Atrial Parasytolute and Tachycardia*  
Modulation and Automodulation of a Parasytolic Focus  
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This report deals with a patient reflecting atrial parasytolute and episodes of atrial tachycardia. The P' waves during tachycardia were identical to the parasytolic P' waves. Atrial parasytolute was at times regular, as revealed by a precise mathematical relationship between the interectopic intervals, and on other occasions irregular. Irregularity was due to modulation, namely electrotonic influence exerted by the sinus impulses upon the parasytolic focus. Atrial tachycardia occurred only during the periods when atrial parasytolute was modulated. Atrial tachycardia has been interpreted as due to automodulation, a situation where the propagated parasytolic impulse exerts an electrotonic influence on the ectopic focus itself, leading to a marked unexpected acceleration of the ensuing parasytolic discharge. *(Ches 1992; 102:622-25)*

Since Jalife and Moe* in 1976 pointed out the mechanism of electrotonic influence (modulation) exerted by the dominant rhythm on a parasytolic focus, several reports on modulated parasytolute have been published.*10 In 1982, Antzelevitch et al* described the phenomenon of "automodulation," namely, the electrotonic influence exerted by the parasytolic impulses on the focus itself. This leads to the so-called "supernormal modulation," a mechanism also recognized in the clinical setting.12-15

This presentation reflects a patient with atrial parasytolute and atrial tachycardia in whom modulation is evident and, in addition, tachycardia may be considered as a result of automodulation.

**CASE REPORT**

A 74-year-old man suffering from mild hypertension and chronic renal failure complained of palpitations. At the time of observation, the patient was drug free. Several electrocardiograms, with duration up to 5 min, were recorded during a day; analysis of these tracings reveals the following.

**Regular Atrial Parasytolute**

This is reflected in Figure 1, where several ectopic P' waves, labeled with dots, are evident. Their coupling intervals are variable, ranging from 76 to 90 (all time intervals are expressed in hundredths of a second), and atrial fusion beats (labeled F) occur. The interectopic intervals are mathematically related to each other, being multiples of 139.5 to 146. This pattern of atrial parasytolute is defined as regular on the basis of a variation index of 4.5. The variation index was introduced to assess the regularity of a parasytolic rhythm, and is calculated according to the following formula:

\[
\text{Maximal difference between calculated values of the parasytolic cycle} \times 100
\]

Average value of the apparent parasytolic cycle

Parasytolute is defined as regular whenever the variation index is ≤4, and irregular in the presence of a variation index >4.5.

**Irregular Atrial Parasytolute**

This is evident from Figure 2, where, at the end of an episode of atrial tachycardia, the sinus rhythm is disturbed by several beats of parasytolic origin. The "pure" parasytolic cycle is 168 and is revealed from two consecutive P' waves, not separated by sinus

**FIGURE 1.** A continuous recording of standard lead 2. Ectopic P' waves are labeled with dots. Numbers express the interectopic intervals in hundredths of a second. Calculation of the parasytolic cycle is shown in parentheses. Atrial fusion beats are labeled F.

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Atrial Parasytolute and Tachycardia (Satullo et al)
impulses. This parasystolic rhythm is irregular, since the mathematical relationship between the interectopic intervals is not precise; for example, the fourth P'–P′ interval measures 197, whereas the ensuing long P′–P′ interval is 474 (158 × 3). The variation index calculated from this tracing is 23.4, which indicates an irregular parasystole. The mechanism leading to irregularity is modulation, namely the electrotonic influence exerted by the dominant rhythm on the parasystolic rhythm, as diagrammed in Figure 2. The irregularity of P′–P′ intervals is explained by the phase-response curve represented in Figure 3. For example, the P′–P′ interval of 197 (top strip of Fig 2) is longer than the “pure” parasystolic cycle of 168. This is due to the effect of the sinus impulse that occurs at 75 from the preceding parasystolic P′ wave. According to the modulation hypothesis, a nonparasystolic impulse manifesting early within the parasystolic cycle delays the ensuing parasystolic impulse, leading to prolongation of the manifest parasystolic interval. On the basis of the phase-response curve (Fig 3), a sinus impulse occurring at 75 from the preceding parasystolic discharge (at 44.5 percent of the parasystolic cycle) prolongs the ectopic cycle by 17.5 percent (29 hundredths of a second).

