P-Pulmonale in the Precordial Leads and
A Review of the Mutability of the
P-Waves in Chronic Cor Pulmonale

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Introduction

While peaked and tall P-Waves in leads two and three are often seen with cor pulmonale, one encounters only occasionally such a formation in lead AVF and rarely in the precordial leads over the right side of the heart. The present study reports the occurrence of a peaked P-Wave of high voltage in leads V-2 and V-3 obtained from two patients with severe chronic cor pulmonale. In addition to that observation, this investigation is devoted to the multiplicity of changes in contour, voltage and direction of the P-Wave in cor pulmonale during protracted electrocardiographic follow-up. An evaluation of such serial changes can be misleading without simultaneous clinical appraisal of the individual. In some patients the peaked and tall P-Wave may lose its characteristics, it may become inverted, then peaking and high voltage may reappear, and the same patient may on and off yield electrocardiograms without the typical pattern of cor pulmonale. Thus temporarily there will be seen merely right axis deviation but not any more the previously diphasic T-2 nor inverted T-3, and the tapering as well as the high amplitude of P-2 and P-3 will have receded. This transient normalcy of the electrocardiogram must not be used as a criterion for the patient’s cardiac normalcy. That consideration is especially important when major surgery is contemplated.

One must bear in mind that peaked P-Waves occur without cor pulmonale due to various factors, such as tachycardia, postural cardiac changes, hyperthyroidism and congenital heart disease with atrial enlargement. It must also be remembered that with right atrial enlargement as seen in cor pulmonale (secondary to lung conditions) the P-Wave will tend to have a pointed summit and high voltage while with left atrial enlargement the P-Wave is usually broad, of low voltage and notched. However, the configuration of the abnormal P-Wave is not always diagnostically conclusive. A tall peaked P-Wave can be encountered with mitral stenosis due to rheumatic heart disease.1 Lowering of blood potassium may increase the height of P-2 and P-3.2

Of special interest are the cases of chronic cor pulmonale without showing at any time a tall or peaked P-Wave. One may see on the electrocardiogram marked right axis deviation, a miniature P-1, extremely shallow T-2 and T-3, small Q-Waves in L-2 and L-3, and P-2 and P-3 will be

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conspicuous due to their normal voltage in contrast to the extremely low voltage of the T-Wave in the same lead. Not infrequently I have seen this phenomenon supported by autopsy findings.

This paper is confined to three case reports: 1. Chronic cor pulmonale secondary to pulmonary artery thrombosis; 2. Chronic cor pulmonale in a patient with pulmonary tuberculosis; 3. Chronic cor pulmonale secondary to asthma and irreversible pulmonary emphysema.

Two patients had episodes of arrhythmia. Inasmuch as they were on digitalis therapy, a drug effect was suspected.

Case 1: A 36 year old white man was admitted on January 23, 1952 to the Veterans Administration Hospital, Bronx, New York, with the chief complaint of shortness of breath and the need of three pillows at night during the past half year. He was in good health until one year prior to hospitalization when, after walking 10 blocks, he suddenly experienced difficulties in breathing and fainted. There was loss of consciousness for five or perhaps 10 minutes. On resuming the walk he noted marked shortness of breath. There was no chest pain or evidence of convulsions. Following evidence of the long or rapid walking would be associated with a sensation of lack of air and a bluish discoloration of the face. Six months before admission he became aware of dyspnea on even slight exertion. Cyanosis had become constant by now, remained even without exertion but increased during physical effort. There was no history of cough, hemoptysis, fever, ankle edema, or nocturia. Occasionally there appeared palpitation with the dyspnea. Several months prior to admission a roentgenogram at the local Health Center was reported as showing some pulmonary infiltrate.

