pulmonic second sound, latent hypertension in the pulmonary artery, normal cardiac output, polycythemia, clubbing, and minimal enlargement of the heart, it still was not certain, because of the persistent rales in the lungs, whether the evidences of congestion seen behind the right ventricle were from right ventricular disease, per se, or from combined left ventricular and right ventricular disease. Under these circumstances the electrocardiogram was not helpful until certain changes became manifest in the last year of life. Would you discuss the electrocardiograms for us, Dr. Rader?

Dr. Rader: Five of the records made between Dec. 4, 1948 and Oct. 4, 1951 are shown in the chart (fig. 1). Others were available, and when precordial leads V3, V4, and V6 were made, each usually resembled the precordial lead just to the right of it. A record with six chest leads made on Dec. 19, 1951 resembled the last one in the chart (Oct. 4, 1951). In it the T wave was inverted in the first three precordial leads, upright in the others.

A review of the records indicates that they were relatively constant between Dec. 4, 1948 and March 11, 1950. Beginning in 1951 the essential changes consisted of an increase in size of the T waves, and a change in direction of the spatial vector of T of such nature as to cause deepening of the T wave in lead III, and inversion in lead V1. There also was some shift of the transitional zone of QRS to the left. In the record of Oct. 4, 1951, and in subsequent records (not shown) these trends were more marked, with the T wave inverted as far to the left as lead V4, and further shift of the transitional zone of QRS to the left so that the R/S ratio was inverted in lead V6. The T wave became inverted in lead aVf. In terms of the spatial vector of the T deflection, it had rotated backward in a counterclockwise direction viewed from above, and upward.

There were, then, some modifications, beginning in January 1951, to suggest a considerable lengthening of the duration of the excited state on the right side and front of the heart such as is often seen more acutely when the pressure is raised in the right ventricle as in acute cor pulmonale. The shift of the transitional zone of QRS probably was the result of gradual dilatation of the right ventricle. To be noted is that there was no important change in the time of the intrinsicoid deflections in leads V1 and V2.

On February 15, five days before death, the electrocardiogram underwent a striking change: It showed, for the only time, a right deviation of the axis of QRS. The QRS interval was not widened, and the deflections in lead V1 had the low W-shaped appearance often seen with right ventricular hypertrophy. It was suspected that the patient had a pulmonary embolus that day. In addition, the serum potassium was 8.3 mEq. per liter. On the next two days the record resumed its original appearance and axis of QRS,
although the voltage of the QRS deflections was low.

In retrospect perhaps too much attention was paid to the direction of the QRS deflections, and perhaps not enough to the T deflections, especially the gradual inversion over a period of time in leads from the right side of the precordium.

**DR. KOSSMANN:** Mr. Lehrer, what was the final diagnosis we made?

**MR. LEHRER:** The diagnoses made were: (1) Cardiac, (a) pulmonary arterial hypertension and arteriosclerosis, (b) enlarged heart, arteriosclerosis of aorta, (c) normal sinus rhythm, (d) IVE; (2) Pulmonary fibrosis and emphysema; (3) Hiatus hernia; (4) Anomaly of arteries to left arm; (5) Syphilis, late latent, Wassermann negative; (6) Possible chronic cholecystitis.

**DR. KOSSMANN:** If there are no other points let us go on to the pathologic findings.

**PATHOLOGIST:** The necropsy was performed 13 hours after death at Goldwater Memorial Hospital by Doctors Rosenkrantz and Vrbano-ovic. The significant findings were as follows:

The heart: weight 450 Gm., moderate hypertrophy of the right ventricle, none of the left (width of left ventricular wall 1.5 cm.); some sclerosis of the coronary vessels with considerable narrowing and calcification of the anterior descending branch of the left coronary; slight atheromatosis of the aorta.

The lungs: weight, right 1100 Gm., left 1000 Gm.; obliteration of the right pleural cavity; numerous small emphysematous blebs over the surface of the right lung, with larger blebs and bullae in the left upper lobe. The bronchi and bronchioles were dilated, and showed evidence of inflammation and proliferation. Sections of the lungs were firm, showed gross streaks of fibrosis, confirmed by microscopic examination. The pulmonary arteries showed moderate to advanced arteriosclerosis, and one large one displayed a recent thrombus with early organization.

The only other significant findings were congestion particularly of the liver (weight 1600 Gm.) which had a characteristic “nutmeg” appearance, and of the spleen (weight 250 Gm.).

