ing complication which could not be ignored, and should compel manufacturers to provide every assurance possible for a break-free catheter tip transducer. Short of this, it may be necessary to limit catheter usage to a restricted number of studies in human subjects.

This study was supported by Grant RR-305 from the General Clinical Research Centers Program of the Division of Research Resources, National Institutes of Health.

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Propranolol and Quinidine

TO THE EDITOR: I read with interest the paper by Fors et al. on the successful use of propranolol and quinidine in your February 1971 issue.[1] May I draw your attention to earlier reports of similar good results arising from the combined clinical use of these drugs[2-5] and to the experimental studies of the Italian group of investigators confirming these results.[6-7] As Kennedy and West[8] demonstrated that the negative inotropism induced by quinidine administration may turn to positive inotropism at slower heart rates, the theory has been put forward[9] that the beneficial effect of the combination therapy may arise from the bradycardia accompanying the administration of propranolol.

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The Authors Reply

We thank Stern for calling our attention to the excellent paper by Kennedy and West. This study showed that the negative inotropic effect and the alternation in contractile force produced by quinidine was related to quinidine-induced changes in excitability rather than a direct effect of quinidine on contractile processes. The negative inotropic effect and depressed excitability were frequency-dependent, being noted at short beat intervals. At slow heart rates quinidine showed a positive inotropic effect in contrast to propranolol. This is good news for a much maligned drug and confirms the observation that quinidine seldom overtly depresses myocardial function. It was with great misgiving that we began using propranolol and quinidine together knowing that both drugs were supposed to have a negative inotropic effect. The number of patients actually experiencing heart failure with this combination was exceedingly small and could be related almost entirely to the use of propranolol alone, since stopping administration of this drug alone would reverse the signs of failure. These new data support our belief that the fear of using the 2 drugs in combination is unwarranted.

We acknowledge that Stern published the results using propranolol and quinidine in combination in 2 patients in an annotation in October 1966. Our first publication did not appear until October 1967 (Circulation 36: suppl 2: 221, 1967).

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Trifascicular Block and Complete Heart Block

TO THE EDITOR: I have read with interest the article by Steiner C, Lau SH, Stein E, et al. (Electrophysiologic documentation of trifascicular block as the common cause of complete heart block, Amer J Cardiol 28:436–441, 1971).

The title indeed suggests that the authors have documented ("proof or evidence to substantiate," Webster's Dictionary) the simultaneous involvement of all 3 segments of the ventricular conduction system in patients with complete heart block. The authors, however, only reiterate the fact, already well known from some of their own previous investigations and those of others, that in patients with acquired complete heart block, His bundle electrocardiography demonstrates that the block is located distal to His potential. I failed to see in their results the actual documentation that proves that the site of block is located in the trifascicular network. Furthermore, in the discussion, they clearly state "When this technique [His bundle electrocardiography] is used it is not possible to be more precise in defining the exact site of block. It may be in the distal common bundle, in the bundle branches or in the peripheral Purkinje system."

This paper confuses further the discussion of complete heart block in that it attempts to provide electrophysiologic documentation for a most interesting concept but fails to do so. It would have been better if this title had been reserved for the publication of re-