Physical Examination: Height 180 cm. Weight: 146 lbs. Blood pressure 104/74. Cyanosis of the lips and nailbeds. Marked cyanosis of the entire face while in the supine position. Distended neck veins which display pulsations and filling from below in the supine position. Orthopnea. Chest of good expansion, lungs normal on percussion and auscultation. Examination of the heart revealed a point of maximum impulse in the sixth ICS, just outside the midclavicular line. The heart sounds were of good quality and P-2 was greater than A-2. There was a harsh long medium pitched systolic murmur heard best in the fourth ICS to the left of the sternum and transmitted towards the apex and pulmonic area. There was a questionable thrill when in the knee elbow position. There was no diastolic murmur and there was NSR with a rate of 92. There was no systolic retraction of the precordium or paradoxical pulse. No gallop or friction rub. The abdomen was slightly protuberant and held tensely but there was no fluid wave or shifting dullness. The abdomen was difficult to palpate and the liver edge could not definitely be felt but there was the impression of a mass and dullness three finger breadths below the RCM. There was no other mass or tenderness to palpation. There was slight clubbing and cyanosis of all fingers and toes.

Laboratory and X-ray findings: Hgb. 14.1 gm. RBC 4,150,000. WBC 9,200 with 64 polyns, 1 L, 3 M, and 2 E. Hematocrit 39 per cent. Sedimentation rate 12. Urinalysis normal. Serum chlorides 100. CO, 50 volume per cent. BUN 19. Sodium 133. Potassium 4.3. Serology negative. Six blood cultures negative. Serum bilirubin 0.8. Thymol turbidity 3.6. Van Den Bergh direct 0.4, indirect 0.4. Cephalin Flocculation 1 plus. Alkaline phosphatase 13.3. Total proteins 6.3 with albumin 4.1, globulin 2.2. One culture of gastric washings for acid fast bacilli on February 5, 1952 was positive. An EKG on admission revealed changes compatible with right ventricular hypertrophy and strain and atrial hypertrophy. X-ray film of the chest revealed a fibronodular and somewhat soft infiltrate in the upper third of the right lung field and some scattered infiltrate in the apex of the left lung field. Cardiovascular shadow showed some enlargement in the transverse diameter and some prominence of the hilar vascular shadow. Angiocardiograms showed marked dilatation of the left main pulmonary artery suggestive of pulmonary hypertension. The pulmonary vasculature in the right lung field appeared relatively normal. In the lower two thirds of the left lung field there was a marked diminution of the pulmonary vasculature with absence of visualization of the main stem pulmonary arteries to this area. Cardiac catheterization revealed marked pulmonary hypertension.

Following clinical improvement the patient left the hospital against medical advice on May 7, 1952. Admitted to Veterans Administration Hospital, Castle Point, New York on September 19, 1952. On admission he appeared acutely ill with extreme dyspnea, orthopnea, edema of right arm and ankles and cyanosis. There was pulsation of neck vein, filling from below. Heart rate 120. Ascites. Liver five finger breadths below costal margin. The roentgenogram showed cardiac enlargement-in all diameters. On oblique views the right ventricular bulge was markedly accentuated. There was no
evidence of left atrial enlargement on esophagrams.

Venous pressure was 200 H2O on March 18, 1953. Circulation time, performed with decholin, was 24 sec.

He expired on May 16, 1953.

**Extract from Necropsy Findings on Full Autopsy**

1. Primary pulmonary atherosclerosis and thrombosis, bilateral, involving small, medium and large vessels.
2. Cor pulmonale. Heart weighed 550 gms. The wall of the right atrium was hypertrophied and 3 to 5 mm. thick.
3. Cardiac cirrhosis of the liver.
4. Chronic pulmonary tuberculosis.
5. Pulmonary emphysema, left upper, left lower, and right upper lobes.
6. Emphysematous cysts, right upper lobe.

**ECG changes.** January 24, 1952. No medication given. Regular sinus rhythm, rate 99. Right axis deviation. PR 0.14. QRS 0.09. QT 0.32. P-1 0.15 mv. P-2 peaked, with ascending limb 0.3 mv. and descending limb 4.0 mv. P in AVL inverted. P in AVF peaked, 0.3 mv. T-1 upright. Tendency to diphasic T-2. T-3 inverted, also T in AVF. Shallow T in AVR and less deep than P wave in same lead. T inverted in V-1 and upright from V-2 to V-6. ST depressed in L-2, V-2, V-3 and V-4. Small Q in L-3 and AVF. Prominent R in AVR and V-1. In V-4 to V-6 the depth of the S-waves causes a reversal of the RS ratio. In V-2 the P-wave is peaked and of 0.3 mv.

