Regional left ventricular wall motion is easily measured by sonomicrometry. Dimension transducers can be placed in the epicardial, middle or endocardial portion of the ventricle to measure segment length, on opposite walls to measure diameter, or on the epicardium and the endocardium to measure wall thickness [3, 11, 23, 29]. Analysis and interpretation of dimension signals may be difficult because of the need to determine the exact timing of end-diastole, onset of ejection, and end of ejection. This is only possible when aortic blood flow and aortic pressure are measured at the same time in the immediate vicinity of the aortic valve. Otherwise transmission delays occur and the timing of these events becomes inaccurate.

Examination of the instantaneous relationship between pressure and dimensions, the pressure–dimension loop, partly overcomes the need to use signals other than pressure and dimensions to determine the timing of end-diastole, onset and end of ejection because they correspond to obvious features of the loop (fig. 1). Moreover, pressure–dimension loops provide additional information regarding synchrony or asynchrony of contraction, segmental work and regional contractility.

This brief review will consider only the pressure–length loop.

SHAPE OF PRESSURE–LENGTH LOOPS

Pressure–length loops consist of four segments corresponding to isovolumic contraction, ejection, isovolumic relaxation and ventricular filling (fig. 1). In the normal heart, most pressure–length loops are approximately rectangular (fig. 2A). However, because of the complexity of the geometry of the left ventricle, some regions may exhibit early shortening (fig. 2B), or early shortening followed by early lengthening (fig. 2C) so that the isovolumic phases of the cardiac cycle may not be isometric. These features are explained by a reduction or an increase in area of the inner surface of the left ventricular cavity. During isovolumic contraction the ventricle becomes more spherical and the area tends to decrease, while during isovolumic relaxation the ventricle becomes more ellipsoidal and the area tends to increase [13]. Furthermore, differences in regional contraction patterns have been observed depending upon the location of the length transducers. Extent of shortening and patterns of wall motion differ between the long and short axis, and also between apical and basal regions [12]. Thus pressure–length loops may manifest different characteristics depending upon which region of the ventricle is examined.

Pressure–length loops may appear to lean to the right (fig. 2D). This is usually caused by ischaemia [5, 29], and results from lengthening during isovolumic contraction and shortening during isovolumic relaxation (post-systolic shortening). Systolic lengthening represents displacement...
Early shortening may represent active shortening that is not contributing to pump function because it occurs after the end of ejection, or pure elastic recoil. With extreme ischaemia the pressure–length loop is even more distorted (fig. 3). Examination of the pressure–length loops make it obvious that the total length change may be partly or fully a result of paradoxical wall motion.

The controversy surrounding the possibility of enhanced function of the normal myocardium when a limited area becomes ischaemic, may be partly resolved by examination of pressure–length loops [15]. As the ischaemic area lengthens during isovolumic contraction, some of the normal myocardium must shorten. While total shortening may increase, ejection shortening may remain unchanged (fig. 3). The early shortening merely causes the transfer of blood within the left ventricle away from the normal area into the ischaemic area. It does not necessarily reflect enhanced function.

Abnormal pressure–length loops have been observed in the absence of ischaemia during studies of the interactions between the calcium channel blocker verapamil and the inhalation anaesthetics halothane and isoflurane. In the absence of reduction of coronary flow, these observations suggest that pressure–length loops may lean to the right because of asynchrony of ventricular contraction as a result of altered excitation–contraction coupling, rather than ischaemia [19, 31].

**Fig. 3.** When a coronary artery is abruptly occluded, the normal pressure–length loop of the dependent segment (top left) becomes distorted (top right) and most of the dimensional length change represents passive lengthening followed by elastic recoil. In neighbouring normal segments, the pressure–length loop, previously rectangular (bottom left), is also modified and leans to the left (bottom right). Its total shortening (TS) increases but ejection shortening (EjS) does not.

**AREA OF THE PRESSURE–LENGTH LOOP: AN INDEX OF SEGMENTAL WORK**

The area of the pressure–volume loop represents ventricular stroke work. By analogy, the area of
the pressure–length loop may be regarded as an index of regional work. However, because it has units of pressure and length and not pressure and volume, it is only an index of segmental work [6, 18, 24, 30]. Increases in preload increase the loop area and make it possible to obtain regional ventricular function curves (fig. 4). Positive inotropic interventions shift the regional function curve upwards and to the left [8].

Interpretation of the area of the pressure–length loop may be difficult when contraction is abnormal (fig. 5). When the loop leans to the right, its area encompasses three regions:

1. The region corresponding to systolic lengthening. This obviously represents work done on the segment, as opposed to work done by the segment.
2. The region delineated by post-systolic shortening. This represents either active work or work caused by elastic recoil. However, it is ineffective in terms of ventricular pump function as it does not contribute to ejection.
3. The middle region of the loop, the only region which represents work contributing to pump function. The ratio of this area to the whole area gives an approximation of the effective contribution of the segment to ejection. In severe ischaemia the area may become negative when the loop is described in an anticlockwise fashion (fig. 3).

