sensitization of the airways in the atopic asthmatic patient could somehow result in a defect in calcium membrane flux or intracellular calcium homeostasis in the airway smooth muscle. This, however, would not explain nonatopic asthma and would be difficult to correlate with asthma and the persistent airway reactivity to mediators such as acetylcholine and histamine during periods when individuals with asthma are not exposed to specific antigens or allergens. Nevertheless, increased airway reactivity to methacholine has been reported following sensitization to Ascaris in monkeys and dogs as reported by Patterson et al and Kepron et al. More importantly, increased airway reactivity to histamine or methacholine does occur following antigen challenges that are associated with late bronchial responses.

Since much of the information derived with respect to smooth muscle pharmacology may be relevant to calcium ion movement and mast cell mediator release, the relationship between airway hyperreactivity and mast cell sensitization deserves further investigation. In this regard, the requirement of calcium for the anaphylactic release of chemical mediators from target cells such as mast cells or basophils is well established.

Since calcium is a common mediator for the various neurotransmitters and physical factors that evoke the bronchial constriction and mast cell mediator release, a defect at the level of cellular calcium regulation may be linked to the etiologies of bronchial hyperreactivity in asthma.

Robert G. Townsley, M.D.
Chief, Allergic Disease Center;
Professor of Medicine, Creighton
University, Omaha

REFERENCES
2 Patterson R, Harris KE. The effect of cholinergic and anticholinergic agents on the primate model of allergic asthma. J Lab Clin Med. 1976; 87:65-72
5 Kazimierczak W, Diamant B. Mechanism of histamine release in anaphylactic and anaphylactoid reactions. Prog Allergy 1978; 24:295-65

Concealed Ventricular Parasytole Exposed by Abrupt Cessation of Pacing

To the Editor:

I should like to comment on the article by Drs. Castellanos and Castillo in the September, 1982 issue of Chest. The evidence these authors present to support their thesis that they are dealing with a concealed parasytolic ventricular pacemaker is both inferential and very soft. The one bit of hard and convincing data that should be presented is missing. In this article, the authors describe the appearance of an alleged parasytole upon cessation of artificial electronic ventricular stimulation. To be completely convincing, they should have once again stimulated the patient electrically and then allowed the so-called parasytolic pacemaker to come out again after stopping stimulation. If we could observe the idioventricular pacemaker to march through the stimulated area and come out again when the stimulated area was stopped, and if we could observe the idioventricular pacemaker to maintain a parasytolic interval even though it would not appear because of an exit block during ventricular stimulation, then we could agree with the authors that there was indeed a parasytolic rhythm. To hide behind the conclusion that this so-called parasytole represents "an intermittent arrhythmia" is a cop-out. This is like saying that any ectopic arrhythmia that intermittently appears alongside another type of dominant cardiac rhythm represents an intermittent parasytole that is constantly being reset by non-parasytolic QRS complexes.

The definition of a ventricular parasytolic pacemaker, whether concealed or not, as shown by Katz and Pick and others, is now and has always been associated with three criteria: first and foremost, there is a constant RR interval of the parasytolic pacemaker that can be measured out at a regular rate irrespective of the dominant rhythm even though, at times, the parasytolic QRS complex may be concealed because there is an exit block due to a refractory period of the dominant rhythm. The second criterion is one of non-fixed coupling with the dominant rhythm. The third criterion is the presence of fusion beats. Fusion beats, at times, might not be present if both the parasytolic and dominant rhythms arise close to the same point in the heart. One should still look for two out of the three criteria prior to concluding the presence of parasytole. In this particular case, however, only one of the three criteria is present, namely: non-fixed coupling.

William S. Breall, M.D., F.C.C.P.
San Francisco

REFERENCE

To the Editor:

Dr. Breall's points are well taken. We did observe that the parasytolic cycle length remained unchanged during the periods of pacing. What we wanted to emphasize, as done by Nau et al., was that the exit block in "concealed" parasytole is different from the usual types of exit conduction disturbances occurring in ventricular parasytole. The latter, usually type 2 (Mobitz) or type 1 (Wenckebach), do not depend so much on concealed penetration, in the ventricular parasytolic junction, of entering impulses. Thus considered "concealed" parasytole is but a variant of continuous parasytole with exit block. Presently, the diagnosis of parasytole has been extended to include a variety of arrhythmias which do not fit "classical" parasytole. These authors have observed that the zone of protection could be traversed by subthreshold, electronic depolarization (not by full action potentials) to produce: a) "modulated" parasytole (time-dependent variations in parasytolic cycle length); b) "extra-trained" ("captured") parasytole; c) "reentrant" ("reflected") parasytole with fixed, and variable, coupling intervals; and d) "pacemaker annihilation" ("extinction") of parasytolic activity. These findings may be construed to indicate that the spectrum of parasytolic activity is much wider than what is classically accepted.

Agustin Castellanos, M.D., F.C.C.P.; and Cesar A. Castillo, M.D.
Miami

Reprint requests: Dr. Castellanos, Cardiology D 30, University of Miami School of Medicine, PO Box 019960, Miami 33101

REFERENCES
1 Nau GJ, Aldariz AE, Acunzo RS, Chiale PA, Elizari MV, Rosenbaum MB. Concealed ventricular parasytole uncovered in the