**Interpretation:** Cor pulmonale. April 16, 1953. P-2 0.2 mv. P in V-2 rose to 0.4 mv.; and P in V-3 is of 0.25 mv. April 16, 1953. As in previous ECGs, the inverted QRS in L-1, the tall R in L-3 and AVF indicates R.A.D. Tall upright T in V-4 and CF-4 points to an added left electrical axis deviation factor. No reversal of the RS ratio as seen January 24, 1952. Rate 90. PR 0.16. QRS 0.10. T-1 upright. T-2 diphasic and miniature, T-3 inverted. T inverted in V-2. P-2 of 0.2 mv. P is peaked in V-3 and of 0.4 mv. P became upright in CF-4. ST depressed in L-2, V-3, V-4 and CF-4. In V-3, V-4 and CF-4 ST merges with miniature diphasic T-waves.

**Comment:** The combined right and left electrical axis deviation indicated either postural cardiac factors or enlargement of the heart to the right as well as to the left. This ECG is probably expressive of the coexistence of both conditions.

**Case 2:** A white man, 40 years of age. Onset of pulmonary tuberculosis in 1945. He had right thoracoplasty with removal of seven ribs completed March 9, 1948. Right upper lobectomy was performed November 30, 1948. Cor pulmonale was diagnosed in 1952. He since had recurrent episodes of cardiac decompensation. The sputum was still positive for tubercle bacilli.

**FIGURE 1:** P-pulmonale in precordial leads over the right side of the heart. Severe cor pulmonale due to pulmonary artery thrombosis. On April 18, 1952 peaked tall P-wave in V-2. On April 16, 1953 peaked tall P-wave in V-3.
**ECG findings.** On June 28, 1954, P-2 reached 0.5 millivolts. P in V-2 rose to 0.3 mvs. as compared with 0.2 mvs. on May 24, 1954. On July 15, 1954, P in V-3 also rose to 0.3 mvs.

This case demonstrates that in severe cor pulmonale there may develop, in addition to a high peaked P-wave in lead two, also a P-pulmonale in the precordial leads over the right heart.

**Case 3:** A white male veteran who at the age of 34 years developed in September 1944 bronchial asthma, probably of allergic etiology and permanent pulmonary emphysema ensued. Since 1949 to December 1953, he represents a respiratory cripple who is dependent on the administration of oxygen even when not being in status asthmaticus. Cor pulmonale was diagnosed in 1949 on the basis of the electrocardiogram. The roentgenogram did not show enlargement of the heart, however the width of the right pulmonary artery measured 14 mm, suggesting the maximal normal width. Digitalis was needed frequently during the protracted period of hospitalization whenever episodes of right cardiac failure occurred.

**Serial ECG changes.** December 29, 1949. P-2 and P-3 0.3 mv. September 20, 1951. P-2 and P-3 inverted. October 18, 1951. P-2 and P-3 upright, and 0.2 mv.

**Interpretation of numerous serial observations:** The low voltage of QRS-1, the relatively high upright R in AVF and the S-waves in left precordial leads point to right axis deviation as seen in pulmonary emphysema. This is here a sustained pattern. The transient P-wave abnormalities observed in connection with severe asthmatic attacks may be expressive of temporary enlargement of the right atrium. One may assume the existence of cor pulmonale in terms of a concentric hypertrophy of the right ventricle which does not need to be recognizable on the x-ray film. Return of the P-waves to a normal pattern indicates that the decrease of the circulatory load on the right heart is
reflected in the improved ECG. The flattening out of T-2 and T-3 during respiratory stress, together with the concomitant P-wave abnormalities, is in line with severe right ventricular strain as seen in cor pulmonale. When the P and T waves become abnormal and ST depression becomes apparent with it, then an element of coronary insufficiency is to be considered, too. However, the effect of electrolyte imbalance and a status approaching asphyxiation may also be reflected in such an ECG.


Comment: A review of serial electrocardiographic changes embracing four years permits the conclusion that in chronic cor pulmonale due to pulmonary emphysema, the P-wave in leads two and three may undergo a transformation from the tall peaked contour to inversion. At times the entire pattern of cor pulmonale may give place to normal complexes, without losing the right axis deviation. Nevertheless, concomitant clinical observation shows that even during the absence of a pathological electrocardiographic pattern pulmonary emphysema may necessitate the continuation of nasal oxygen administration.