**Atrial Tachycardia**

An episode of atrial tachycardia is reflected in Figure 4. The P′ wave configuration during tachycardia is identical to that observed in atrial parasystole (Fig 1). Eight episodes of atrial tachycardia were available for analysis. Each of them occurred in the presence of irregular parasystole, namely when the variation index was >5, and the modulation mechanism was evident. On the contrary, no atrial tachycardia occurred throughout the recordings reflecting regular atrial parasystole.

**DISCUSSION**

**The Coexistence of Regular and Irregular Parasystole**

The first and most evident feature of these tracings is that parasystole is at times regular and on other occasions irregular. Moreover, calculation or measurement of the “pure” parasystolic cycle leads to a relatively wide range of values (from 135 to 170). These observations are in agreement with data obtained by Holter monitoring, demonstrating that modulation can be intermittent, occurring in only some of several samples within the same day. This is not surprising, since experimental studies have clarified that the occurrence and the extent of parasystolic modulation depend on the characteristics of the inexcitable gap that separates the parasystolic focus from the surrounding myocardium. The variation of electrical resistance representing the inexcitable gap leads to change from “classical” or regular parasystole to modulated parasystole and vice versa. Al-

**Figure 2.** A continuous recording of standard lead 3, diagrammed to show the effects of parasystolic modulation. Numbers within the tracing express the interectopic (P′–P′) intervals. Vertical bars reflect the ectopic discharges. Line C = scheduled discharge; line B = intermediate discharge; line A = actual discharge. Numbers in line A reflect the time intervals between the delivery of the ectopic impulse and the ensuing sinus beat(s). The intervals between an ectopic discharge and the second consecutive modulating sinus beat(s) are shown in parentheses. Dotted areas represent the refractory period of the atria. The detailed calculation of each modulation is shown in line D. For example, 75 = 44.5 percent − > + 17.5 percent (+29) means that a sinus impulse occurring at 0.75 s from the preceding ectopic discharge falls at 44.5 percent of the parasystolic cycle and prolongs the ectopic cycle by 17.5 percent, which corresponds to 0.29 s. The first ectopic impulse in the bottom strip is assumed to undergo an exit block, namely, it does not result in a P′ wave despite occurring outside the atrial refractory period.

**Figure 3.** The derived phase-response curve. The abscissa reflects the ectopic cycle length percentage. The ordinate reflects the positive or negative variations of the ectopic cycle length, expressed as percentage of basic value.
though there is no in vitro demonstration of hour-to-hour variations in the protection mechanism of parasystole, autonomic influence is likely to affect the electrical resistivity of the protection gap in some way. This may explain the reason why modulation occurs only in some of the tracings recorded throughout one day.

The Mechanism of Tachycardia

A major problem in the patient whose case is reported is the relationship between parasystole and tachycardia. It should be pointed out that tachycardia occurred only when parasystole was modulated. The precise mechanism of tachycardia cannot be stated with certainty, but coincidence with modulated parasystole suggests a link between tachycardia and modulation. This tachycardia, in fact, may be a parasystolic tachycardia due to automodulation of the focus (see below). The cycle of tachycardia is relatively short (from 52 to 61), which makes this hypothesis unlikely at first glance, provided that the parasystolic cycle of discharge is far longer (168). Theoretically, the true cycle could be shorter than 168 as a consequence of exit block, for example 3:1 exit block. In this sense, tachycardia could be due to sudden disappearance of exit block. Nevertheless, the rate of tachycardia can be explained better by supernormal modulation. In 1982, Antzelevitch et al reported a triphasic phase-response curve where very early dominant impulses, occurring during a brief "supernormal" phase, caused an unexpected precipitation of the ensuing parasystolic discharge. This resulted in dramatic shortening of the manifest ectopic cycle. Such a mechanism can explain, for example, the association between parasystole and couplets. The parasystolic impulse propagated to the surrounding myocardium exerts an electrotonic influence on the parasystolic focus itself: whenever this influence occurs during the "supernormal" phase, a further parasystolic discharge will ensue. If this mechanism occurs again and again, tachycardia will result. This situation, defined as automodulation, is indistinguishable from, and possibly identical to, a reentrant tachycardia originating from the parasystolic focus. Parasystole and reentry, indeed, are, in a certain sense, not opposite phenomena, but merely represent the extremities of a continuous spectrum. Laboratory research has demonstrated that the same experimental setting (the three-bath preparation of canine false tendon) can result either in modulated parasystole or in reflected reentry (an electrotonically mediated reentry) depending on the electrical characteristics of the excitable gap.