**PHYSICIAN:** Was there any disease of the extrahepatic biliary tract or of the gastrointestinal tract?

**PATHOLOGIST:** Both were free of disease.

**DR. KOSSMANN:** It is evident from the post-mortem findings that this patient had heavy, fibrotic lungs, predominantly right ventricular disease, and arteriosclerosis of the pulmonary arteries and arterioles. The latter finding is anatomic evidence of prolonged hypertension in that circuit. The stenotic coronary artery described may have been responsible for the subxiphoid pain. Before discussing the electrocardiograms in relation to the morbid anatomy, I believe it would facilitate matters to consider the contrasting case. Mr. Nash, will you proceed with the story of the second man’s life?

**CLINICAL CLERK NASH:** F. A., a 52 year old male Puerto Rican elevator operator, was admitted to Bellevue Hospital on Oct. 25, 1951, because of increasing cough, dyspnea, orthopnea, fatigue, anorexia, and insomnia of three days duration.

The patient’s symptoms began in 1950 when he was at another hospital for nonproductive cough, dyspnea, orthopnea and fatigue. He was told he had hypertension, was digitalized, and placed on a low salt diet. He remained on digitalis from that point on.

He had tonsillitis in 1934 and subsequently a tonsillectomy was performed. He had pleurisy in 1939 but denied pneumonia or respiratory sequelae. He never worked in an occupation where dust was a problem. An appendectomy was performed in April 1951. The patient was born in Puerto Rico, moved to the U. S. in 1920, and returned to Puerto Rico for a month in 1931; there was no other travel.

The family history was noncontributory.

Examination on admission on Oct. 25, 1951, revealed a poorly developed, dyspneic male, 64 inches tall, weighing 105 pounds, and appearing older than his stated age. Positive findings included: A diffuse maculopapular rash on the trunk; questionable hyper-resonance of the lungs; medium moist inspiratory rales posteriorly on both sides below T10. The point of maximal impulse of the heart was felt in the sixth intercostal space at the anterior axillary line. There were no thrills or murmurs. The pulmonary second sound was louder than the aortic second. There were a few premature systoles. An apical gallop rhythm was present on admission, but disappeared later. The blood pressure was 190/118 mm. Hg. There were no signs of congestion of the abdominal viscera although the cervical veins were distended. There was retinal arteriolosclerosis.

There was 2 plus or more proteinuria on repeated urinalyses, although the specific gravity varied from 1.004 to 1.022 on a concentration and dilution test. The blood count was normal as was a Mazzini test.
The blood non-protein nitrogen was 45 mg. per 100 ml. The venous pressure was 112 mm. H₂O; the enlargement was principally to the left with enlargement of the inflow and outflow tracts of the left ventricle. The left border was straight, although the pulmonary artery was not enlarged. With barium swallow there was some displacement of the esophagus posteriorly by an enlarged left atrium (fig. 3).

X-rays (fig. 3) displayed a vertical heart with an increase in the transverse diameter. On fluoroscopy circulation times were ether 15 seconds, decholin 28 sec.

Fig. 3. Patient F. A. Teleoentgenograms with right anterior oblique (lower left) and left anterior oblique (lower right) views made on dates indicated. In the x-ray of Jan. 11, 1952, a patch of consolidation can be seen near the right costophrenic sulcus.
Pulmonary function studies are summarized in table 3. The significant changes were in the vital, total, and maximum breathing capacities which were reduced. The absolute residual volume was normal but the relative residual volume increased. The

<table>
<thead>
<tr>
<th>TABLE 3.—Studies of Pulmonary Function of Patient F.A. Made on Nov. 3, 1951</th>
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<tbody>
<tr>
<td>Observed Value</td>
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<tr>
<td>----------------</td>
</tr>
<tr>
<td><strong>A. Lung Volumes (Supine Position in cc., BTPS)</strong></td>
</tr>
<tr>
<td>Respiratory rate</td>
</tr>
<tr>
<td>Tidal volume</td>
</tr>
<tr>
<td>Inspiratory reserve</td>
</tr>
<tr>
<td>Inspiratory capacity</td>
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<tr>
<td>Expiratory reserve</td>
</tr>
<tr>
<td>Vital capacity (VC)</td>
</tr>
<tr>
<td>Functional residual capacity (Exp. Res. + RV)</td>
</tr>
<tr>
<td>Residual volume (RV)</td>
</tr>
<tr>
<td>Total capacity (VC + RV)</td>
</tr>
<tr>
<td>Index of pulmonary mixing</td>
</tr>
<tr>
<td><strong>B. Maximal Breathing Capacity</strong></td>
</tr>
<tr>
<td>(MBC)—Standing Breathing Reserve (BR)</td>
</tr>
<tr>
<td>Maximal breathing capacity (MBC)</td>
</tr>
<tr>
<td>Minute volume (MV)</td>
</tr>
<tr>
<td>Breathing reserve (BR)</td>
</tr>
<tr>
<td>(MBC-MV)</td>
</tr>
<tr>
<td>BR</td>
</tr>
<tr>
<td>MBC × 100 = Dyspneic index</td>
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</tbody>
</table>