Temporary loss of the characteristic contour of the P-pulmonale and episodes of electrocardiographic remissions of the cor pulmonale pattern toward normal complexes do not necessarily signify anatomical cardiac normalcy. Such electrocardiographic improvements may frequently accompany the clinical improvement of cardio-pulmonary insufficiency during long standing cor pulmonale before the fatal cardiac decompensation ensues.

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Addendum: Autopsy performed on Case No. 2 in November, 1964 showed marked cor pulmonale with conspicuous enlargement of the right atrium.

SUMMARY AND CONCLUSIONS

In marked right atrial enlargement due to thrombosis of the pulmonary arteries there were observed not only peaked P-Waves in leads two and three but also in the precordial leads V-2 and V-3 where the amplitude reached 0.4 millivolts.

In severe cor pulmonale resulting from pulmonary tuberculosis there were noticed, in addition to a high peaked P-Wave in lead two, serial changes in leads V-2 and V-3 with an increase in the P-Waves to 0.3 millivolts in these two leads.

In the absence of other causes the tall peaked P-Wave in the precordial leads over the right side of the heart may serve as a confirmatory sign of severe cor pulmonale.

The variability in shape, size and direction of the P-pulmonale during protracted illness is demonstrated in a more common case, namely chronic cor pulmonale due to pulmonary emphysema.

RESUMEN Y CONCLUSIONES

En el marcado crecimiento atrial debido a trombosis de las arterias pulmonares se observaron no sólo angulación de las ondas P en dos y tres, pero también en las precordiales V2 y V3 cuando la amplitud llegó a 0.4 milivots.

En el cor pulmonale severo resultante de tuberculosis se notaron además de una onda alta angulada P en dos, cambios en la serie en V2 y V3 con un aumento de lasondas P a 0.3 en estos dos puntos.
En ausencia de otras causas la onda P elevada en las precordiales durante enfermedad prolongada, se demuestra en un caso más común o sea en el cor pulmonale debido al enfisema pulmonar.

RESUME

Dans l’hypertrophie de l’oreillette droite, due à la thrombose des artères pulmonaires, on observa des ondes P en pic, non seulement dans les dérivations 2 et 3, mais encore dans les dérivations précordiales V2 et V3 où l’amplitude atteignit 0,4 millivolts.

Dans un cas de coeur pulmonaire grave secondaire à une tuberculose pulmonaire, on nota en plus d’une onde P en pic dans la dérivation 2, des altérations en série dans les dérivations V2 et V3, avec augmentation des ondes P jusqu’à 0,3 millivolts dans ces deux dérivations.

En l’absence d’autre cause, l’onde P en pic dans les dérivations précordiales droites peut servir à confirmer le diagnostic de coeur pulmonaire grave.

La variabilité de la forme, de la dimension et de la direction des ondes P au cours d’une maladie prolongée se trouve démontrée dans plus d’un cas, en particulier dans le coeur pulmonaire chronique dû à l’emphysème pulmonaire.

ZUSAMMENFASSUNG UND SCHLUSSFOLGERUNGEN

Bei ausgeprägter Vergrößerung des rechten Vorhofes infolge Thrombose der Lungenarterien wurden nicht nur erhöhte P-Zacken in Ableitung 2 und 3 beobachtet, sondern auch in den Brustwandableitungen V-2 und V-3, wo die Amplitude 0,4 Millivolt erreichte.

Bei schwerem cor pulmonale als Folge einer Lungentuberkulose wurde zusätzlich zu einer hohen P-Zacke in Ableitung 2 reihenweise Veränderungen in den Ableitungen V-2 und V-3 beobachtet mit einem Anstieg in den P-Zacken bis 0,3 Millivolt in diesen beiden Ableitungen.

Beim Fehlen anderer Ursachen kann die stark erhöhte P-Zacke in dem Brustwandableitungen über der rechten Seite des Herzens als ein bestätiges Zeichen eines schweren cor pulmonale dienen.

Die Veränderlichkeit in Gestalt, Grösse und Richtung des P-pulmonale während einer protrahierten Erkrankung wird an einem landläufigen Fall dargestellt, nämlich einem cor pulmonale infolge eines Lungenemphysems.

REFERENCES
