of end-diastolic radius!* Thus, Dr. Shimosato's data, properly interpreted, indicate that *CE* V_{max} is markedly sensitive to ventricular and diastolic fiber length and therefore that it cannot uniquely reflect the inotropic state of the muscle. In other words the data show, contrary to his own conclusion, that *CE* V_{max} is not a valid index of cardiac muscle contractility.

Dr Shimosato's reliance upon the *two*-element Hill model for his analysis also leads to erroneous conclusions. Modern studies agree that at least *three* elements are necessary in a model of cardiac muscle. Although some have assumed that a three-element model may be reduced to a two-element model during isovolumic contraction, it can be shown that this assumption is invalid when calculating *CE* V_{max} of cardiac muscle (Pollack, *ibid*).

Finally, certain geometric assumptions which are implicit in Dr. Shimosato's analysis should be drawn to the attention of the reader. His application of the "Law of Laplace" to his data presupposes that:

1. The ventricle is spherically shaped;
2. It has a thin wall; this generally means that the wall thickness must be less than 10 per cent of the radius if reasonable accuracy is desired;
3. The wall is homogeneous and isotropic, *i.e.*, the fibers are arranged *uniformly* around the sphere and their contractile properties in all tangential directions are identical;
4. The sphere does not change shape during isovolumic contraction.

Thus, even if one disregards the earlier-mentioned reservations, Dr. Shimosato's calculation of *CE* V_{max} would be valid only to the extent that these four assumptions are valid.

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To the Editor:—Dr. Gerald H. Pollack's letter suggests that the validity of the maximal intrinsic velocity (V_{max}) as an index of myocardial contractility is being seriously challenged. Unfortunately, this statement was made without established evidence. Certain comments in his criticism are appreciated; however, most of the points are minor and do not relate the the fundamental basis for the concept of force–velocity relations as a reliable and accurate index for evaluation of changes in myocardial contractility owing to anesthetics.

Several important points relevant to Doctor Pollack's comments need clarification.

1) There has been no scientific evidence indicating that the use of maximal intrinsic velocity (V_{max}) as an index of contractility is invalid. On the contrary, most cardiovascular researchers use the concept of V_{max} as the index of contractility (Henderson *et al.*, Am. J. Physiol. 217: 1273, 1969; Parmley, Brutsaert and Sonnenblick, Circulation 22: 521, 1969; Gault, Ross and Braunwald, Circulation 22: 451, 1968; Siegel, ANESTHESIOLOGY 30: 519, 1969).

2) Evidence that V_{max} is dependent upon fiber length is not conclusive. Data reported by Noble *et al.* (Circ. Res. 24: S21, 1969) only suggests that the shape of the quick-release force–velocity curves is dependent upon muscle length. However, the quick-release method itself has been criticized on the grounds that any change in muscle strain (release or stretch) tends to promote relaxation (*CE* uncoupling) (Brady, Physiol. Rev. 48: 570, 1968). On the other hand, it has been well demonstrated that V_{max} is independent of muscle length in isotonically-contracting cardiac muscle (Sonnenblick, Am. J. Physiol. 202: 931, 1962).

It has also been shown that cardiac muscle, unlike skeletal muscle, has a slow onset and decay of the active state, and that the quick-release method for obtaining an instantaneous force–velocity relation inherits the problem of uncertainty in determining the maximal velocity (Brady, Physiol. Rev. 48: 570, 1968). Noble *et al.* demonstrated that the quick-release method for the assessment of V_{max} was not precise when the myocardial fiber length was varied.

3) Stiffness of the series elastic element (SE) increases as a linear function of load (Sonnen-

* This is easily calculated by choosing typical values for *K* and *C* from Yeatman *et al.* (*ibid.*), selecting two values for *r*, calculating corresponding values for *h* (assuming myocardial-wall volume remains constant), and estimating (from the literature) two values of initial *dP/dt* corresponding to the two different radii. Substitution of these two sets of parameters into equation 4, letting *P* approach zero in each case, generates the two values of *CE* V_{max}.

blick, Am. J. Physiol. 207: 1330, 1964). As Dr. Pollack indicates, this relation may be expressed as $dP/dl = k \cdot P + C$, where P is load, k a constant for the slope of the linear equation, and C a constant for the linear intercept. Since C, which is dP/dl at zero load, is consistently quite small and not influenced by any inotropic intervention or temperature (Parmley and Sonnenblick, Circ. Res. 20: 112, 1967; Yeatman *et al*; Am. J. Physiol. 217: 1030, 1969), it has been disregarded, and the equation becomes $dP/dl = k \cdot p$.

Thus, for use in the intact heart, $k = 24.7$ (corrected for a muscle 1 cu cm in dimension), and dP/dl becomes $k \cdot p$.

4) Several mechanical analogues of muscle have been proposed. Recent study shows the Maxwell-type model a preferred approximate model of cardiac muscle. However, according to Hill (Proc. Roy. Soc. Med., Ser. B., 126: 136, 1938), muscular contraction can be considered conceptually in terms of two series components: an active contractile element (*CE*), and an undamped passive series elastic element (*SE*). Both *CE* and *SE* are coupled to a parallel elastic component (*PE*). The parallel elastic component (*PE*) does not appear to participate directly in contraction and may contribute to resting tension only when relatively large initial muscle lengths are attained. It has been shown sufficient to use the simplified form of the two-component systems for analysis of muscular contraction (Sonnenblick, Am. J. Physiol. 21: 975, 1962). Use of a more complicated model only leads to unnecessary complicated mathematical calculation without yielding additional fruitful information.

5) Finally, the use of thin-wall spherical configuration for the left ventricle has been accepted by many investigators. Recent study of a comparison of calculations of circumferential wall-stress in the left ventricle, based on the thin-wall model and the thick-wall model, revealed that the thin-wall model overestimates only 5 to 15 per cent mean stress, as compared with the thick-wall model (Hood *et al.*, Circ. Res. 24: 575, 1969). Therefore, calculation of V_{max} is not seriously affected by the assumption that the left ventricle is a thin-wall spherical or ellipsoidal model. I believe that mathematics are a convenient means for the modern cardiovascular researcher, but the mere use of mathematics without thorough understanding of basic cardiovascular dynamics leads to erroneous conlcusions. Certain valid assumptions are necessary, so long as the user is aware of error related to the assumptions and the basic fundamental concept is not seriously distorted.

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Halothane for Cesarean Section

To the Editor:—In the paper "Intravenous Thiobarbiturate Anesthesia for Cesarean Section" by Kosaka, Takahashi and Mark ANESTHESIOLOGY 31: 489, 1969), it is stated that halothane was used in some cases for the induction of anesthesia and it was used routinely following delivery of the baby.

In my limited experience I thought that halothane's relaxing effect on uterine musculature made it unsafe to use in operations on the pregnant uterus. Will you please comment on the use of halothane under these circumstances.

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Reply to Dr. Neal:—*You are correct in implying that halothane, according to the evidence available, may increase uterine bleeding, possibly through an effect on uterine muscle tone. It is not my role as editor to comment on the safety of the drug, since much depends upon how an anesthetic is given.—The Editor*