**END-SYSTOLIC PRESSURE–LENGTH RELATIONSHIPS: AN INDEX OF REGIONAL CONTRACTILITY**

In 1895, Otto Frank used the pressure–volume relationships in the frog’s heart in his detailed analysis of cardiac contraction [10]. More recently, the pressure–volume relationships at the end of systole have been extensively studied in perfused isolated left ventricle preparation and in the intact heart [20, 25, 26]. At the end of systole there is a linear relationship between pressure and volume that is very sensitive to inotropic interventions (fig. 6).

Suga and his colleagues [27] developed the concept of time-varying elastance: wall stiffness increases during systole and decreases during diastole. This concept has been extensively studied, and the end-systolic pressure–volume relationship is considered to be insensitive to changes in preload and afterload. Thus, the pressure–volume line at end-systole has been regarded as an index of contractility independent of preload and afterload [21]. This concept has been challenged and recent evidence suggests that altered resistance [17], and changes in preload [8] may influence the slope of the end-systolic pressure–volume relation. Changes in inotropy may cause not only a change in slope but also a shift of the end-systolic pressure–volume line [9, 22].

By extending the concept of time-varying
elastance to pressure-length relationships, it can be shown that a linear relationship exists between pressure and diameter [4, 16], and pressure and length at the end of systole (P-Les) (fig. 7) and that increases in inotropy increase its slope (fig. 8) [7, 8]. However, myocardial depression may cause a shift of the line towards larger dimensions, but without change in slope (fig. 9) [8, 9]. It must be noted that end-systolic pressure-length relationships can be obtained either by increasing afterload or by increasing preload. Although both interventions increase end-systolic pressure and length, the slope of the P-Les is always flatter when preload, as opposed to afterload, challenges are applied in order to generate the end-systolic pressure-length points. Thus for the intact heart, the way in which changes in pressure and length are obtained influences the slope of the P-Les [2, 8]. Interpretation of the data must take into account the way in which pressure was altered.

An early tenet of the analysis of end-systolic pressure-dimension relationships was that a constant volume or dimension intercept exists at zero pressure (figs 6, 8) [27]. This has been subse-
Fig. 9. While positive inotropic interventions increase the slope of the end-systolic pressure-length relationship (left hand panel), negative inotropic interventions (such as the addition of halothane to pentobarbitone) may cause a shift to the right without change in slope (right hand panel) [9].

sequently disproved [4] and, at regional level, the intercept of the P–L_es line with the length axis is not constant [8].

Another tenet is that increases and reductions in loading have the same effect. This is not confirmed in the intact heart where, after a rapid reduction in afterload, the end-systolic pressure–length co-ordinates do not immediately fall on the P–L_es line: the length remains longer for several beats (fig. 10). This indicates that the viscoelastic properties of the myocardial segment influence the end-systolic pressure–length relationship. The passive stretch of the segment, demonstrated by the rightward displacement of the loop, results in hysteresis. However, when the preload is high, the initial fibre length utilizes all potential passive stretch and hysteresis does not occur [32].

Severe regional myocardial ischaemia limited to a small area of the left ventricle renders the analysis of the end-systolic pressure–length relationships almost meaningless [28]. In these circumstances pressure reflects the global ability of the ventricle to develop tension, while changes in regional dimensions are determined by the viscoelastic properties rather than the performance of

Fig. 10. Series of pressure–length loops generated by partial aortic occlusion. The first loop (1) after releasing the occlusion is to the right of the control loop. This demonstrates that a decrease in pressure does not have the same effect on the pressure–dimension relationships as does an increase in pressure [32].

Fig. 11. When a segment is rendered ischaemic, the loop is deformed and the end-diastolic length may be greatly increased. Provided the ischaemic area is small, the remainder of the left ventricle is able to develop pressure and the slope of the end-systolic pressure–length line may remain essentially unchanged.
the ischaemic segment. The end-systolic pressure–length co-ordinates may continue to fall on a straight line, and the slope may remain almost unchanged even though active contraction has ceased (fig. 11).

Examination of the pressure–length loop greatly facilitates detection of segmental asynchrony. The contribution of the segment to pump function can be appreciated by assessment of the effective loop area. Assessment of regional contractility based on the end-systolic pressure–length relationship is also possible. However, depending upon the level of contractility, changes in slope or shifts of the end-systolic pressure–length line may occur. Moreover, analysis of the end-systolic pressure–length relationship must take into account the way in which pressure has been altered.

REFERENCES