The minute volume was also increased. The arterial pO₂ was 91.5 mm. Hg, pCO₂ 38.65 mm. Hg, and oxygen saturation 96 per cent. The findings were interpreted as being compatible with left ventricular failure without intrinsic pulmonary disease.

The electrocardiograms, with one exception, displayed a normal sinus rhythm or sinus tachycardia with right axis deviation and low voltage of QRS (fig. 4).

Therapy consisted of digitalis 0.1 and 0.2 Gm. on alternate days, a salt free diet, and bed rest. He improved gradually and became less dyspneic. However, medium rates persisted at both bases. After 20 days he was discharged to the Adult Cardiac Clinic with the diagnosis: Cardiac (a) hypertension, (b) enlarged heart, (c) normal sinus rhythm, (d) class IIIC. Accompanying condition: pulmonary fibrosis and emphysema.

He was seen in the clinic three times in as many weeks, each time complaining of cough, dyspnea, orthopnea and insomnia. The physical findings were as previously with the additions of 3 plus ankle edema, and a liver edge palpable 2.0 cm. below the costal margin. The apical gallop reappeared. Digitalis was increased to 0.2 Gm. once daily, but he became toxic after five days. He stopped the drug for three days and resumed the day before his last clinic visit on Dec. 20, 1951. In view of the problem of digitalis toxicity and continued heart failure, the patient was readmitted to the hospital that night.

In the hospital the second time the course was irregularly febrile and downhill with increasing and intractable edema. There was a moderate leukocytosis with a persistent shift of the index to the left. The blood nonprotein nitrogen gradually rose from 59 to 100 mg. per 100 ml. Dullness was noted over both lower lobes. A few days before death a thick, tenacious, bloody material was found in the mouth, but there was no frank hemoptysis. The clinical impression was bronchopneumonia. A culture of the sputum grew out gram positive diplococci.

Despite antibiotics, oxygen, mercurial diuretics, and the usual supportive therapy, tachypnea was progressive, the blood pressure fell to 110/94 on January 21, and the patient died the following day in heart failure.

Dr. Kossmann: The problem presented by the second case is a little different from the first. He was about the same height as the first patient but weighed only 100 pounds. He displayed in life definite left ventricular disease and heart failure, and peripheral sclerosis, but also showed questionable hyper-resonance of the lungs, a cough, a pulmonary second sound louder than the aortic, pulmonary functional tests revealing moderate dysfunction, and an electrocardiogram showing with one exception (fig. 4) right deviation of the electrical axis of QRS. Dr. Rader, would you discuss the electrocardiographic data further?

Dr. Rader: Between Oct. 26, 1951 and Jan. 22, 1952, 10 electrocardiograms were recorded. The patient was receiving digitalis the entire
time. Four of these are shown in figure 4. All but the second record (fig. 4, first line, Oct. 31, 1951) showed distinct right deviation of the electrical axis of QRS (angle alpha approximately +120 degrees) and left deviation of the electrical axis of the T wave such as is often seen in patients with advanced hypertrophy of the right ventricle. Further, there were high, pointed P waves in leads II and III, and a prominent diphasic P wave in lead V1.

The precordial leads show prominent S waves far to the left except in the first record, but no late intrinsicoid deflections in lead V1. Interesting, too, is the absence of a late R wave either in lead aVF or lead aVR, although a notch on the descending limb of the R wave of lead aVF in the records of Dec. 21, 1951 and Jan. 11, 1952 is fairly late (between 0.05 and 0.06 second from the beginning of QRS in that lead). Lead V6 (not shown) usually looked like V5 in the upper record (Oct. 31, 1951) with an inverted T wave. The R wave in it was early (0.03 second).