The tachycardia reported herein could be due either to automodulation of a parasystolic focus or to reflected reentry at the site of the focus itself. Nevertheless, the occurrence of tachycardia only when the parasystolic rhythm is modulated makes the parasystolic mechanism of tachycardia more likely.

The triphasic phase-response curve associated with "supernormal" modulation of a parasystolic rhythm has been reported several times, but the present study is, to our knowledge, the first one where this mechanism is applied to explain a clinical example of ectopic atrial tachycardia.

REFERENCES

3 Jalife J, Antzelevitch C, Moe GK. The case for modulated parasystole. PACE 1982; 5:911-26
We report an adult who contracted measles and developed severe complications.

**Case Report**

A 36-year-old female emergency department nurse with no prior health problems was exposed to a patient with active measles while at work in October 1989. Ten days later, she developed a typical viral prodrome with a low-grade fever, nausea, and fatigue, followed by a temperature of 40.5°C, nonproductive cough, coryza, and conjunctivitis.

She was seen by her local physician 13 days after her exposure to measles. A chest roentgenogram was normal. Over the next two days, she developed a maculopapular rash and was hospitalized for dehydration. A consultant noted Koplick spots, and based on the history of exposure and classic physical findings, a diagnosis of measles was made.

Forty-eight hours after hospital admission the patient developed progressive respiratory distress and an arterial blood gas sample obtained while breathing room air showed a PaO₂ of 29 mm Hg, PaCO₂ of 27 mm Hg, and a pH of 7.34. Hypoxemia persisted despite supplemental oxygen administered by high-flow face mask; endotracheal intubation and mechanical ventilation were instituted. While being mechanically ventilated with FiO₂ of 1.0 and PEEP of 15 cm H₂O, the arterial PaO₂ was 155 mm Hg, and respiratory system compliance was determined to be only 17 ml/cm H₂O. A chest roentgenogram showed diffuse bilateral alveolar and interstitial infiltrates (Fig 1).

Cytologic brushings obtained from a lower lobe bronchial tube and multiple sputum samples revealed no pathogens on culture or cytologic staining and analysis. Measles infection was confirmed by a fourfold rise in measles serology from baseline. Supportive care and empiric broad-spectrum antibiotic coverage was associated with gradual improvement and extubation after 11 days of mechanical ventilation. The patient was discharged home 29 days after her admission to the hospital.

Because of the severity of this patient’s measles infection, we performed an immunologic evaluation. Serologic testing revealed a negative human immunodeficiency virus (HIV), normal T-helper to suppressor cell ratio, a positive skin test response to mumps,

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**Hypoxemic Respiratory Failure Complicating Nosocomial Measles in a Healthy Host**

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Since 1983 there has been a steady increase in the number of cases of measles reported to the Centers for Disease Control (CDC), and recent large-scale outbreaks have heightened public awareness. Although mortality from measles is considered rare, systemic complications occur frequently. This case highlights the significant morbidity and potential mortality of measles infection in a normal adult host who did not meet CDC guidelines for measles vaccination.

(CHEST 1992; 102:625-28)

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**Figure 1.** Chest roentgenogram shows diffuse bilateral alveolar and interstitial infiltrates.