DR. KOSSMANN: The course of this patient's heart disease was less than two years from the time he first developed manifestations of diminished cardiac reserve until death. In that period, nine months before his death, he was able to go through an appendectomy. With the exception of the terminal state, the blood pressure remained elevated with the diastolic never below

![Fig. 4. Patient F. A. Electrocardiograms made during the last three months of life on dates indicated. The symbols have the usual meaning; the precordial leads were recorded with the sensitivity reduced to one half (1 mv. = 0.5 cm.). The last record (Jan. 22, 1952) was made on the day of death, and discloses widespread defects of conductivity. In the record of Dec. 21, 1951, the P wave in lead III is higher than in leads I and II because there was some shifting of the pacemaker.](http://circ.ahajournals.org/DownloadedFrom/fig4.png)
110 mm. Hg. The data on the mildly impaired pulmonary function gave no exact indication of the possible anatomical cause. What was the final diagnosis we made in the clinic?

**MR. NASH:** (1) Cardiac: (a) hypertension, arteriosclerosis, and unknown, (b) enlarged heart, coronary sclerosis, myocardial fibrosis, (c) normal sinus rhythm, gallop rhythm, congestive heart failure, (d) class IVE; (2) pulmonary fibrosis and emphysema.

**DR. KOSSMANN:** It is obvious that something further occurred in the lungs just before death. If there are no questions on the clinical data we will go on to the pathologic findings.

**Pathologist:** The necropsy was performed on Jan. 22, 1952, by Drs. David Schwartz and Norman Cooper. The examination of the brain was done by Dr. L. D. Stevenson. The essential findings were as follows:

The heart: The heart weighed 525 Gm. All chambers were markedly dilated. The left atrial appendage contained a small thrombus. There were several small thrombi, the largest 1.0 by 0.5 by 0.2 cm., between the columnae carneae of the apical region of the right ventricle. The left ventricle contained a larger number of thrombi, similarly distributed. The largest of these, 3 by 2 by 3 cm., was in the posterior apical area of the left ventricle. Small areas of gray-yellowish discoloration of the myocardium were found under this thrombus. There were no areas of myocardial softening or hemorrhage, and the myocardium in general was red-brown. The major coronary arteries were patent and had a moderate amount of atherosclerosis. There were foci of marked constriction in the left circumflex artery. The foramen ovale and ductus arteriosus were obliterated. The left ventricular wall was 1.9 cm. thick, the right 2 mm. The valve ring circumferences were: tricuspid 13 cm.; pulmonic 8 cm.; mitral 10 cm.; aortic 7 cm.

Great vessels: There was a relatively small amount of atherosclerosis through the course of the aorta. There was a moderate reduction of its elasticity. The inferior vena cava, portal vein and iliac veins appeared normal.

Lungs: The left lung weighed 300 Gm., the right 250 Gm. There was no fluid in the pleural cavities. The pleural surfaces were smooth and shiny. There were small bilateral apical pleural scars. A major part of the right lower lobe was hard and dark blue. Its cut surface was dark red, shiny, and roughly wedge-shaped. It was moderately well demarcated from adjacent tissue; small interspersed uninvolved areas made it evident that the lesion was multiple. There was a 2 by 2 cm. similar area in the left lower lobe, and small areas in the right upper lobe and left lower lobe; some of these were paler gray-red. The major artery to the right lower lobe contained a thrombus which did not conform to its shape. Small arteries in all lobes contained thrombi and propagated thrombi. The bronchi were normal. Uninfarcted pulmonary parenchyma was congested posteriorly, normal anteriorly.

Microscopic sections revealed moderate hypertrophy of the myocardial cells, but, in addition, an inordinately large amount of interstitial fibrosis. Most myocardial fibers were surrounded by thin laminae of reticulum or denser collagen. There were some areas of replacement by scar tissue. The thrombus in the left ventricle was undergoing organization but not the one in the left atrium.

Sections of the lungs confirmed the gross impression of infarcts.

**Other significant findings included:** Chronic passive congestion of the liver and pancreas; thrombosis of the periprostatic veins; arteriolar hyalinization and thickening in the spleen, pancreas, adrenals, kidneys, and brain.

In summary, the heart displayed evidence of dilatation, and predominantly of left ventricular hypertrophy. Although there was coronary stenosis, it did not cause myocardial infarction, and the degree of interstitial fibrosis and the intracardiac thrombi could not be accounted for on the basis of the coronary disease alone. There were stigmata of systemic hypertension and of prolonged cardiac insufficiency.

**DR. KOSSMANN:** Any questions?

**Clinical Clerk:** Is there no explanation at all for the myocardial fibrosis and mural thrombi?

**DR. BERGER:** Apparently the pathologist was impressed with the paucity of coronary disease to account for these findings. If this patient did not have hypertension during life the patho-
logic findings would be reminiscent of several patients we have seen recently at necropsy. These patients showed hypertrophy, mural thrombi, variable amounts of myocardial fibrosis, and little or no coronary disease. In some instances these findings were probably the end result of repeated bouts of failure on the basis of thiamin deficiency such as seen in chronic alcoholism. However, in some they occurred in the absence of vitamin deficiency. Clinically the dominant picture is one of heart failure, and the patients usually are classified as having "unknown" heart disease. It seems that with refinements in clinical technics we are making this etiologic diagnosis of "unknown" more often. This fact points up the considerable limitation in our knowledge at present about the real causes of impaired contractility of the myocardial cell.

This patient was unusual in that he had hypertension during life sufficient to cause hypertrophy of his heart, but in addition showed an unaccountably large amount of myocardial fibrosis.

DR. KOSSMANN: We are now ready to attempt an over-all summary and an explanation of the atypical electrocardiograms in the light of the anatomic alterations found at necropsy. These two men happened to be approximately the same height but one was fat and one was lean. Each presented himself with signs and symptoms referable to dysfunction either of the heart or of the lungs. Clinically, at least, symptoms from the latter organ dominated in the fat man; symptoms from the former dominated in the thin man. The pulmonary functional studies bore out the clinical impression, and hemodynamic studies in the first patient gave a very good suspicion of primary pulmonary disease as the cause for pulmonary arterial hypertension.

The laboratory data which did not fit with the others were the electrocardiograms. The man with proven right ventricular hypertrophy displayed deviation of the electrical axis of QRS to the left; the man with proven left ventricular hypertrophy displayed deviation of the electrical axis of QRS to the right.

The electrophysiologic reasons are clear enough from an inspection of the records (figs. 1 and 4). In the fat man the potential of the left arm (aV_L) was positive with respect to the potential of the left leg (aV_F). Originally, Einthoven made connections between the body and the galvanometer in such a way that with a potential of these two extremities as indicated, a record of the difference between them (lead III) would display a downward deflection. In lead III there was a deep S wave (fig. 1) indicating a mean direction of excitation of the heart with respect to that lead from below upward. In so far as excitation was from right to left in lead I, as can be seen from an inspection of this lead and the potential of its component extremities, the mean electrical axis was "deviated to the left." In the thin man the situation was reversed with the potential of the left leg and of the right arm (with one exception) more positive than of the left arm. This electrical situation resulted in "right deviation" of the electrical axis in the frontal plane.

It is worth noting that the QRS deflections in the precordial leads (figs. 1 and 4) of the two patients were not too dissimilar. This would seem to indicate that the important differences in ventricular excitation between the two were in the frontal plane, but not in the sagittal plane. One might go further, on the basis of known data, and strongly suspect that the differences noted in the two records depended not so much on the relative size of the right and left ventricles in each case but rather on the different relationships each of these chambers bore to the left arm and left leg in the two men. This is equivalent to saying that hypertrophy had little to do with the mean direction of ventricular depolarization except in so far as it determined the position of the heart in two patients with widely different thoracic configurations. Further, the combinations—short, round thorax with right ventricular hypertrophy; long, flat thorax with left ventricular hypertrophy—were of such nature that in each instance position of the chambers with respect to the extremities used for leading in the frontal plane far outweighed the effect of any preponderance caused by hypertrophy of one or the other ventricle.

It was pointed out that the T waves gradually underwent inversion in leads from the
right side of the precordium in the patient with chronic cor pulmonale. This alteration, if it occurs with any great frequency in this disease, is probably of considerable diagnostic value, and certainly was not properly evaluated in this instance. Of interest, too, is the fact that the transitional zone of QRS was shifted to the left in both patients.

This brings us back to the matter of “incidence” mentioned at the beginning of the conference. Probably less—and our own observations indicate considerably less—than 70 per cent of patients with right or left ventricular hypertrophy will show the electrocardiographic configuration “characteristic” for the chamber involved. Correlations in general will be better the greater the hypertrophy. Under the circumstances it is almost incorrect to speak of the configuration as “characteristic.” Certainly to do so leads to error; and error is especially common if the electrocardiogram has a contour usually associated with hypertrophy of the contralateral ventricle, as was true in these two patients.

In conclusion, I would say that, in addition to others, the points learned here were: (1) A knowledge of incidence of a phenomenon in a disease, though helpful, must be properly evaluated before being used in an absolute way; (2) it is unsound practice to make final anatomic cardiac diagnoses from the form of the electrocardiogram without recourse to other clinical and laboratory data.

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CHARLES E. KOSSMANN

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